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Pain catastrophizing predicts alcohol craving in heavy drinkers independent of pain intensity

Steven J. Nieto [a], ReJoyce Green [a], Erica N. Grodin [a], Catherine M. Cahill [b], Lara A. Ray [a,b,*]

[a] Department of Psychology, University of California at Los Angeles, Los Angeles, CA, United States
[b] Department of Psychiatry and Biobehavioral Sciences, University of California at Los Angeles, Los Angeles, CA, United States

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ABSTRACT

Background: Chronic pain and alcohol use disorder (AUD) are often co-occurring conditions. Pain catastrophizing, an emotional component of pain, and pain intensity are related to alcohol use as a coping mechanism; however, how pain interacts with tonic alcohol craving is an understudied area. This study sought to determine the unique and independent effects of pain intensity and pain catastrophizing on alcohol craving in heavy drinkers.

Method: Non-treatment seeking heavy drinkers ($n = 128$) completed self-report measures of pain (both intensity and catastrophizing), depression, alcohol use and problems, and reasons for heavy drinking. A hierarchical regression examined the unique contribution of pain intensity to alcohol craving. Depression, pain catastrophizing, and alcohol use measures were added to the hierarchical model in sequential blocks.

Results: The final model of the hierarchical regression demonstrated that pain catastrophizing has an independent effect on alcohol craving over and above demographic, pain intensity, depression, and alcohol measures. Exploratory analyses suggest that individuals in the high intensity pain grade have higher levels of depression symptomology, pain catastrophizing, alcohol use and problems, as well as engaging in heavy drinking to "feel normal" compared to the no pain and low intensity pain grades.

Conclusions: These results demonstrate that pain catastrophizing predicts alcohol craving independent of self-reported chronic pain intensity. Individuals with high intensity chronic pain have more severe alcohol use and mood-related symptomology. Upon replication in clinical samples, these findings can inform clinical care for pain management.

1. Introduction

Pain and problematic alcohol consumption frequently co-occur in adults and have a bi-directional relationship. That is, problematic alcohol use can exacerbate pain while pain experiences can escalate alcohol consumption (Edwards et al., 2020). Individuals who experience chronic pain (i.e. subjective pain lasting longer than 3 months) report higher levels of alcohol use and are more likely to have an alcohol use disorder (AUD) relative to the general population (Hoffmann et al., 1995; Vowles et al., 2018). Among treatment-seeking individuals with AUD, 30−50% report chronic, recurrent pain (Boissoneault et al., 2017; Caldeiro et al., 2008; Jakubczyk et al., 2015) and those who do experience pain spend fewer days in AUD treatment and have a lower likelihood of abstinence from alcohol after treatment (Jakubczyk et al., 2016; Witkiewitz et al., 2015). While the association between pain and alcohol use is complex, how pain interacts with other proximal predictors of alcohol consumption (i.e. alcohol craving) is an understudied but promising area.

Addiction theories suggest that people drink alcohol for positive and negative reinforcement (DeMartini and Carey, 2011; Grodin et al., 2019; Kuntsche et al., 2006), to satisfy urges/craving (Anton et al., 1996), out of habit (Vollstädt-Klein et al., 2010), or to feel normal (Koob, 2003). Among these motives, negative reinforcement models of alcohol use posit that alcohol consumption is maintained in order to remove, or to lessen, the impact of negative emotional stimuli (Baker et al., 2004; Cappell and Herman, 1972). In line with this model, escalated pain-related alcohol use may be a coping mechanism wherein alcohol is consumed to alleviate pain and pain-related dysphoria. Indeed, individuals experiencing chronic pain often endorse using alcohol to cope with pain symptoms (Alford et al., 2016; Brennan et al., 2011; Riley and

* Corresponding author at: University of California, Psychology Department, 1285 Franz Hall, Box 951563, Los Angeles, CA 90095-1563, United States.
E-mail address: lararay@psych.ucla.edu (L.A. Ray).

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King, 2009). In addition, repeated cycles of alcohol intoxication and withdrawal can dysregulate overlapping pain and addiction brain circuits resulting in both higher pain sensitivity and higher emotional pain/negative affect, which then contribute to further alcohol use (Egli et al., 2012). Pain-related motivation for alcohol use is complex and influenced by interactions between individual differences and affective measures. Problem drinking associates with greater pain and greater use of alcohol to manage pain symptoms (Brennan et al., 2005). Men experiencing chronic pain may be more at risk of AUD and depression, as well as report stronger associations between pain, depression, and alcohol use, compared to women with chronic pain (Barry et al., 2013; Brown, 2015; Manubay et al., 2015). Pain-related anxiety is also positively associated with alcohol-related problems in males, but not females (Zale et al., 2019). Thus, individual differences (e.g., sex) and affective measures are relevant when examining the relationship between pain and alcohol-related outcomes, including motivational factors that contribute to heavy drinking.

Pain catastrophizing is a cognitive-affective construct indexing the tendency to interpret pain as harmful, incessant, and uncontrollable (Sullivan et al., 1995). Pain catastrophizing associates with physical and emotional distress in response to acute pain, among non-chronic pain samples (Sullivan 1995; Kristiansen 2014), although the mechanisms by which pain catastrophizing influences pain processing are not well understood. Individuals with high pain catastrophizing experience greater emotional responses to pain (Martel et al., 2013) and report greater fear and worry about their pain (Edwards et al., 2011). Thus, it is possible that individuals may engage in escape or avoidant behaviors to deal with this heightened emotional response to pain. Specifically, stronger negative emotions are more likely to motivate behavior that can provide rapid relief. Higher pain catastrophizing associates with greater pain-related disability (Severeijns et al., 2001) and escape/avoidance behaviors, such as opioid misuse after controlling for pain severity (Martel et al., 2014, 2013). Pain catastrophizing can also be a target for intervention to help control pain intensity. Reducing pain catastrophizing leads to reductions in the sensory (Taub et al., 2017) and affective experience of pain (Kjøgx et al., 2016; Terry et al., 2015) in healthy individuals and among those with chronic pain. While it is unknown whether heavy drinkers are more likely to report higher levels of pain catastrophizing, smokers are more likely to report more pain catastrophizing compared to nonsmokers (Hooten et al., 2009). Further, whether pain catastrophizing uniquely impacts alcohol use, above and beyond pain intensity, remains to be tested.

To our knowledge, no studies to date have examined the relationship between chronic pain and alcohol craving. A small number of studies utilizing experimental pain paradigms find that acute pain provocation increases urge to drink alcohol (Moskal et al., 2018) or to smoke (Ditre and Brandon, 2008). In a sample of moderate-heavy drinkers, pain induced using a capsaicin-heat model increased alcohol urge and intention to use alcohol (Moskal et al., 2018). This relationship was mediated by pain-induced increases in negative affect. Similarly, laboratory-provoked pain, via cold-pressor task, increased smoking urge and produced shorter latencies to smoke (Ditre and Brandon, 2008). Thus, it may be the case that using alcohol to relieve chronic pain may be explained, at least in part, by greater desire/urge to drink when experiencing pain.

Given the lack of an experimental clinical model of chronic pain, cross-sectional studies including self-report measures of chronic pain are critical for examining the relationship between chronic pain and alcohol-related outcomes, including alcohol craving. To this end, the purpose of this study is to test the independent effects of pain catastrophizing and pain intensity on tonic alcohol craving in a sample of non-treatment seeking heavy drinkers. Using a hierarchical regression approach, we hypothesize that individuals with higher levels of pain intensity and pain catastrophizing will have greater alcohol craving after accounting for demographic, depression, alcohol motives, and alcohol use measures. An exploratory aim is to compare medical pain grades, intended as categorical variables, on demographic and clinical characteristics. Medical pain grades consider both pain intensity and pain-related disability; thus, phenotyping within these classes may further our understanding of the complex relationship between pain and alcohol.

2. Methods and materials

2.1. Participants and procedures

Participants were men and women reporting heavy drinking and completing an in-person assessment battery for a behavioral pharmacology study of ibudilast for AUD (NCT03489850). Although part of the study involved pharmacological manipulations, all data were collected at a baseline screening assessment visit (i.e., prior to medication randomization or any experimental procedures). All study procedures were approved by the University of California, Los Angeles Institutional Review Board, and all participants provided written informed consent after receiving a full explanation of the study procedures.

Interested individuals called the laboratory and completed a phone interview for preliminary eligibility. Likelihood of heavy drinking was initially screened by a score of 2 or higher on the CAGE questionnaire (Ewing, 1984), a mnemonic for questions focused on cutting down, annoyance by criticism, guilty feeling, and eye-openers. In addition, participants also had to report drinking at or above heavy drinking criteria (14+ drinks/week for men and 7+ drinks/week for women) over the last 30 days. Exclusion criteria were: (i) current involvement in treatment programs for alcohol use or have received treatment in the prior 30 days to study participation; (ii) use of non-prescription psychoactive drugs or use of prescription medications for recreational purposes; (iii) self-reported history of major mental illness (i.e., bipolar disorder or psychotic disorders); (iv) current use of antidepressants, mood stabilizers, sedatives, anti-anxiety medications, seizure medications, or prescription painkillers; (v) self-reported history of contraindicated medical conditions (e.g., chronic liver disease, cardiac disease); (vi) if female, pregnant (as verified by a urine sample), nursing, or planning to get pregnant in the next 6 months or refusal to use a reliable method of birth control; (vii) breath alcohol concentration greater than 0.000 g/dl as measured by the Dräger Inc. Alcotest® 6510; and (viii) positive urine toxicology screen for any drug (other than cannabis), as measured by Medimpex United Inc. 10 panel drug test. Eligible participants were invited to the laboratory to complete an in-person testing battery that included sociodemographic variables, self-report questionnaires, and interview-based assessments (described below). Smoking status (categorical; Smoker vs Non-Smoker) was determined using the first question on The Fagerström Test for Nicotine Dependence (Heatherton et al., 1991), which asks participants if they currently smoke cigarettes.

2.2. Measures

2.2.1. Alcohol use and alcohol problems

were assessed using (a) the Timeline Followback (Sobell and Sobell, 1992) to determine alcohol use quantity and frequency over the past 30 days; (b) Clinical Institute Withdrawal Assessment for Alcohol-Revised (CIWA-Ar) (Sullivan et al., 1989), to evaluate alcohol withdrawal symptoms; (c) the Alcohol Dependence Scale (ADS) (Skinner et al., 1984) to assess for problems related to excessive drinking; (d) the Obsessive-Compulsive Dependence Scale (OCDS) (Anton et al., 1995) to measure tonic alcohol craving (Hartwell and Ray, 2018); and (e) the Reasons for Heavy Drinking Questionnaire (RHDQ), which is comprised of heavy drinking for normalizing and heavy drinking for reinforcement subscales (Adams et al., 2016). The Structured Clinical Interview for DSM-5 (First et al., 2016) was administered by a master's level clinician to assess for current (i.e., past 12-months) AUD symptoms. The OCDS, developed from a framework that incorporates similarities between

obsessive-compulsive phenomenology and alcohol craving, provides a multi-dimensional assessment of general, unprovoked craving that includes obsessive thoughts about drinking, compulsive drinking behavior, and subsequent interference with functioning (Anton, 2000; Moak et al., 1998; Ray et al., 2013). The OCDS has also been shown to have high reliability (Anton, 2000; Moak et al., 1998) and correlate with the Alcohol Urge Questionnaire (AUQ; (Bohn et al., 1995), Alcohol Craving Questionnaire (ACQ; (Raabe et al., 2005), and ADS (Anton et al., 1996).

2.2.2. Mood and pain were self-reported using

(a) the Beck Depression Inventory II (BDI-II) to capture depressive symptoms over the past two weeks (Beck et al., 1996), (b) the Graded Chronic Pain Scale (GCPS) (Von Korff et al., 1992), which captures pain severity and is widely used in medical pain research, and (c) the Pain Catastrophizing Scale (PCS) (Sullivan et al., 1995) which focuses on the emotional experience of physical pain. The first three items of the GCPS yielded mean pain intensity calculated as the average of current pain intensity, worst pain intensity, and average pain intensity over the last six months, each rated on a 0–10 scale with the endpoints of "no pain" (0) and "a lot of pain" (10). Exploratory analyses were conducted examining clinical and demographic differences between pain grades, which are categorical. As recommended, pain grades were categorized using mean pain intensity scores (ranging from 0−100) according to the original GCPS, resulting in no pain (Grade 0), low pain intensity (Grade I; mean pain intensity less than 50), and high pain intensity (Grade II; mean pain intensity 50 or greater) groups (Von Korff et al., 1992). All individuals in the current study had low pain disability (< 3 disability points). Grades III and IV, which are characterized by moderate and high levels of pain-related disability, respectively, could not be classified because no participants in this sample reported such levels of pain-related disability. Thus, only pain grades 0-II were classified.

2.3. Data analytic plan

Pearson correlation analyses were used to examine the relationship among measures of pain, affect, and alcohol use/problems. A hierarchical regression framework was used to test the independent effects of pain intensity and pain catastrophizing on alcohol craving. Demographic characteristics (sex, age, race, smoking status) and pain intensity were included in the lowest block. Pain intensity was included in this lowest block in order to assess change in the parameter estimate as sequential blocks were added into the model. Affective measures (BDI-II and pain catastrophizing) were included in the second block. The third block included alcohol use (ADS and drinks per drinking day) and alcohol motives (RHDQ) indices. Parameter estimates are adjusted for all variables in the model as each block is added. The final model (Model 3) reports the adjusted parameter estimates for all measures included in the hierarchical regression. For exploratory analyses, a series of one-way ANOVAs or chi-square tests were used to assess for demographic and clinical differences between no pain, low pain intensity, and high pain intensity grades. In order to control Type I error, Tukey *post hoc* tests were used to conduct pairwise comparisons among pain grades following significant omnibus ANOVA tests. Effect sizes for exploratory analyses are reported as partial eta-squared (η_p^2) for ANOVAs and phi (Φ) for chi-square tests. All analyses were conducted using SAS 9.4. Statistical significance was set at $p < 0.05$.

3. Results

3.1. Sample characteristics and correlation analyses

Participants were, on average, 31.53 (SD = 8.55) years old and were 70 % male. Participants reported an average of 5.33 (SD = 2.92) drinks per drinking day and an average pain intensity of 25.57 (SD = 19.97) representing low intensity pain. Average ADS scores were 12.72 (SD = 7.19). Additional clinical variables are presented in Table 1. Pearson's correlations among study variables included in the regression analyses are provided in Table 2. Pain intensity significantly correlated with pain catastrophizing ($p < 0.001$), BDI-II ($p = 0.002$), ADS ($p < 0.001$), drinks per drinking day ($p = 0.042$), OCDS ($p < 0.001$), and heavy drinking to feel normal ($p < 0.001$). Pain catastrophizing significantly correlated with BDI-II ($p < 0.001$), ADS ($p < 0.001$), OCDS ($p < 0.001$), and heavy drinking to feel normal ($p < 0.001$). Pain catastrophizing did not associate with drinks per drinking day ($p > 0.05$) and neither pain measure associated with heavy drinking for the reinforcing effects of alcohol (p's > 0.05).

3.2. Hierarchical regression

Results from the hierarchical regression for OCDS scores are presented in Table 3. Pain intensity and demographic variables accounted for 25.6 % of the variance ($p < .001$) such that smoking status ($p = .002$) and pain intensity ($p < .0001$) predicted greater OCDS total scores. Sex, age, and race did not significantly predict OCDS total scores in the first block (p's > 0.05). The addition of affective measures significantly predicted an additional 22.9 % (R^2 of the model = .485; $p < .0001$). When the second block was added, age ($p = .003$) from the first block then became a significant predictor of greater OCDS total scores. Smoking status ($p = .002$) and pain intensity ($p = 0.037$) remained significant. Of the affective measures in the second block, BDI-II ($p < .0001$) and pain catastrophizing ($p = .005$) significantly predicted greater OCDS total scores. The addition of alcohol measures significantly predicted an additional 17.0 % (R^2 of the model = .655; $p < .0001$). When this third block was added, pain intensity, which was significant in the second block, did not significantly predict OCDS total scores ($p > 0.05$). Age ($p = .012$), smoking status ($p = .039$), BDI-II ($p = .003$), and pain catastrophizing ($p = .018$) remained significant. Of the alcohol measures in the third block, ADS scores ($p < .0001$) and self-reported heavy drinking to feel normal ($p = .043$) predicted greater OCDS total scores. Drinks per drinking day and self-reported heavy drinking for the reinforcing effects of alcohol did not reach statistical significance (p's > 0.05) in the third block.

Table 1
Sample (n = 128) demographic and clinical characteristics.

Variables (scale ranges)	Means (SD) or N (%)
Age	31.53 (8.55)
Sex - Male	89 (69.53 %)
Race	
White	55 (42.97 %)
Black	24 (18.75 %)
Asian	10 (7.81 %)
Mixed	17 (13.28 %)
Another	22 (17.19 %)
Cigarette smoker	64 (50.00 %)
BDI-II (0−63)	11.35 (9.48)
AUD	20/25/40/43
None/Mild/Moderate/Severe	
Drinking days	18.49 (7.36)
Drinks per week	23.13 (17.62)
Drinks per drinking day	5.33 (2.92)
CIWA-Ar (0−67)	1.05 (2.23)
ADS (0−40)	12.72 (7.19)
AUDIT (0−40)	15.78 (7.25)
OCDS (0−40)	13.69 (7.66)
RHDQ-Reinforcement (0−30)	22.33 (5.09)
RHDQ-Normalizing (0−30)	8.31 (7.32)
Pain intensity (0−100)	25.57 (19.97)
Pain catastrophizing (0−50)	10.12 (10.64)

BDI-II, Beck Depression Inventory; AUD; Alcohol Use Disorder; Clinical Institute Withdrawal Assessment for Alcohol-Revised; ADS, Alcohol Dependence Scale; AUDIT, Alcohol Use Disorder Identification Test; OCDS, Obsessive Compulsive Drinking Scale; RHDQ, Reasons for Heavy Drinking Scale.

Table 2
Zero-order correlations between pain, affect, and alcohol measures.

	1	2	3	4	5	6	7	8
1. Pain intensity	1.000							
2. Pain catastrophizing	.435***	1.000						
3. BDI-II	.269**	.443***	1.000					
4. ADS	.300***	.351***	.427***	1.000				
5. Drinks per drinking day	.177*	.114	.323***	.477***	1.000			
6. OCDS	.359***	.453***	.542***	.719***	.397***	1.000		
7. RHDQ-Reinforcement	.132	.137	.030	.215*	.208*	.191*	1.000	
8. RHDQ-Normalizing	.301***	.299***	.424***	.595***	.478***	.611***	.226*	1.000

BDI-II, Beck Depression Inventory; ADS, Alcohol Dependence Scale; OCDS, Obsessive Compulsive Drinking Scale; RHDQ, Reasons for Heavy Drinking Scale. * indicates a significant association at $p < 0.05$. ** indicates a significant association at $p < 0.01$. *** indicates a significant association at $p < 0.001$.

Table 3
Hierarchical regression of predictors of alcohol craving.

	OCDS-Total Scores								
	Model 1			Model 2			Model 3		
	$R^2/\Delta R^2$	*b*	Std. Err.	$R^2/\Delta R^2$	*b*	Std. Err.	$R^2/\Delta R^2$	*b*	Std. Err.
	.256			.485 / .229***			.655 / .170***		
Block 1 Demographic characteristics									
Sex[a]		.474	1.335		1.502	1.167		.082	.955
Age		.134	.075		.198**	.064		.137*	.054
Race = Black[b]		−2.682	1.746		−2.352	1.496		−1.711	1.237
Race = Asian		−3.064	2.409		−1.683	2.045		−.417	1.641
Race = Mixed		0.744	1.941		.469	1.677		.513	1.372
Race = Another		2.717	1.729		1.747	1.466		−.182	1.211
Cigarette Smoker[c]		3.949**	1.251		3.286**	1.060		1.85*	0.885
Pain intensity									
Pain Intensity		.135***	.031		.062*	.029		.013	.024
Block 2									
Affective measures									
BDI-II					.309***	.062		.172**	.055
Pain catastrophizing					.174**	.061		.122*	.051
Block 3									
Alcohol measures									
ADS								.454***	.082
Drinks per drinking day								−.049	.176
RHDQ-Reinforcement								.046	.089
RHDQ-Normalizing								.153*	.077

BDI-II, Beck Depression Inventory; PCS, Pain Catastrophizing Scale; ADS, Alcohol Dependence Scale; DPDD, Drinks per Drinking Day; OCDS, Obsessive Compulsive Drinking Scale; RHDQ, Reasons for Heavy Drinking Scale. [a]Female is reference group for Sex. [b]White is reference group for Race variable. [c]Non-Smoker is the reference group for Cigarette Smoker. * indicates a significant estimate at $p < 0.05$. ** indicates a significant estimate at $p < 0.01$. *** indicates a significant estimate at $p < 0.001$.

3.3. Exploratory analyses

Exploratory analyses among medical pain intensity grades on demographic and clinical variables are presented in Table 4. In terms of pain severity, participants were classified into one of three GCPS pain grades (Von Korff et al., 1992): Grade 0: No pain ($n = 24$, 18.75 %); Grade I: low pain intensity-low pain disability ($n = 85$; 66.40 %), Grade II: high pain intensity-low pain disability ($n = 19$; 14.84 %). Tukey *post hoc* tests showed that among the pain intensity grades, individuals who self-reported no pain were significantly older than individuals in the low intensity pain group. The high intensity pain group had greater depressive symptomology and more drinks per week compared to the low intensity pain group. Relative to both no and low intensity pain groups, the high intensity pain group had greater drinks per drinking day and alcohol dependence severity, more alcohol craving, and were more likely to self-report engaging in heavy drinking to "feel normal" (i.e., significantly higher scores on the normalization subscale of the RHDQ). The high intensity pain group also reported more pain catastrophizing relative to both no and low pain groups.

4. Discussion

The purpose of the current study was to determine the independent contribution of pain intensity and pain catastrophizing to alcohol craving, measured using OCDS scores, in a sample of heavy drinkers. Using a hierarchical regression framework, we found that pain catastrophizing predicted alcohol craving over and above demographics, pain intensity, depression, alcohol motives, and alcohol use measures in the final model. Exploratory analyses between GCPS categorical pain grades showed that individuals in the high intensity pain group self-reported higher levels of pain catastrophizing, higher depression symptomology, higher alcohol use and tonic alcohol craving, and higher levels of drinking to feel "normal" compared to the lower pain grades. These analyses largely confirm the strong affective component of chronic pain and extend this literature to heavy drinkers. The present study isolates pain catastrophizing as a predictor of alcohol craving, which may serve as a potential intervention target for heavy drinkers who experience chronic pain.

A moderate positive correlation was observed between pain intensity and pain catastrophizing. Both pain catastrophizing and pain intensity were associated with depressive symptomology, alcohol dependence, alcohol craving, and drinking for the normalizing effects of alcohol. However, pain intensity and not pain catastrophizing, was associated with self-reported drinks per drinking day. This suggests that pain intensity has a unique relationship with self-reported alcohol consumption. Neither pain measure related to self-reported heavy drinking for

Table 4
Comparisons among pain intensity grades on demographic and clinical characteristics.

	Means (SD) or N (%)					
	No Pain (n = 24; 18.75%)	Low Intensity Pain (n = 85; 66.40%)	High Intensity Pain (n = 19; 14.84%)	Statistic	P	Effect Size
Age[a]	36.04 (9.17)	30.59 (8.27)	30.05 (7.46)	$F = 4.36$	0.01	$\eta_p^2 = 0.07$
Sex (Male)	18 (75.00 %)	56 (65.88 %)	15 (78.95 %)	$\chi^2 = 1.67$	0.43	$\phi = .114$
Cigarette smoker	13 (54.17 %)	40 (47.06 %)	11 (61.11 %)	$\chi^2 = 0.93$	0.63	$\phi = .127$
BDI-II[c]	11.04 (13.46)	10.11 (7.56)	17.32 (9.42)	$F = 4.79$	0.01	$\eta_p^2 = 0.07$
AUD[b,c] None/Mild/Moderate/ Severe	4/5/5/10	15/21/27/22	0/0/8/11	Fisher's Exact Test	0.01	–
Drinking days	19.91 (7.34)	17.85 (7.57)	19.53 (6.43)	$F = 0.49$	0.61	$\eta_p^2 = 0.01$
Drinks per eek[c]	26.34 (17.78)	19.59 (13.61)	34.54 (26.36)	$F = 6.37$	<0.01	$\eta_p^2 = 0.09$
Drinks per drinking day[b,c]	5.67 (3.17)	4.72 (2.27)	7.54 (3.95)	$F = 8.31$	<0.001	$\eta_p^2 = 0.12$
CIWA-Ar	0.96 (1.85)	0.92 (2.01)	1.74 (3.31)	$F = 1.08$	0.34	$\eta_p^2 = 0.02$
ADS[b,c]	11.96 (7.04)	11.86 (6.56)	17.53 (8.51)	$F = 5.32$	<0.01	$\eta_p^2 = 0.08$
AUDIT[b,c]	14.67 (6.98)	14.95 (6.94)	20.89 (7.17)	$F = 6.01$	<0.01	$\eta_p^2 = 0.09$
OCDS[b,c]	13.45 (7.50)	12.47 (7.07)	19.42 (8.19)	$F = 7.01$	0.01	$\eta_p^2 = 0.10$
RHDQ-Reinforcement	23.21 (5.78)	21.78 (4.88)	23.68 (4.92)	$F = 1.55$	0.22	$\eta_p^2 = 0.02$
RHDQ-Normalizing[b,c]	7.29 (6.73)	7.25 (6.50)	14.37 (8.78)	$F = 8.55$	<0.001	$\eta_p^2 = 0.12$
Pain catastrophizing[b,c]	5.38 (8.93)	9.69 (9.13)	18.00 (14.55)	$F = 8.58$	<0.001	$\eta_p^2 = 0.12$

BDI-II, Beck Depression Inventory; AUD; Alcohol Use Disorder; Clinical Institute Withdrawal Assessment for Alcohol-Revised; ADS, Alcohol Dependence Scale; AUDIT, Alcohol Use Disorder Identification Test; OCDS, Obsessive Compulsive Drinking Scale; RHDQ, Reasons for Heavy Drinking Scale; Bold type indicates significant differences between groups.

[a] No Pain and Low Intensity Pain groups differ, $P < 0.05$.

[b] No Pain and High Intensity Pain groups differ, $P < 0.05$.

[c] Low Intensity Pain and High Intensity Pain groups differ, $P < 0.05$.

the reinforcing effects of alcohol. Few studies include measures of emotional components of pain, such as pain catastrophizing. Previous reports indicate that pain catastrophizing positively associates with pain intensity in individuals with rheumatoid arthritis (Keefe et al., 1989), and we extend this finding to a sample of heavy drinkers, most of whom (84 %) meet DSM-5 AUD criteria

In the final model of the hierarchical regression, pain catastrophizing, but not pain intensity, predicted tonic alcohol craving scores after controlling for alcohol measures. The lack of an independent effect of pain intensity in the final model was surprising given evidence that individuals who experience chronic pain report more alcohol use (Hoffmann et al., 1995) and incidences of AUD (Vowles et al., 2018) relative to the general population. Our findings suggest that both pain measures independently predict tonic alcohol craving until alcohol use measures are considered. Compared to the work on pain intensity and alcohol use, the role of pain catastrophizing in alcohol use and AUD is relatively unknown. However, pain catastrophizing contributes to greater use of prescription opioids (Sharifzadeh et al., 2017), moderates the relationship between pain intensity and urge to smoke (Kosiba et al., 2018), and associates with greater drug craving in an inpatient sample of individuals with substance use disorder who report chronic pain (Kneeland et al., 2019). The current study demonstrates an independent effect of pain catastrophizing on tonic alcohol craving, but future work is needed to examine the relationship between pain measures on both tonic and phasic measures of alcohol craving. The lack of sex-specific effects on alcohol craving is also notable, considering previous findings implicating sex differences in motivational factors that contribute to pain-related alcohol use (Barry et al., 2013; Brown, 2015; Manubay et al., 2015; Zale et al., 2019).

Exploratory analyses revealed differences between pain grades on demographic and clinical measures. Individuals who experienced high intensity pain had more severe alcohol use, alcohol dependence severity, and pain catastrophizing compared to the no pain and low pain intensity groups. These initial findings complement previous work showing a bidirectional relationship between chronic pain and alcohol use (Edwards et al., 2020). Of note, individuals in the high intensity group had greater pain catastrophizing and reported more heavy drinking to feel "normal" (i.e., scored higher on the normalization subscale of the RHDQ), consistent with studies demonstrating that alcohol use among individuals experiencing pain is motivated by pain relief (Riley and King, 2009) or to avoid negative physiological consequences associated with alcohol dependence and withdrawal (Adams et al., 2016). It may be the case that emotional responses to pain exacerbate painful experiences, contributing to a tendency to engage in heavy drinking to feel normal and /or reinstate a homeostatic balance. These findings, while exploratory, deserve further attention in clinical samples and the RHDQ measure may be sensitive to differences between heavy drinking for negative reinforcement and heavy drinking to normalize certain phenomena among individuals experiencing pain. The RHDQ scales may also be useful in tailoring treatments depending on an individual's reason for engaging in heavy drinking (Adams et al., 2016). For instance, individuals with higher scores on the RHDQ-Normalizing factor might benefit from pharmacotherapies that target physiological imbalances due to repeated cycles of heavy drinking and alcohol withdrawal (Johnson, 2004; Roberto et al., 2008) compared to interventions that target alcohol's reinforcing effects.

Negative reinforcement theories of addiction posit that relief or elimination of aversive stimuli motivates continued pathological alcohol consumption (Egli et al., 2012). In the case of pain, the experience of painful stimuli can drive heavy alcohol drinking, as found in experimental studies (Hoffmann et al., 1995; Vowles et al., 2018). However, the relationship is bidirectional, such that heavy alcohol drinking can also exacerbate the experience of pain. In addition, alcohol withdrawal induces hyperalgesia in humans (Jochum et al., 2010) and laboratory animals (Edwards et al., 2020; Robins et al., 2019). In the wake of the opioid crisis, the alcohol field has given particular attention to the relationship between alcohol and pain (Edwards et al., 2020). The current study adds to this line of inquiry by highlighting a negative emotional component of pain that also relates to alcohol use severity and motivation to drink, in this case predicting alcohol craving independent

of actual pain intensity. Pain catastrophizing may also serve as an intervention target in chronic pain patients as therapeutic techniques, such as pain education coupled with imagining/mindfulness education, can decrease pain catastrophizing and, thereby, reduce pain perception (Kjøgx et al., 2016; Terry et al., 2015). Cognitive-behavioral therapy has demonstrated small to moderate effect sizes in treating chronic pain (Richmond et al., 2015; Williams et al., 2012). Changes in maladaptive cognitions have been proposed as one mechanism to promote reductions in pain catastrophizing that may not only be specific to cognitive-behavioral therapy, but also extend to physical exercise and pain education (Smeets et al., 2006; Terry et al., 2015). Lastly, self-efficacy has been shown to be a robust correlate of pain-related outcomes (e.g. pain intensity and affective distress) (Jackson et al., 2014) and can be a target for psychosocial interventions for pain and depressive symptomology (Damush et al., 2016; Turner et al., 2005). Thus, in order to appropriately disentangle associations between pain and alcohol use, inclusion of negative emotional aspects of pain and related cognitions in alcohol studies are warranted.

Although this study is novel in that it assesses both pain intensity and pain catastrophizing, there are limitations. The study sample was comprised of heavy drinkers; thus, we were not able to assess self-reported pain intensity across levels of alcohol consumption (i.e., light and moderate drinkers) and/or among treatment-seekers. In addition, the sample size of the high intensity pain group was relatively small, and the study sample did not meet criteria for medical pain grades characterized by moderate and severe pain-related disability. It may be the case that the relationship between pain intensity and pain catastrophizing may differ among these individuals. Lastly, our sample excluded individuals taking prescription opioids, which limits the generalizability of the findings to heavy drinkers who are not receiving pharmacological pain management through opioids.

On balance, the current study highlights the contribution of pain to alcohol craving, especially the affective component of pain, in a sample of non-treatment seeking heavy drinkers. That is, pain catastrophizing predicted higher alcohol craving scores independent of actual pain intensity after controlling for alcohol measures in the final model of the hierarchical regression. Individuals categorized in the high intensity pain group consumed more alcohol and had greater dependence severity, as well as reported heavy drinking to feel normal. The high intensity pain group also had higher levels of pain catastrophizing. Given these findings, future work is needed examining alcohol use in clinical samples that include a wider range of medical pain grades, and consider emotional aspects of pain, pain intensity, and pain management with opioid analgesics.

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Contributors

SJN conducted statistical analyses, interpreted the data, and drafted the manuscript. RG, ENG, and CMC interpreted the data and provided edits on the manuscript. LAR designed the main study, supervised data collection, interpreted the data, and provided edits on the manuscript. All authors have approved the final article.

Declaration of Competing Interest

The authors report no declarations of interest.

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