

**The prospective association of negative urgency with hazardous drinking via  
impaired control: A moderating role of alcohol sensitivity**

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## Abstract

**Objective:** Trait negative urgency is consistently associated with alcohol problems, and cross-sectional findings have suggested a mediational role of impaired control over alcohol. Initial evidence also suggests that individual differences in self-reported sensitivity to alcohol's effects may moderate the association between urgency and alcohol outcomes. The aim of this study was to replicate and extend these findings using prospective data. **Method:** Young adult drinkers (N=159, mean age=18.87, *SD*=1.16; 70.4% female) from Montreal, Quebec, completed an online survey at baseline and again six months later. Participants completed self-report measures of negative urgency, alcohol sensitivity, impaired control over alcohol and hazardous drinking. **Results:** Moderated mediation analyses revealed that the prospective indirect association between negative urgency at baseline and hazardous drinking at follow-up mediated via increased impaired control at follow-up was statistically significant only for young adults who reported relatively low alcohol sensitivity at baseline. The moderating impact of alcohol sensitivity on this indirect pathway was driven primarily by a significant interaction between baseline alcohol sensitivity and impaired control at follow-up in the prediction of hazardous drinking at follow-up; the interaction between baseline alcohol sensitivity and baseline negative urgency predicting impaired control at follow-up was not statistically significant. **Conclusions:** Using prospective data from a unique sample of young adults, the present study partially replicates prior cross-sectional findings suggesting that the indirect association between urgency and hazardous drinking via impaired control over alcohol is moderated by alcohol sensitivity.

**Keywords:** Urgency, subjective response to alcohol, impulsivity, impaired control, alcohol problems

## Introduction

Impulsive traits are consistently associated with hazardous drinking (Adan et al., 2017). Of several facets comprising the impulsivity construct, negative urgency (i.e., the tendency to engage in rash behaviors in response to negative affect; Whiteside et al., 2005) has shown the strongest associations with alcohol problems and dependence (Coskunpinar et al., 2013). Thus, it is important to elucidate potential mechanisms of this association. Recent findings suggest that impaired control over alcohol (i.e., diminished ability to abstain or reduce alcohol intake; Heather et al., 1993) may play a role in the association between negative urgency and problem drinking. As a cardinal symptom of alcohol use disorder (AUD), impaired control has typically been studied in individuals with AUD. However, emerging evidence supports the relevance of impaired control in youth as an early sign of alcohol-related problems that may develop, in part, as a function of impulsive traits (Leeman et al., 2009, 2012, 2014). Previous cross-sectional studies support this notion, finding that the association between impulsive traits and problem drinking is partly mediated by impaired control (Patock-Peckham et al., 2018; Wardell et al., 2015; Wardell et al., 2016). Importantly, impaired control is more strongly associated with urgency (Wardell et al., 2016) – especially negative urgency (McCarty et al., 2017) – than with other facets of impulsivity. Conceptually, when individuals high on negative urgency drink in the context of negative mood, they may do so in a rash or impulsive manner, which could impair their ability to control their drinking, in turn leading to negative alcohol outcomes (see Wardell et al., 2015). Thus, impaired control may be a particularly relevant mechanism in the link between negative urgency and problem drinking.

Further, individual difference in alcohol sensitivity – an early indicator of heavy drinking risk (Morean & Corbin, 2010; King et al., 2011) – may be an important moderator of this pathway. We previously found that lower self-reported sensitivity to alcohol's

sedative/intoxicating effects strengthened both links in the indirect pathway from negative urgency to impaired control to alcohol problems (Wardell et al., 2015). Perhaps increased negative urgency may be more likely to result in impaired control for individuals with relatively low alcohol sensitivity, as they are potentially less aware of the internal warning signs of intoxication, and thus may be more likely to drink “too much” (i.e., impaired control) when drinking in a negative mood state (see Wardell et al., 2015). Consequently, individuals low on alcohol sensitivity may achieve a higher blood alcohol concentration (BAC) before they perceive that too much alcohol has been consumed, leading to greater alcohol-related negative outcomes as a function of impaired control.

Although existing evidence suggests the relevance of impaired control as a developmental antecedent mediating the effect of negative urgency on hazardous drinking, there are no prospective studies examining this indirect pathway. The goal of the present study was replicate and extend our prior cross-sectional findings (i.e., Wardell et al., 2015) using prospective data from a distinct sample of youth. We hypothesized that the prospective indirect relationship between negative urgency and hazardous drinking mediated via impaired control would be stronger for youth reporting relatively lower sensitivity to alcohol.

## **Method**

### **Participants and Procedures**

Participants were 159 young adults (mean age=18.87,  $SD=1.16$ , range=18-25; 70.4% females) from Montreal, Quebec, Canada, who completed an online baseline survey. All participants reported prior alcohol use and were above the legal drinking age of 18 years old in Quebec. Participants were recruited from local area Collège d'enseignement général et professionnel (CEGEP), a level of education unique to Quebec that follows high school and offers both technical and pre-university programs. The recruitment of participants from this

unique educational context, and from the Montreal area, resulted in a sample distinct from prior studies on impaired control and youth drinking. Participants were recruited from predominantly English-language schools, with ( $n=119$ , 75.8%) participants reporting English as their primary language. Participants reported their racial/ethnic background as follows: Caucasian/White ( $n=84$ , 52.8%), Asian ( $n=38$ , 23.9%), Middle Eastern ( $n=17$ , 10.7%), South Asian ( $n=12$ , 7.5%), Hispanic/Latino ( $n=10$ , 6.3%), Black/African American ( $n=6$ , 3.8%) and other ( $n=5$ , 3.1%).

Those participants who consented to follow-up contact were sent a follow-up online survey 6 months post-baseline (retention rate: 83.65%). Participants were compensated with a \$20 gift card for completing each survey (baseline and follow-up). Procedures were approved by the Concordia University Human Research Ethics Committee.

## Measures

**Negative Urgency (Lynam et al., 2006).** The negative urgency subscale of the UPPS-P (Lynam et al., 2006) contains 12-items measuring the tendency to act rashly under negative affective states (e.g., ‘When I feel bad, I will often do things I later regret in order to make myself feel better now’). The negative urgency scale showed good internal consistency ( $\alpha=.84$ ) in the present sample.

**Self-Rating of the Effects of Alcohol (SRE; Schuckit et al., 1997).** The SRE assesses how many drinks a person needs to feel different effects of alcohol intoxication (e.g., dizziness/slurred speech, stumbling/loss of coordination). The “first five” subscale, which is presumed to index innate (versus acquired) differences in alcohol sensitivity by focusing on the first five earliest drinking episodes, was used for the present analyses. Higher SRE scores indicate lower alcohol sensitivity (i.e., more drinks needed to feel the effects). As some items are not applicable to participants who never experienced the effect during the first five

drinking occasions, standardized person-mean imputation was performed for items that were not endorsed (see Lee et al., 2015). Due to sex differences in alcohol sensitivity, this variable was standardized separately in males and females.

**Impaired Control Scale (ICS; Heather et al., 1993).** The ICS assesses impairments in one's ability to limit alcohol use (Heather et al., 1993). Given the focus on a young adult sample, Part 3 of the ICS (perceived control, 10 items) was used to assess anticipated difficulty controlling alcohol intake in different situations, which showed good internal consistency at both assessments ( $\alpha=.80$  and  $.81$ , respectively).

**Alcohol Use Disorder Identification Test (AUDIT; Saunders et al., 1993).** The AUDIT was used to assess hazardous drinking in the past six months. This 10-item questionnaire assesses alcohol quantity/frequency, alcohol-related problems, and AUD symptoms. In the current sample, internal consistency was good at baseline ( $\alpha=.81$ ) and follow-up ( $\alpha=.85$ ).

### **Data Analysis**

Descriptive and correlation analyses were performed in order to examine the sample characteristics, variable distributions, and bivariate associations between variables. Four extreme outliers (i.e., z-scores greater than 3.29) were recoded to one unit greater than the next most extreme value (Tabachnick & Fidell, 2007).

To examine the hypothesized associations, a prospective moderated mediation model was specified using Mplus v.8 (Muthén & Muthén, 2017). Negative urgency at baseline (T1) was specified as the predictor variable, and impaired control over alcohol and AUDIT at six months (T2) were specified as the mediator and outcome, respectively (controlling for baseline levels of each variable). Alcohol sensitivity at T1 was specified as a moderator of

both links in this indirect pathway by including the interactions between T1 negative urgency and T1 alcohol sensitivity predicting T2 impaired control, and between T2 impaired control and T1 alcohol sensitivity predicting T2 AUDIT. Variables were standardized prior to creating interaction terms.

For the few cases ( $n=12$ ) with partially complete data on a measure, the total score for the measure was treated as missing. Twenty-six (16.35%) participants had no data on either ICS or AUDIT at T2 (i.e., lost to follow-up). Another 11 participants (8.27%) provided T2 AUDIT data but were missing T2 ICS data. Participants with missing data were retained in the analysis using full information maximum likelihood estimation (total  $N=159$ ). The robust estimator was used to adjust standard errors for violations of the assumption of normality. Bootstrapping was used to derive bias-corrected 95% confidence intervals for indirect paths.

Interactions were probed by examining conditional direct and indirect associations at low ( $-1SD$ ), average (mean) and high ( $+1SD$ ) levels of alcohol sensitivity using Stride's codes (Stride et al., 2015). Good model fit was supported by the root-mean square error of approximation (RMSEA)  $\leq .08$ , comparative fit index (CFI)  $\geq .95$  and Standardized Root Mean Square Residual (SRMSR)  $\leq .05$ . Fully- vs. partially-mediated models were compared by examining differences in Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC).

## Results

Descriptive statistics and bivariate correlations among the study variables are shown in Table 1. Participants who completed all measures at follow-up reported lower baseline AUDIT scores ( $M=5.15$ ,  $SD=4.44$ ) than those with missing data at follow-up ( $M=7.24$ ,  $SD = 5.48$ ;  $t(156)=-2.09$ ,  $p=.038$ ). No other variables differed significantly for those with vs.

without missing follow-up data ( $p > .05$ ). There were no sex ( $p$ s ranged from .067-.806) or race ( $p$ s ranged from .061-.999 for white vs. non-white) differences on any variable.

We first compared the fit of a fully mediated model versus a partially mediated model (i.e., including direct paths from predictors to AUDIT). The fully mediated model (AIC=3150.923, BIC=3270.611) provided better fit to the data than the partially mediated model (AIC =3154.431, BIC=3280.256), and none of the direct paths from negative urgency, alcohol sensitivity, or their interaction were significant predictors of T2 AUDIT ( $p$ s ranged .281-.936). Thus, the fully mediated model was used for the subsequent analyses, and provided good fit to the data,  $\chi^2(5)=9.41, p=.094$ ; RMSEA=.074, CFI=.953, SRMR=.031.

Figure 1 shows the model results. Contrary to the hypothesis, the interaction between T1 negative urgency and T1 alcohol sensitivity predicting T2 impaired control was not statistically significant ( $p=.138$ ), and the lower-order prospective association between T1 negative urgency and T2 impaired control (conditioned on average levels of alcohol sensitivity) was not significant ( $p=.065$ ). However, consistent with expectations, the interaction between T1 alcohol sensitivity and T2 impaired control predicting T2 AUDIT was statistically significant when controlling for T1 AUDIT ( $p=.035$ ; see Figure 1). Follow-up analyses revealed that elevated impaired control at T2 predicted AUDIT at T2 when the model was conditioned on low ( $\beta=0.35, SE=0.14, p=.009$ ) and moderate ( $\beta=0.27, SE=0.10, p=.004$ ) levels of alcohol sensitivity (i.e., higher SRE score), but not high alcohol sensitivity ( $\beta=0.20, SE=0.11, p=.071$ ).

Given that alcohol sensitivity was a significant moderator of one of the links in the indirect pathway, we proceeded to probe the significance of the indirect association conditioned on high, moderate, and low levels of alcohol sensitivity. The indirect association between T1 negative urgency and T2 AUDIT mediated through T2 impaired control was



statistically significant among participants reporting relatively low alcohol sensitivity (estimate=0.10, 95% CI [0.01, 0.27]), but not among those reporting moderate (estimate=0.04, 95% CI [0.000, 0.13]) or high alcohol sensitivity (estimate=0.01, 95% CI [-0.01, 0.08]).

Finally, as the AUDIT includes an item that assesses impaired control over alcohol (item 4), we re-analyzed the data after excluding this item from the AUDIT score to examine how this impacted the results. However, doing so did not lead to any substantive changes in the findings, and the statistical significance of all interactions and indirect associations remained unchanged.

## Discussion

This study is the first to examine the prospective indirect association between negative urgency and hazardous drinking via impaired control over alcohol. We aimed to replicate our previous cross-sectional finding that alcohol sensitivity moderates this indirect pathway (Wardell et al., 2015) using prospective data from a unique sample of young adults. Our hypotheses were partially supported; although alcohol sensitivity did not moderate the prospective association between negative urgency at baseline (T1) and impaired control at 6-month follow-up (T2), alcohol sensitivity did moderate the effect of T2 impaired control on T2 hazardous drinking. Further, as hypothesized, the prospective indirect association between higher negative urgency at T1 and greater hazardous drinking at T2 mediated through increased impaired control at T2 was significant only for participants with relatively low levels of self-reported alcohol sensitivity at baseline.

Consistent with our prior work (Wardell et al., 2015), lower alcohol sensitivity appeared to strengthen the association between impaired control and hazardous drinking. Because those with low alcohol sensitivity can drink more alcohol before feeling the effects

of intoxication, they may have a higher BAC threshold for perceiving that they have had “too much” alcohol, and so the experience of impaired control may emerge at relatively higher BACs for these individuals. This, in turn, may strengthen the association between impaired control and hazardous drinking outcomes for these individuals. However, as the mechanism explaining the interaction between alcohol sensitivity and impaired control could not be tested directly in this study, this remains speculative and should be further investigated in future studies.

We found some support in our prospective analysis for an indirect effect of negative urgency on hazardous drinking mediated via impaired control, consistent with prior cross-sectional studies (Patock-Peckham et al., 2018; Wardell, et al., 2016). However, as previously observed in cross-sectional analyses (Wardell et al., 2015), this prospective indirect effect was qualified by level of alcohol sensitivity, such that negative urgency only predicted hazardous drinking via impaired control among individuals with relatively lower levels of alcohol sensitivity. These findings suggest that increased impaired control over alcohol may be one mechanism that helps to explain the effect of negative urgency on hazardous drinking among young adults, particularly among those who are relatively low on sensitivity to sedative or impairing alcohol effects. However, this finding must be interpreted with the caveat that the interaction between negative urgency and alcohol sensitivity at T1 did not reach statistical significance in the prospective prediction of increased impaired control at T2. This may be due, in part, to the short duration of the follow-up period (6 months). Further, the small sample size limited our power to detect interactions, which tend to have smaller effect sizes relative to main effects.

Some limitations should be considered when interpreting these findings. Although we were able to examine the prospective association between negative urgency and impaired control, impaired control and AUDIT were assessed concurrently. Future studies should

perform a fully longitudinal mediation analysis with three waves of data. Also, as there were no formal validity checks in the online survey, we cannot rule out potential sources of bias associated with web-based self-report methods. Further, relying on retrospective self-report to assess alcohol sensitivity is another limitation, as is the use of an instrument that focuses primarily on sedative/impairing effects and does not include sensitivity to alcohol's stimulant effects. Moreover, the study was not powered to detect small effect sizes, and the follow-up period was limited to six months. In addition, although negative urgency is among the most relevant facets of impulsivity for predicting impaired control and hazardous drinking (Coskunpinar et al., 2013, McCarty et al., 2017, Wardell et al., 2015), larger prospective studies with greater power to examine multiple simultaneous pathways involving several facets of impulsivity are needed in order to isolate the unique contribution of negative urgency. Finally, the sample was comprised of young adults in Quebec, the majority of whom were female and White, limiting generalizability to other populations.

In summary, the present study partially replicates prior cross-sectional findings, supporting a moderating role of alcohol sensitivity in the prospective indirect association between negative urgency and hazardous drinking via impaired control. We encourage future studies in larger, more heterogeneous samples with longer follow-up periods and additional measures of alcohol sensitivity to further clarify these prospective relationships.

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**Table 1**

Means, standard deviations, observed ranges, and correlations among variables in the models.

	1	2	3	4	5	6	Mean	SD	Min-Max
1. Negative urgency (T1)	1						26.98	6.92	12 - 46
2. Alcohol sensitivity (T1)	.062	1					2.95	2.25	0 - 9.75
3. Impaired Control (T1)	.379**	.189*	1				18.50	6.93	9 - 37
4. Impaired Control (T2)	.252**	.069	.483**	1			19.28	7.29	10 - 37
5. AUDIT (T1)	.269**	.504**	.325**	.302**	1		5.47	4.66	0 - 21
6. AUDIT (T2)	.213*	.407**	.260**	.443**	.666**	1	4.63	4.78	0 - 27

*Notes.* T1 = Time 1 (baseline), T2 = Time 2 (six month follow-up), AUDIT= Alcohol Use Disorder Identification Test.

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

### Figure Legend

Figure 1. Final model of the indirect pathway between negative urgency and hazardous drinking via impaired control over alcohol, with alcohol sensitivity as a moderator. Solid lines depict significant associations and dashed lines depict nonsignificant associations. Standardized coefficients (with standard errors in parentheses) are shown. Covariances among all exogenous variables were freely estimated but were omitted from the figure.

T1 = Time 1 (baseline), T2 = Time 2 (six month follow-up), AUDIT = Alcohol Use Disorder

Identification Test

†  $p < .10$ ; \*  $p < .05$ ; \*\*  $p < .01$



