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Behavioral Impulsivity Does Not Predict Naturalistic Alcohol Consumption or Treatment Outcomes

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Abstract

Objective—The purpose of this study was to determine if behavioral impulsivity under multiple conditions (baseline, after alcohol consumption or after serotonin depletion) predicted naturalistic alcohol use or treatment outcomes from a moderation-based contingency management intervention.

Method—The current data analysis pulls information from three phases of a large study: 1) Phase 1 examined baseline and the effects of alcohol use and serotonin depletion on three types of behavioral impulsivity: response initiation (IMT task), response inhibition (GoStop task), and delay discounting (SKIP task); 2) Phase 2 involved 28 days of naturalistic drinking; and 3) Phase 3 involved 3 months of contingency management. During phases 2 and 3 alcohol use was measured objectively using transdermal alcohol monitors. The results of each individual phase has been previously published showing that at a group level the effects of alcohol consumption on impulsivity were dependent on the component of impulsivity being measured and the dose of alcohol consumed but serotonin depletion had no effect on impulsivity, and that a moderation-based contingency management intervention reduced heavy drinking.

Results—The current analysis combining data from those who completed all three phases ($n = 67$) showed that impulsivity measured at baseline, after alcohol consumption, or after serotonin depletion did not predict naturalistic drinking or treatment outcomes from a moderation-based CM treatment.

Conclusions—Contingency management interventions may prove to be an effective intervention for impulsive individuals, however, normal variations in measured impulsivity do not seem to

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Conflicts of Interest

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relate to normal variations in drinking pattern or response to moderation-based contingency management.

Keywords

Impulsivity; Contingency Management; Alcohol; Treatment Outcomes; Transdermal Alcohol Monitoring

Introduction

Impulsivity is considered a risk factor for both the initiation of alcohol use and the development of alcohol use disorders,^{1,2} with more impulsive individuals more likely to develop more severe alcohol use problems.^{2,3} Research, although limited in number, has also shown that individuals with lower levels of impulsivity are more likely to have better alcohol-use treatment outcomes compared to highly impulsive individuals.^{4,5} These studies, however, have typically involved abstinence-based treatment programs focused on relapse to alcohol use among dependent individuals.^{4,5} Moderation-based treatment programs, however, have the additional challenge that acute alcohol consumption may exacerbate impulsivity leading to greater alcohol use during a drinking episode. For instance, many studies show increased impulsivity following alcohol administration,^{6–10} which is thought to potentially result from lower serotonergic function in vulnerable individuals.¹¹ Indeed, several studies have shown increased impulsivity after reduced serotonin synthesis,^{12–14} and that acute alcohol consumption can exacerbate this effect.¹⁵ Thus, those trying to moderate their drinking might fail to achieve their goal because alcohol consumption leads to increased impulsivity (i.e., increased impairment of inhibitory control), which in turn may lead to continued alcohol consumption during the drinking episode. Taken together, impulsivity may have important clinical implications, therefore targeting impulsive decision-making through pharmacological approaches (i.e., targeting low serotonin function) or by optimizing behavioral therapies to suit highly impulsive individuals may enhance treatment success.

Contingency management (CM) may be a promising behavioral therapy for alcohol use. CM interventions are based on the principle that alcohol and drug use are sensitive to systematically applied reinforcement.¹⁷ Typically, individuals earn incentives when objectively verified (i.e., urinary analysis or transdermal alcohol concentrations) treatment goals of abstinence or moderation of substance use are achieved.^{17–19} Compared to other behavioral interventions, CM has been shown to be particularly effective for reducing substance (cocaine) use in highly impulsive individuals such as those with anti-social personality disorder.¹⁶ CM may therefore be an optimal treatment for highly impulsive individuals as it may counteract impulsive decision-making in favor of substance use or continued use by offering immediate rewards for behaviors resulting in abstinence or reduced use. Although CM treatments have been shown to be efficacious in reducing substance use and improving abstinence rates for a variety of substances in both abstinence and moderation-based programs,^{18, 20–23} the relationship between impulsivity and CM treatment outcomes remains unclear.

The current study, by combining data from a large multi-phase project, aims to examine whether impulsivity predicts naturalistic drinking as well as treatment outcomes from a moderation-based CM intervention. We previously reported group based analyses from this project showing that the effects of alcohol consumption on impulsivity are dependent on the component of impulsivity being measured and the dose of alcohol consumed.²⁴ Although we found that impulsivity was not changed following low levels of alcohol consumption or serotonin depletion,²⁴ this does not preclude changes at the individual level, which may relate to individual responses to CM. We also reported that a 12-week moderation-based CM intervention reduced heavy drinking among at-risk drinkers.^{18,21} The current study extends these findings by combining data from the initial phase wherein impulsivity was measured by multiple tasks under multiple conditions (i.e., at baseline, after alcohol consumption, and after tryptophan depletion) and later phases that involved 28 days of naturalistic drinking and 12 weeks of moderation-based contingency management, to evaluate the ability of impulsivity to predict objectively measured naturalistic drinking and treatment outcomes.

Materials and Methods

Study Design

The current data analysis pulls information from all three phases of a large study that was divided into 3 phases: 1) Phase 1 involved 5 days of behavioral impulsivity testing; 2) Phase 2 involved 28 days of naturalistic drinking; and 3) Phase 3 involved 12-weeks of contingency management. See Figure 1 for a schematic of this design. We previously reported the laboratory results of Phase 1,²⁴ and the drinking results from Phases 2 and 3.^{18,21} The current analysis extends these findings by combining all three phases to determine whether the behavioral impulsivity testing in Phase 1 was predictive of either naturalistic drinking observed in Phase 2 or the Contingency Management outcomes observed in Phase 3.

Subjects and Criteria

Subjects ($n = 67$) were non-treatment seeking heavy drinkers recruited from the San Antonio area using TV, radio, online, and print advertisements. Potentially eligible participants, identified through a brief phone screen, were invited to the laboratory for additional screening that included: an alcohol consumption history (Alcohol Use Questionnaire),²⁵ a 28-day alcohol Timeline Followback interview (TLFB)²⁶, psychiatric screening (Structured Clinical Interview for DSM-IV-TR Axis I Disorders: Research Version, Non-Patient Edition),²⁷ intelligence screening,²⁸ urine-drug screen, pregnancy test and a medical history and physical examination. Participants who self-reported patterns of drinking that met or exceeded “at-risk” drinking criteria²⁹ (i.e., > 3 drinks for women or > 4 drinks for men on at least 3 occasions in the 28 days prior to the laboratory screening interview) were considered eligible for all three phases. Participants were considered ineligible for the following reasons: (1) drinking less than once per week; (2) smoking more than 1 pack of cigarettes per day; (3) having a current DSM-IV-TR Axis I psychiatric diagnosis; (4) an IQ less than 70; (5) a positive urinalysis for cocaine, opiates, methamphetamines, barbiturates, benzodiazepines, or THC; (6) pregnancy; (7) a medical condition that would be contraindicated for alcohol consumption; or (8) body mass index greater than 30.

Participants received \$70 for completing the screening visit. Written informed consent was obtained from all individuals, and the Institutional Review Board at The University of Texas Health Science Center at San Antonio reviewed and approved the experimental protocols.

Procedure

Phase 1: behavioral impulsivity testing—As described previously,²⁴ Phase 1 was a randomized, double-blind, placebo-controlled crossover design. Participants were required to complete one baseline performance day followed by four non-consecutive experimental days of testing, over which four different experimental conditions were counterbalanced across participants: (1) tryptophan depletion followed by alcohol; (2) tryptophan depletion followed by placebo; (3) tryptophan-balanced control followed by alcohol; and (4) tryptophan-balanced control followed by placebo. On each day of testing, participants completed a baseline behavioral impulsivity testing session (session 1). Participants were then given an amino-acid drink (tryptophan depletion or balanced control) and allowed twenty minutes to consume it. Using a multiple-dosing procedure, participants received an alcohol/placebo beverage at 5, 6, and 7 hours after consumption of the amino-acid drink to simulate an alcohol binge.³⁰ Ten minutes after each alcohol/placebo beverage was consumed, participants completed a behavioral impulsivity testing session. Participants received up to \$85 per visit for successfully completing each visit.

Behavioral impulsivity testing sessions—Each behavioral impulsivity testing session included three different tasks, all of which were completed at each of the 5 days of behavioral impulsivity testing sessions. As previously reported, performance across the multiple testing sessions was stable for each of the different tasks.²⁴

Immediate Memory Task (IMT): IMT uses responses to target and non-target 5-digit numbers, to measure “response initiation.”³¹ The two variables of interest were: (1) *correct detections*, correctly responding to a target stimulus; and (2) *commission errors*, responding to a catch stimulus (a 5-digit number that differs from the target stimulus by one digit). The primary dependent measure for this task was the IMT ratio (the proportion of commission errors to correct detections); higher ratios indicate greater response initiation impulsivity.

GoStop Impulsivity Paradigm (GoStop): The GoStop uses target and non-target 5-digit stimuli and color cues to measure response inhibition impulsivity.³² The primary dependent measure for this task was the GoStop Ratio [the proportion of inhibition failures (failure to inhibit responses to a target stimulus when a stop cue was delivered) relative to Go responses (correct response to a target stimulus when a go cue only was delivered)]; higher ratios indicate greater response inhibition impulsivity.

Single Key Impulsivity Paradigm (SKIP): The SKIP, a measure of consequence sensitivity, consists of a 20-minute period where participants are free to respond as often as desired by clicking a computer mouse to accumulate points.³³ The magnitude of the reward is related to the length of delay between two consecutive responses whereby the longer the delay the greater the reward. The primary dependent measure for this task was the longest latency (seconds) between two consecutive responses during the entire session.

Phase 2: naturalistic drinking—Following the completion of Phase 1, participants entered Phase 2. During the four weeks of the naturalistic drinking phase, participants wore continuously a Secure Continuous Remote Alcohol Monitor (SCRAM, Alcohol Monitoring Systems Inc. [AMS], Highlands Ranch, CO), which measures transdermal alcohol concentrations (TAC). Participants were required to attend the laboratory each week for approximately 30 minutes to transfer the TAC data from the SCRAM monitor and provide a self-report of alcohol consumption for the previous 7 day period using the TLFB.²⁶ During this phase participants were asked to drink alcohol as normal (i.e., no restrictions were placed upon alcohol consumption).

Phase 3: contingency management—Following the completion of Phase 2, participants continued into Phase 3 where they were required to continue to wear the SCRAM for a further 12 consecutive weeks and attend weekly clinic visits for TAC data downloads and self-reports of drinking using TLFB methods as described for Phase 2. In contrast to Phase 2, however, participants were informed that they would be paid an additional weekly bonus of \$50 for each week that they did not exceed a contingency criteria set to prevent heavy drinking. Those criteria required that on no day, did we observe 3 or more TAC readings above 0.03 g/dl as confirmed by Alcohol Monitoring Systems. This criterion equates to TAC readings resulting from heavy drinking episodes.³⁴

Data Analysis

Demographic characteristics (age, BMI) and alcohol use characteristics for the 28 days prior to study entry (average number of drinks per drinking day and number of at-risk drinking days) were compared between men and women using independent sample *t* tests. These statistical tests were conducted at a 2-sided significance level of 0.05.

A series of linear regression analyses were used to test the ability of Phase 1 laboratory measures of impulsivity to predict alcohol use measured in Phases 2 and 3. Separate linear models were used to test each of the three types of impulsivity (i.e., IMT ratio for response initiation, GoStop ratio for response inhibition, and SKIP longest latency for consequence sensitivity). Each of these types of impulsivity were examined under each of the three different contexts (i.e., at baseline, following alcohol, and following tryptophan depletion). For these analyses baseline impulsivity score was defined as the average of the scores of interest at session 1 (pre-drink session) across all four experimental laboratory days. Impulsivity following alcohol was defined as the peak change from baseline expressed as a difference between alcohol and the placebo control condition. Impulsivity scores following tryptophan depletion were defined as the peak changes from baseline expressed as a difference between depletion and non-depletion control conditions. Separate linear regression models were analyzed for each of the four alcohol use variables: percent days of any drinking in Phase 2; percent days of heavy drinking in Phase 2; change in percent days of any drinking from Phase 2 to Phase 3; change in percent days of heavy drinking from Phase 2 to Phase 3. Note that alcohol use variables were based solely on objectively-observed TAC data, not self-report. All linear regressions were tested with and without adjusting for sex as a covariate. Because Sex was not significant in any analyses, the results reported herein were all unadjusted for sex. Bonferroni corrections were applied to account

for multiple comparisons. All analyses were performed using Stata/SE (Version 13, College Station, TX).

Results

Participant Characteristics

A total of 67 (men = 41, women = 26) adults participated in all three phases. The majority of the participants were of white race (men = 53.7%, women = 65.4%) and of Hispanic ethnicity (men = 53.7%, women = 80.8%). Men and women did not differ on age ($M = 29.63$, $SD = 8.25$ and $M = 30.73$, $SD = 8.69$, respectively) or body mass index ($M = 26.41$, $SD = 3.09$ and $M = 26.25$, $SD = 4.16$, respectively). Participants showed a wide range of drinking, with the number of at-risk drinking days in the 28 days prior to study entry ranging from 3 to 25 days, however, there were no difference in the number of days between men and women ($M = 8.63$, $SD = 3.96$ and $M = 9.88$, $SD = 5.70$, respectively). There was a trend indicating that men ($M = 8.31$, $SD = 3.32$ drinks) drank more drinks per drinking day compared to women ($M = 6.65$, $SD = 3.01$), $t(1, 65) = -2.04$, $p = 0.05$.

Impulsivity as a Predictor of Alcohol Use

Table 1 shows descriptive statistics for each of the impulsivity measures (IMT, GoStop, and SKIP) measured at baseline, under acute alcohol consumption, and under tryptophan depletion as well as the observed drinking behavior (the percent of any days drinking and the percent of heavy days drinking) during phase 2 and the change in drinking behavior between phase 2 and 3 (the change in percent days of any drinking and the change in percent days of heavy drinking).

Naturalistic drinking—IMT ratio scores, GoStop ratio scores, and SKIP latency at baseline, under alcohol consumption or under tryptophan depletion did not significantly predict the percent days of drinking or the percent of heavy drinking days during Phase 2 (see Table 2).

Contingency management treatment outcomes—Although the results indicated that GoStop ratio scores under alcohol consumption predicted the change in the percent of days in which alcohol was consumed between Phase 2 and Phase 3, this was no longer significant after correcting for multiple comparisons. The analysis showed that IMT ratio scores, GoStop ratio scores, and SKIP latency at baseline, under alcohol consumption or under tryptophan depletion did not significantly predict the change in percent days of drinking or the percent of heavy drinking days between Phase 2 and 3 (see Table 3).

Discussion

We had hypothesized that impulsivity measured in the laboratory might predict naturalistic drinking or treatment outcomes from our moderation-based CM intervention. We examined multiple components of impulsivity (response initiation, response inhibition and consequence sensitivity), under different contexts (baseline, after alcohol consumption and after tryptophan depletion). The results demonstrated that impulsivity at baseline, after alcohol consumption or tryptophan depletion did not predict either naturalistic drinking or

treatment outcomes from a moderation-based CM treatment. Such findings were clear despite considerable variation in impulsive responding; demonstrated alcohol-induced increases in impulsivity; a wide range of drinkers; and objective measurements of alcohol use (i.e., TAC).

These findings, however, are inconsistent with the limited number of studies that have been conducted, which have typically shown that more impulsive individuals are more likely to have poorer alcohol-use treatment outcomes compared to less impulsive individuals^{4,5} but there are several possible explanations. The first is that existing research primarily relied on self-report personality measures to assess impulsivity yet in the current study we assessed behavioral impulsivity and did so under different contexts. Previous research shows that personality measures and behavioral measures are not always correlated.³⁵ Indeed, a recent meta-analysis suggested that self-report personality measures of impulsivity and behavioral measures of impulsivity accounted for only a small amount of variance for similar underlying constructs,³⁶ and so they may be measuring conceptually different factors. Additionally, given the multi-faceted nature of impulsivity³⁷ and the many different approaches to impulsivity assessment, it is possible that other components of behavioral impulsivity which were not assessed in the current study such as delay discounting or waiting impulsivity, or different assessments of similar constructs may predict CM treatment outcomes.

Furthermore, previous research examined whether impulsivity predicted treatment outcomes in samples of adults with alcohol use disorders^{4,5} but few in the current study met clinical diagnostic criteria for an alcohol use disorder. Despite having adults with a range of drinking patterns and considerable variability with regards to impulsivity scores, it is possible that impulsivity may only be a predictor of alcohol use treatment outcomes among clinical populations. Indeed, both higher self-report and behavioral impulsivity is typically associated with more severe alcohol problems and alcohol use disorders.^{2,3}

To our knowledge this is the first study to examine impulsivity as a predictor of treatment outcomes with regards to contingency management as the mode of treatment. Therefore, it is possible that the effects of impulsivity on alcohol use treatment success are potentially dependent on the mode of treatment and so CM may prove to be the preferred intervention for highly impulsive individuals. Indeed, as highlighted previously, CM compared to other behavioral interventions has been shown to effectively reduce cocaine use in highly impulsive individuals such as those with anti-social personality disorder.¹⁶ The results of the present study, however, also showed that the multiple components of behavioral impulsivity measured under different contexts failed to predict naturalistic alcohol consumption among heavy drinkers, suggesting that impulsivity, as measured in the current study, would therefore unlikely influence treatment outcome irrespective of the mode of treatment, at least in such samples.

The finding that impulsivity did not predict either naturalistic drinking or CM treatment outcomes could be positive as it means that other factors, which are potentially more malleable to change (i.e., self-efficacy), could be leveraged to improve CM treatment success. Research examining the extent to which impulsivity may predict treatment success

and therefore be utilized to improve treatment outcomes is still in the early stages and more research is required before more definitive conclusions can be drawn. Future research should look to concurrently examine multiple components of impulsivity using both self-report personality measures and behavioral measures of impulsivity, as well as incorporating components not measured in the current study. In addition, such research should be conducted in both clinical and non-clinical samples.

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References

1. de Wit H. Impulsivity as a determinant consequence of drug use: a review of underlying processes. *Addict Biol.* 2009; 14:22–31. [PubMed: 18855805]
2. Verdejo-Garcia A, Lawrence AJ, Clark L. Impulsivity as a vulnerability marker for substance use disorders: review of findings from high risk research, problem gamblers and genetic association studies. *Neurosci Biobehav R.* 2008; 32:777–810.
3. Poikolainen K. Risk factors for alcohol dependence: A case-control study. *Alcohol Alcoholism.* 2000; 35:190–196. [PubMed: 10787396]
4. Loree AM, Lundahl LH, Ledgerwood DM. Impulsivity as a predictor of treatment outcome in substance use disorders: Review and synthesis. *Drug Alcohol Rev.* 2015; 34:119–134. [PubMed: 24684591]
5. Stevens L, Verdejo-Garcia A, Goudriaan AE, et al. Impulsivity as a vulnerability factor for poor addiction treatment outcomes: A review of neurocognitive findings among individuals with substance use disorders. *J Subst Abuse Treat.* 2014; 47:58–72. [PubMed: 24629886]
6. Abroms BD, Fillmore MT, Marczinski CA. Alcohol-induced impairment of behavioral control: effects on the alteration and suppression of pre-potent responses. *J Stud Alcohol Drugs.* 2003; 64:687–695.
7. Dougherty DM, Marsh-Richard DM, Hatzis ES, et al. A test of alcohol dose effects on multiple behavioral measures of impulsivity. *Drug Alcohol Depen.* 2008; 96:111–120.
8. Dougherty DM, Marsh DM, Moeller FG, et al. Effects of moderate and high doses of alcohol on attention, impulsivity, discriminability, and response bias in immediate and delayed memory task performance. *Alcohol Clin Exp Res.* 2000; 24:1702–1711. [PubMed: 11104118]
9. Dougherty DM, Moeller FG, Steinberg JL, et al. Alcohol increases commission error rates for a continuous performance test. *Alcohol Clin Exp Res.* 1999; 23:1342–1351. [PubMed: 10470977]
10. Marczinski CA, Combs SW, Fillmore MT. Increased sensitivity to the disinhibiting effects of alcohol in binge drinkers. *Psychol Addict Behav.* 2007; 21:346–354. [PubMed: 17874885]
11. Pattij T, Vanderschuren LJ. The neuropharmacology of impulsive behaviour. *Trends Pharmacol Science.* 2008; 29:192–199.
12. Crean J, Richards JB, de Wit H. Effect of tryptophan depletion on impulsive behavior in men with or without a family history of alcoholism. *Behav Brain Res.* 2002; 136:349–357.
13. Schweighofer N, Bertin M, Shishida K. Low-serotonin levels increase delayed reward discounting in humans. *J Neurosci.* 2008; 28:4528–4532. [PubMed: 18434531]
14. Waulderhaug E, Landrø NI, Magnusson A. Lowering of serotonin by rapid tryptophan depletion increases impulsiveness in normal individuals. *Psychopharmacology.* 2002; 164:385–391. [PubMed: 12457268]

15. Dougherty DM, Marsh DM, Mathias CW, et al. The effects of alcohol on laboratory-measured impulsivity after L-tryptophan depletion or loading. *Psychopharmacology*. 2007; 193:137–150. [PubMed: 17377773]
16. Messina N, Farabee D, Rawson R. Treatment responsivity of cocaine-dependent patients with antisocial personality disorder to cognitive behavioral interventions and contingency management interventions. *J Consult Clin Psych*. 2003; 71:320–329.
17. Higgins ST, Budney AJ, Bickel WK, et al. Incentives improve outcome in outpatient behavioral treatment of cocaine dependence. *Arch Gen Psychiat*. 1994; 51:568–576. [PubMed: 8031230]
18. Dougherty DM, Karns TE, Mullen J, et al. Transdermal alcohol concentration data collected during a contingency management program to reduce at-risk drinking. *Drug Alcohol Depen*. 2015; 148:77–84.
19. Stitzer M, Petry N. Contingency management for the treatment of substance abuse. *Annu Rev Clin Psycho*. 2006; 2:411–434.
20. Barnett NP, Tidey J, Murphy JG, et al. Contingency management for alcohol use reduction: a pilot study using a transdermal alcohol sensor. *Drug Alcohol Depen*. 2011; 118:391–399.
21. Dougherty DM, Lake SL, Hill-Kapturczak N, et al. Using contingency management procedures to reduce at-risk drinking in heavy drinkers. *Alcohol Clin Exp Res*. 2015; 4:743–751.
22. Lussier JP, Heil SH, Mongeon JA, et al. A meta-analysis of voucher-based reinforcement therapy for substance use disorders. *Addiction*. 2006; 101:192–203. [PubMed: 16445548]
23. Prendergast M, Podus D, Finney J, et al. Contingency management for treatment of substance use disorders: A met-analysis. *Addiction*. 2006; 101:1546–1560. [PubMed: 17034434]
24. Dougherty DM, Mullen J, Hill-Kapturczak N, et al. Effects of tryptophan depletion and a simulated alcohol binge on impulsivity. *Exp Clin Psychopharm*. 2015; 23:109–121.
25. Mehrabian A, Russell JA. A questionnaire measure of habitual alcohol use. *PsycholRep*. 1978; 43:803–806.
26. Sobell, LC.; Sobell, MB. Timeline follow-back: A technique for assessing self-reported alcohol consumption. Humana Press; 1992.
27. First, MB.; Spitzer, RL.; Gibbon, M., et al. Structured Clinical Interview for DSM-IV-TR Axis I Disorders, Research Version, Non-patient Edition (SCID-I/NP). New York: Biometrics Research, New York State Psychiatric Institute; 1992.
28. Wechsler, D. Wechsler Abbreviated Scale of Intelligence (WASI). London: Psychological Corporation; 1999.
29. National Institute on Alcohol Abuse and Alcoholism (NIAAA). Washington, DC: 2010. Rethinking Drinking: U.S. Department of Health and Human Services. NIH Publication No. 13-3770
30. National Institute on Alcohol Abuse and Alcoholism (NIAAA). National Institute of Alcohol Abuse and Alcoholism Council Approves Definition of Binge Drinking. 2004 NIAAA Newsletter, No. 3.
31. Dougherty DM, Marsh DM, Mathias CW. Immediate and delayed memory tasks: A computerized behavioral measure of memory, attention, and impulsivity. *Behav Res Meth Instr*. 2002; 34:391–398.
32. Dougherty, DM.; Mathias, CW.; Marsh, DM. GoStop Impulsivity Paradigm (Version 1.0). Houston, TX: 2003.
33. Dougherty DM, Marsh DM, Mathias CW, et al. Bipolar disorder and substance use: The conceptualization and role of impulsivity. *Psychiatric Times*. 2005; 22:32–35.
34. Roache JD, Karns T, Hill-Kapturczak, et al. Using transdermal alcohol monitoring to detect low-level drinking. *Alcohol Clin Exp Res*. 2015; 39:1120–1127. [PubMed: 25988708]
35. Reynolds B, Ortengren A, Richards JB, et al. Dimensions of impulsive behavior: Personality and behavioral measures. *Personality and Individual Differences*. 2006; 40:305–315.
36. Cyders MA, Coskunpinar A. Measurement constructs using self-report and behavioral lab tasks: is there overlap in nomothetic span and construct representation for impulsivity? *Clin Psychol Rev*. 2011; 31:965–982. [PubMed: 21733491]

37. Dougherty DM, Mathias CW, Marsh DM, et al. Laboratory behavioral measures of impulsivity. *Behav Res Methods*. 2005; 37:82–90. [PubMed: 16097347]

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Figure 1.
Study design illustration

Table 1**Descriptive Statistics of Impulsive Responding and Drinking Behavior**

	Men (<i>n</i> = 41) M (SD)	Women (<i>n</i> = 26) M (SD)
Impulsivity Measures		
GoStop		
Baseline	0.20 (0.19)	0.17 (0.14)
Alcohol	0.04 (0.23)	0.07 (0.17)
Tryptophan Depletion	0.02 (0.18)	−0.00 (0.23)
IMT		
Baseline	0.26 (0.14)	0.18 (0.12)
Alcohol	0.02 (0.11)	0.04 (0.07)
Tryptophan Depletion	−0.01 (0.10)	−0.00 (0.11)
SKIP		
Baseline	504 (79.1)	442 (121)
Alcohol	13.6 (133)	−44.1 (243)
Tryptophan Depletion	32.9 (124)	−35.1 (188)
	%	%
Drinking Behavior		
Phase 2		
Avg % Any Drinking Days	69.7	70.2
Avg % Heavy Drinking Days	49.1	49.0
Phase 3		
Avg % Any Drinking Days	50.8	49.5
Avg % Heavy Drinking Days	22.7	17.6

Note. Baseline refers to the average of the scores of interest at session 1 (pre-drink session) across all four experimental laboratory days. Alcohol and Tryptophan Depletion refers to the peak change from baseline expressed as a difference between the experimental condition (alcohol or tryptophan depletion) and the corresponding placebo control condition.

Table 2

Regression model results examining impulsivity as a predictor of naturalistic drinking

	β	R^2	F	95% CI	p
% Days of Any Drinking					
GoStop					
Baseline	0.19	0.03	1.94	-0.81 – 0.45	0.17
Alcohol	0.13	0.02	1.35	-0.09 – 0.36	0.25
Tryptophan Depletion	-0.12	0.02	1.09	-0.35 – 0.11	0.30
IMT					
Baseline	-0.01	0.00	0.00	-0.35 – 0.34	0.97
Alcohol	-0.31	0.02	1.60	-0.80 – 0.17	0.21
Tryptophan Depletion	-0.19	0.01	0.75	-0.63 – 0.25	0.39
SKIP					
Baseline	-0.00	0.04	2.63	-0.00 – 0.00	0.11
Alcohol	-0.00	0.00	0.13	-0.00 – 0.00	0.72
Tryptophan Depletion	0.00	0.00	0.00	-0.00 – 0.00	0.99
% Days of Heavy Drinking					
GoStop					
Baseline	0.19	0.01	1.67	-0.10 – 0.48	0.20
Alcohol	0.97	0.00	0.62	-0.15 – 0.34	0.43
Tryptophan Depletion	-0.05	0.00	0.14	-0.30 – 0.20	0.71
IMT					
Baseline	-0.01	0.00	0.00	-0.38 – 0.36	0.96
Alcohol	-0.24	0.01	0.78	-0.77 – 0.30	0.38
Tryptophan Depletion	0.11	0.00	0.00	-0.47 – 0.49	0.96
SKIP					
Baseline	-0.00	0.02	1.00	-0.00 – 0.00	0.32
Alcohol	-0.00	0.00	0.27	-0.00 – 0.00	0.60
Tryptophan Depletion	-0.00	0.01	0.60	-0.00 – 0.00	0.44

Note. Baseline refers to the average of the scores of interest at session 1 (pre-drink session) across all four experimental laboratory days. Alcohol and Tryptophan Depletion refers to the peak change from baseline expressed as a difference between the experimental condition (alcohol or tryptophan depletion) and the corresponding placebo control condition.

Table 3

Regression model results examining impulsivity as a predictor of change in drinking behavior from Phase 3 to Phase 2

	β	R^2	F	95% CI	p
% Days of Any Drinking					
GoStop					
Baseline	0.16	0.02	1.49	-0.10 – 0.41	0.23
Alcohol	-0.31	0.12	8.38	-0.52 – -0.96	0.005
Tryptophan Depletion	0.04	0.00	0.09	-0.20 – 0.27	0.76
IMT					
Baseline	-0.02	0.00	0.01	-0.35 – 0.32	0.93
Alcohol	0.16	0.00	0.46	-0.31 – 0.64	0.50
Tryptophan Depletion	0.13	0.01	0.35	-0.30 – 0.55	0.56
SKIP					
Baseline	0.00	0.01	0.80	-0.00 – 0.00	0.37
Alcohol	-0.00	0.00	0.20	-0.00 – 0.00	0.65
Tryptophan Depletion	-0.00	0.01	0.88	-0.00 – 0.00	0.35
% Days of Heavy Drinking					
GoStop					
Baseline	0.07	0.00	0.39	-0.17 – 0.32	0.53
Alcohol	-0.13	0.03	1.61	-0.33 – 0.07	0.21
Tryptophan Depletion	0.12	0.02	1.21	-0.09 – 0.32	0.28
IMT					
Baseline	0.01	0.00	0.00	-0.30 – 0.32	0.95
Alcohol	0.00	0.00	0.00	-0.44 – 0.45	0.99
Tryptophan Depletion	-0.04	0.00	0.04	-0.44 – 0.36	0.85
SKIP					
Baseline	0.00	0.00	0.18	-0.00 – 0.00	0.67
Alcohol	-0.00	0.00	0.62	-0.00 – 0.00	0.43
Tryptophan Depletion	-0.00	0.00	0.10	-0.00 – 0.00	0.75

Note. Baseline refers to the average of the scores of interest at session 1 (pre-drink session) across all four experimental laboratory days. Alcohol and Tryptophan Depletion refers to the peak change from baseline expressed as a difference between the experimental condition (alcohol or tryptophan depletion) and the corresponding placebo control condition.