

# Pain Intensity, Emotion Dysregulation, and Hazardous Drinking Among Adults With Chronic Pain

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**ABSTRACT. Objective:** Chronic pain and hazardous alcohol use (i.e., a pattern of alcohol consumption that increases risk for harmful consequences) are prevalent and frequently comorbid conditions that have been posited to interact in a bidirectional manner, leading to greater pain and heavier drinking. Despite evidence that emotion dysregulation (i.e., difficulty modulating emotional responses when experiencing negative emotions) is independently associated with both greater pain and greater alcohol consumption, we are not aware of any previous research examining relations between emotion dysregulation, pain intensity, and hazardous alcohol use among individuals with chronic pain. **Method:** Participants included 125 past-month alcohol users with chronic musculoskeletal pain (38.4% female; mean age = 32.97 years; mean drinks/day = 1.62) who were recruited for an online survey study of pain and

substance use. **Results:** As expected, emotion dysregulation was positively associated with increased odds of hazardous alcohol use. We also observed a significant indirect association, such that higher levels of emotion dysregulation were associated with greater pain intensity, which in turn was associated with a greater likelihood of scoring above the Alcohol Use Disorders Identification Test cutoff for hazardous alcohol use. **Conclusions:** These findings suggest that emotion dysregulation may contribute to hazardous drinking among individuals with chronic pain, perhaps indirectly via pain amplification. Emotion dysregulation warrants consideration as a potential transdiagnostic vulnerability factor in comorbid chronic pain and hazardous drinking. Future prospective research is needed to examine causal pathways and establish temporal precedence. (*J. Stud. Alcohol Drugs*, 83, 223–230, 2022)

ALCOHOL USE AND CHRONIC PAIN are frequently

aco-occurring public health issues that engender significant economic burden in terms of healthcare costs and lost productivity (Sacks et al., 2015). The prevalence of hazardous alcohol use (i.e., a pattern of alcohol consumption that increases risk for harmful consequences) is substantially higher among individuals with chronic pain, relative to the general population (Saunders et al., 1993a), and nationally representative data further indicate that individuals who endorse pain are twice as likely to meet criteria for alcohol use disorder (AUD; i.e., a problematic pattern of alcohol use that leads to clinically significant impairment and/or distress; American Psychiatric Association, 2013; Subramaniam et al., 2013; Von Korff et al., 2005). There is also evidence that hazardous alcohol consumption increases the likelihood of reporting moderate-to-severe past-month pain (Brennan et al., 2005; Kim et al., 2013; Lawton & Simpson, 2009), and hazardous alcohol use has been associated with adverse pain-related outcomes, including greater pain severity and increased risk of developing pain following acute injury (Castillo et al., 2006; Holmes et al., 2010; Zale et al., 2015).

Pain and alcohol use are posited to interrelate in the manner of a positive feedback loop, leading to the progression and maintenance of both pain and drinking over time (Ditre et al., 2019; Zale et al., 2015). Indeed,

chronic pain has been positively associated with greater alcohol consumption, rates of hazardous drinking (Lawton & Simpson, 2009), and current drinking problems, with up to 25% of treatment-seeking pain patients endorsing heavy drinking (Brennan & Soohoo, 2013). Additionally, pain can be a potent motivator of drinking, and alcohol has been shown to reduce pain in the short term (Lawton & Simpson, 2009; Moskal et al., 2018; Thompson, 2017). These analgesic effects are theorized to drive the use of alcohol to cope with pain, and there is evidence that drinkers report using alcohol for this purpose (Ditre et al., 2019; Goebel et al., 2011; Sheu et al., 2008). Conversely, heavy alcohol consumption has been associated with the onset and severity of painful conditions (Banks et al., 2010; Cheng et al., 2000; Chopra & Tiwari, 2012; Sá et al., 2008). Finally, there has been increased empirical interest in factors that may influence interrelations between pain and alcohol use and serve as potential treatment targets among individuals with comorbid pain and AUD (Zale et al., 2015).

Emotion dysregulation, or difficulty modulating emotional responses when experiencing negative affect (Gratz & Roemer, 2004), has been associated with both chronic pain (Koechlin et al., 2018; Overstreet & Goodin, 2018; Paulus et al., 2016a) and greater alcohol consumption separately (Bradizza et al., 2018; Paulus et al., 2016b, 2017). There is converging evidence that emotion dysregulation may amplify the experience of pain (Kököneyi et al., 2014; Overstreet & Goodin, 2018; Paulus et al., 2017; Rogers et al., 2020), and difficulties with emotion regulation have been associated with both pain intensity and pain-related

Received: February 23, 2021. Revision: September 7, 2021.

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disability (Koechlin et al., 2018; Rogers et al., 2020). Emotion dysregulation has also been linked to heavier/more frequent drinking among individuals with AUD (Bradizza et al., 2018) and more hazardous drinking among primary care-based populations (Paulus et al., 2016b, 2017), and has been suggested as a mechanism underlying problematic alcohol use (Bandura et al., 2003; Barlow et al., 2004; Bradizza et al., 2018). Previous investigations of the relationship between pain intensity and alcohol use have suggested emotion dysregulation as a potential explanatory factor underlying these associations (Paulus et al., 2016b, 2017), but these relationships have not been explored among individuals with chronic pain.

Given the paucity of research examining emotion dysregulation and hazardous drinking among individuals with pain, one goal of the present study was to examine cross-sectional relations between emotion dysregulation and hazardous alcohol use in a chronic pain population. Specifically, we hypothesized that higher levels of emotion dysregulation would be associated with increased odds of hazardous alcohol use among individuals with chronic pain. We also hypothesized that higher levels of emotion dysregulation would be associated with more granular indices of alcohol consumption (e.g., frequency/quantity of drinking), dependence (e.g., impaired control over drinking), and harmful use (e.g., consequences and alcohol-related injuries) among individuals with pain. In addition, although emotion dysregulation has been independently linked with more severe pain and hazardous drinking, we are not aware of any previous research examining the role of pain intensity in relations between emotion dysregulation and hazardous alcohol use among individuals with chronic pain. Thus, a second goal of this study was to test the hypothesis that emotion dysregulation would be indirectly associated with the likelihood of engaging in hazardous alcohol use via pain intensity. Finally, we conducted a series of exploratory analyses examining associations between emotion dysregulation, pain intensity, and indices of alcohol consumption, dependence, and harmful use.

## Method

### *Participants and procedures*

Participants were 125 current alcohol users (38.4% female;  $M_{age} = 32.97$ ;  $M_{drinks/day} = 1.62$ ) recruited for an online survey study of pain and substance use that was approved by an institutional review board (LaRowe et al., 2021). Participants were required to be at least 21 years of age, currently reside in the United States, endorse past-month alcohol use and current chronic musculoskeletal pain, and be able to read English. As a screening question for chronic pain, participants were asked, "Do you currently suffer from any type of chronic pain, that is, pain that occurs

constantly or flares up frequently? Do not report aches and pain that are fleeting or minor." They were further queried about the location of their pain to ascertain whether the pain was musculoskeletal in nature (i.e., pain of the muscles, ligaments/tendons, bones, or joints). Participants provided informed consent before completing the approximately 40-minute survey. The current analyses were limited to those who completed the Difficulties in Emotion Regulation Scale (DERS; completed by  $n = 139$  participants). We also included a response accuracy check ("To monitor quality, please respond with a two for this item"), and participants who responded incorrectly to this item were excluded from analyses ( $n = 14$ ). Thus, the final sample consisted of 125 participants.

### *Measures*

**Emotion dysregulation.** Emotion dysregulation was assessed using the DERS (Gratz & Roemer, 2004). The DERS consists of 36 items that assess typical levels of difficulties in emotion regulation (e.g., "I experience my emotions as overwhelming and out of control") and are rated on a 5-point Likert scale ranging from 1 = *almost never* to 5 = *almost always*. Higher total scores (possible range: 36–180) indicate greater emotion dysregulation. The DERS has previously been validated for use in chronic pain populations (Kököneyi et al., 2014) and demonstrated excellent internal consistency in the current sample ( $\alpha = .94$ ).

**Clinical pain variables.** The Graded Chronic Pain Scale (GCPS; Von Korff et al., 1992) is a reliable and valid method of assessing chronic pain and global pain intensity across a range of chronically painful conditions (Von Korff, 2011). The GCPS provides a categorical classification of chronic pain by grade (severity) that ranges from Grade 1 (low intensity, low interference) to Grade 4 (severe interference). Consistent with scoring instructions (Von Korff et al., 1992), the characteristic pain intensity score was computed by summing ratings (0 = *no pain* to 10 = *pain as bad as it could be*) of pain "right now," "on average," and at its "worst" in the past 3 months, with higher total scores (range: 0–30) corresponding with more intense pain. The GCPS-Characteristic Pain Intensity (CPI) subscale demonstrated acceptable internal consistency in the current sample ( $\alpha = .70$ ).

**Hazardous alcohol use.** The Alcohol Use Disorders Identification Test (AUDIT) includes 10 items that are rated on a 5-point scale ranging from 0 (*never*) to 4 (*4 or more times a week*) and summed to generate a total score (Babor et al., 1992). A total score cutoff of 7 or more for women and 8 or more for men is considered indicative of hazardous alcohol use (e.g., Babor et al., 1992; Saunders et al., 1993b). The AUDIT also includes three subscales that assess patterns of alcohol use. The AUDIT-Consumption subscale assesses quantity/frequency of alcohol use, the

AUDIT–Harmful Use subscale assesses drinking that results in consequences to physical and mental health, and the AUDIT–Dependence subscale assesses drinking that has resulted in dependence/addiction. Previous research has consistently demonstrated the reliability and validity of the AUDIT (e.g., Reinert & Allen, 2002). In the current sample, the AUDIT demonstrated excellent internal consistency ( $\alpha = .90$ ).

*Quantity/frequency of alcohol consumption.* The average number of drinks consumed per day was assessed using the Modified Daily Drinking Questionnaire (DDQ-M; Dimeff, 1999). The DDQ-M allows for the calculation of the average number of drinks consumed each day and asks participants to indicate the typical number of drinks they have consumed each day of the week on average over the past 90 days. The DDQ-M is a valid and reliable instrument that is commonly used to examine patterns of drinking behavior and was used to calculate the average daily drinks score in the present study.

*Sociodemographic and pain characteristics.* Participants were asked to report sociodemographic information (e.g., age, gender, race, ethnicity, education, employment status, income) and information on their pain (location and duration).

#### *Data analytic plan*

All analyses were conducted using IBM SPSS Statistics for Windows Version 25 and PROCESS for SPSS (IBM Corp, Armonk, NY; Hayes, 2013). First, a logistic regression was run to ascertain the effect of emotion dysregulation on the likelihood that participants meet criteria for hazardous alcohol use as measured by the AUDIT. Second, a series of hierarchical linear regressions were conducted to test associations between emotion dysregulation and (a) AUDIT–Consumption scores, (b) AUDIT–Harmful Use scores, and (c) AUDIT–Dependence scores. Given previously observed associations with alcohol use severity (Brennan & Soohoo, 2013; Brennan et al., 2005, 2011; Chartier & Caetano, 2010; Nolen-Hoeksema, 2004), gender, race, ethnicity, and age were included as covariates in both the logistic regression and hierarchical linear regression models. For each model, predictors were entered in the following order: Step 1 (covariates); Step 2 (DERS). The relative contribution of emotion dysregulation in explaining observed variance in each criterion variable was assessed by examining change in the  $R$ -squared statistic ( $\Delta R^2$ ) at the second step of each model. The  $\Delta R^2$  represents the incremental increase in the predictive value of the model resulting from the addition of a predictor (i.e., emotion dysregulation) at Step 2.

Indirect associations were tested using a series of conditional process models using the PROCESS macro for SPSS (Preacher & Hayes, 2008), which employs an ordinary least-squares–based approach. Individual paths (for indirect associations) are considered statistically significant if the

upper and lower limits of the confidence intervals (CIs) do not cross zero (Hayes, 2013). Emotion dysregulation was examined as a predictor, with pain intensity as the explanatory variable, and AUDIT scores (using the AUDIT Hazardous Drinking cutoff and the AUDIT subscales) as separate outcomes. Recommended 95% CIs and path coefficients were generated via bootstrapping set at 10,000 resamples with replacement (Hayes, 2013; Preacher & Hayes, 2008). Bootstrapping is a nonparametric approach to path analysis, which reduces limitations associated with statistical power and Type 1 error inherent to stepwise regression (Preacher & Hayes, 2004). Direct, indirect, and total effects were examined.

## **Results**

### *Participant characteristics*

Participant characteristics are presented in Table 1. The sample was mostly male (61.6%), White (57.6%), and well educated (72.8% completed at least a 4-year college degree). A total of 24% of the sample identified as Hispanic or Latino. The mean age was 32.97 years ( $SD = 9.7$ , range: 22–66). Participants reported drinking approximately 1.62 alcoholic beverages each day on average ( $SD = 1.27$ ), and the mean AUDIT total score was 16.70 ( $SD = 11.41$ , range: 1–36), indicating a relatively high level of drinking problems. About two thirds of participants (67.2%) scored above the AUDIT cutoff for hazardous drinking. DERS scores in this sample ( $M = 98.49$ ,  $SD = 24.85$ , range: 41–163) were also relatively high; this is expected, as clinical samples tend to evince higher scores on the DERS than healthy controls (Giromini et al., 2017). The mean GCPS-CPI rating among this sample was 18.71 ( $SD = 4.71$ , range: 8–29), indicating moderately high characteristic pain intensity. Participants reported 3.12 pain locations on average ( $SD = 2.38$ , range: 1–15). The most commonly endorsed primary pain locations were back/neck (40.8%), head/face (23.2%), and lower extremities (14.4%), and nearly half of the sample (46.4%) reported that their current pain problem lasted more than 1 year. The majority of participants (66.4%) endorsed either Grade 3 or Grade 4 chronic pain, indicating high levels of pain-related disability.

### *Emotion dysregulation, hazardous alcohol use, and AUDIT subscale scores*

Greater emotion dysregulation was associated with an increased likelihood of hazardous alcohol use (odds ratio [OR] = 1.05, 95% CI [1.03, 1.08],  $p < .001$ ). Every 1-point increase in DERS score was associated with a 1.05 times greater likelihood (i.e., 5% increase in the likelihood) of meeting AUDIT criteria for hazardous alcohol use. The model explained 47.8% (Nagelkerke  $R^2$ ) of the variance

TABLE 1. Sociodemographic, emotion dysregulation, alcohol, and pain characteristics ( $n = 125$ )

Variable	$n$ (%)
Gender	
Male	77 (61.6%)
Race	
White	72 (57.6%)
Black or African American	15 (12%)
Asian	29 (23.2%)
American Indian/Alaska Native	5 (4%)
Other	4 (3.2%)
Ethnicity	
Hispanic	38 (24%)
Marital status	
Single	58 (46.4%)
Married	60 (48%)
Divorced	7 (5.6%)
Education	
Did not graduate high school	1 (0.8%)
High school graduate or GED	5 (4%)
Some college/technical school/associates degree	28 (22.4%)
4-year college degree	77 (61.6%)
Some school beyond 4-year college degree	6 (4.8%)
Professional degree (e.g., M.D., J.D., Ph.D.)	8 (6.4%)
Primary pain location	
Back/neck	51 (40.8)
Head/face	29 (23.2)
Upper extremities	10 (8%)
Lower extremities	18 (14.4)
Chest/breast	7 (5.6%)
Stomach/abdomen	8 (8%)
Variable	$M$ ( $SD$ )
Age	32.97 (9.72)
Average daily drinks	1.62 (1.27)
AUDIT–total score	16.7 (11.41)
AUDIT–Hazardous Drinking cutoff (%)	67.2 ( $n = 84$ )
AUDIT–Consumption	5.34 (2.34)
AUDIT–Dependence	5.11 (4.83)
AUDIT–Harmful Use	6.25 (5.55)
GCPS–Pain Intensity	18.71 (4.71)
DERS score	98.49 (24.85)

Notes: GED = General Educational Development credential; AUDIT = Alcohol Use Disorders Identification Test; GCPS = Graded Chronic Pain Scale; DERS = Difficulties in Emotion Regulation Scale.

in hazardous alcohol use and correctly classified 80.8% of cases. Emotion dysregulation was also positively associated with each of the AUDIT subscale scores (Step 2  $ps < .01$ ; Table 2). In all models, greater DERS scores were associated with greater scores reflecting quantity/frequency of alcohol consumption, alcohol dependence symptoms, and harmful patterns of drinking.

#### *Indirect associations between emotion dysregulation and alcohol use outcomes via pain intensity*

We observed a statistically significant indirect association between emotion dysregulation and likelihood of scoring above the AUDIT hazardous alcohol use cutoff via pain intensity ( $b = 0.01$ ,  $SE = 0.01$ , bootstrapped 95% CI [0.004, 0.03]), such that higher levels of emotion dysregulation were associated with greater pain intensity ( $b = 0.07$ ,  $p < .001$ ),

which in turn was associated with a greater likelihood of endorsing hazardous drinking ( $b = 0.20$ ,  $p < .01$ ; Table 3). There was a direct association between DERS scores and the AUDIT hazardous alcohol use cutoff ( $b = 0.05$ ,  $SE = 0.01$ , bootstrapped 95% CI [0.02, 0.07],  $p < .001$ ). Exploratory analyses further revealed significant indirect associations between emotion dysregulation and all AUDIT subscales separately via pain intensity ( $ps < .01$ ; Table 3), such that higher levels of emotion dysregulation were associated with greater pain intensity, which was in turn associated with (a) greater quantity/frequency of alcohol consumption, (b) dependence symptoms, and (c) harmful patterns of drinking.

## Discussion

The current study presented a novel test of associations between emotion dysregulation, pain intensity, and hazardous alcohol use among individuals with chronic pain. Among the sample of past-month alcohol users with chronic pain, emotion dysregulation was found to be positively associated with the likelihood of scoring above the AUDIT cutoff indicating hazardous alcohol use, quantity/frequency of alcohol consumption, alcohol dependence symptoms, and harmful patterns of drinking. Importantly, these relations were observed after accounting for age, race, ethnicity, and gender, which have previously been associated with alcohol use outcomes (Brennan & SooHoo, 2013; Brennan et al., 2005, 2011; Chartier & Caetano, 2010; Nolen-Hoeksema, 2004; Paulus et al., 2017). We also observed a significant indirect association, such that greater emotion dysregulation was associated with greater pain intensity, which in turn was associated with greater AUDIT total and subscale scores. Collectively, these findings are the first to suggest that pain intensity may mediate the relationship between emotion dysregulation and hazardous drinking among individuals with chronic pain.

Given previously demonstrated associations between emotion dysregulation and pain intensity (Koechlin et al., 2018; Kökönyei et al., 2014; Overstreet & Goodin, 2018; Rogers et al., 2020), one possible interpretation of these results is that emotion dysregulation contributes to hazardous alcohol use among individuals with chronic pain indirectly via pain amplification. Individuals with chronic pain who experience difficulty regulating their emotions may experience their pain as more intense/impactful than those who are more adaptively able to respond to emotionally dysregulating situations (Kökönyei et al., 2014; Overstreet & Goodin, 2018; Rogers et al., 2020). Drinkers often report using alcohol to cope with pain (Brennan et al., 2005; Zale et al., 2015), and pain has been suggested to motivate alcohol consumption via desire to alleviate pain-related negative affect (Moskal et al., 2018; Zale et al., 2015). Thus, drinkers with chronic pain who have greater emotion regulation difficulties may be more likely to use alcohol



TABLE 2. Emotion dysregulation and alcohol outcomes

Variable	$\beta$	$t$	$p$	$\Delta R^2$	$p$
AUDIT–Consumption					
Step 1				.09	.15
Age	-.16	-1.64	.10		
Ethnicity	.10	1.06	.29		
Gender	.06	0.64	.53		
Race–American Indian/Alaska Native	.08	0.86	.39		
Race–Asian	-.03	-0.26	.80		
Race–Black	-.17	-1.79	.08		
Race–Other	.08	-0.87	.39		
Step 2				.09	.003*
Emotion dysregulation	.34	3.53	.001**		
AUDIT–Dependence					
Step 1				.34	<.001**
Age	-.20	-2.36	.02*		
Ethnicity	.29	3.48	.001**		
Gender	.07	0.89	.38		
Race–American Indian/Alaska Native	.13	1.68	.10		
Race–Asian	.19	2.17	.03*		
Race–Black	-.19	-2.34	.02*		
Race–Other	-.01	-0.10	.92		
Step 2				.13	<.001**
Emotion dysregulation	.40	5.22	<.001**		
AUDIT–Harmful Use					
Step 1				.38	<.001**
Age	-.22	-2.66	.01**		
Ethnicity	.35	4.29	<.001**		
Gender	.01	0.09	.93		
Race–American Indian/Alaska Native	.13	1.79	.08		
Race–Asian	.23	2.77	.01**		
Race–Black	-.03	-0.42	.67		
Race–Other	-.08	-1.11	.27		
Step 2				.16	<.001**
Emotion dysregulation	.46	6.47	<.001**		

Notes:  $n = 125$ . AUDIT = Alcohol Use Disorders Identification Test.

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

to cope in the context of greater pain. Indeed, hazardous drinking may be conceptualized as a maladaptive emotion regulation strategy aimed at alleviating both pain and negative affect experienced by individuals with chronic pain. Recent work focusing on the relationship between emotion dysregulation and pain has suggested that individuals who struggle to effectively manage the negative affect associated with pain experience an exacerbation of the severity of their pain symptoms (Rogers et al., 2020). The present findings extend this work by providing evidence linking the amplified experience of pain as a result of greater emotion dysregulation to an increase in the likelihood of engaging in risky alcohol use behaviors.

One potential clinical implication of these findings is that emotion dysregulation may function as a transdiagnostic vulnerability factor in comorbid pain and hazardous drinking. Transdiagnostic vulnerability factors are processes common across multiple disorders that are thought to be efficient targets for intervention (Krueger & Eaton, 2015). Decreasing these vulnerabilities may have general impacts across comorbid conditions (Krueger & Eaton, 2015), particularly among individuals with comorbid pain and substance use disorders (Ditre et al., 2019). Among

substance users with pain, pain-related anxiety, anxiety sensitivity, and distress intolerance have been implicated as factors contributing to both pain and substance use behaviors (Ditre et al., 2019; LaRowe et al., 2018, 2020; Rogers et al., 2019). These factors have been shown to influence pain experience and increased use of a variety of substances, including cigarettes, opioids, and alcohol (LaRowe et al., 2018, 2020; Rogers et al., 2019), and there is empirical interest in identifying other targets influencing these relationships (Zale et al., 2015). Because individuals with difficulties with emotion regulation may be more likely to turn to alcohol to self-medicate pain symptoms, implementing a transdiagnostic treatment approach may offer more adaptive strategies to improve pain-coping and decrease the likelihood of using alcohol to cope (Bradizza et al., 2018). Transdiagnostic interventions that address emotion regulation among individuals with chronic pain may thus simultaneously decrease pain and alcohol dependence. Characterizing the role of potential transdiagnostic factors, such as emotion dysregulation, in pain–substance relations is a necessary first step toward investigating their utility as intervention targets.

Several limitations of the present study should be noted.

TABLE 3. Emotion dysregulation and alcohol use: Indirect role of pain intensity

Model	Path	Path description	<i>b</i>	<i>SE</i>	<i>t</i> ( <i>z</i> )	<i>p</i>	CI (l)	CI (u)
Model 1 (Y1) criterion variable: AUDIT–Hazardous								
	<i>a</i>	DERS → GCPS-CPI	0.07	0.02	4.06	<.001**	0.03	0.10
	<i>b</i>	GCPS-CPI → AUDIT-Haz	0.20	0.07	-2.91	<.01**	0.06	0.33
	<i>c'</i>	DERS → AUDIT-Haz	0.05	0.01	-3.82	<.001**	0.02	0.07
Model 2 (Y2) criterion variable: AUDIT–Consumption								
	<i>a</i>	DERS → GCPS-CPI	0.07	0.02	4.06	<.001**	0.03	0.10
	<i>b</i>	GCPS-CPI → AUDIT-C	0.12	0.05	2.59	.01**	0.03	0.22
	<i>c</i>	DERS → AUDIT-C	0.03	0.01	3.67	<.001**	0.01	0.05
	<i>c'</i>	DERS → AUDIT-C	0.02	0.01	2.62	.01**	0.01	0.04
Model 3 (Y3) criterion variable: AUDIT–Harmful Use								
	<i>a</i>	DERS → GCPS-CPI	0.07	0.02	4.06	<.001**	0.03	0.10
	<i>b</i>	GCPS-CPI → AUDIT-H	0.33	0.08	3.97	<.001**	0.17	0.49
	<i>c</i>	DERS → AUDIT-H	0.12	0.02	7.19	<.001**	0.08	0.15
	<i>c'</i>	DERS → AUDIT-H	0.09	0.02	5.77	<.001**	0.06	0.12
Model 4 (Y4) criterion variable: AUDIT–Dependence								
	<i>a</i>	DERS → GCPS-CPI	0.07	0.02	4.06	<.001**	0.03	0.10
	<i>b</i>	GCPS-CPI → AUDIT-D	0.47	0.07	6.47	<.001**	0.33	0.62
	<i>c</i>	DERS → AUDIT-D	0.09	0.02	5.76	<.001**	0.06	0.12
	<i>c'</i>	DERS → AUDIT-D	0.06	0.01	4.00	<.001**	0.03	0.08

Notes: *n* = 125. CI (l) = confidence interval, lower limit; CI (u) = confidence interval, upper limit; DERS = Difficulties in Emotion Regulation Scale; GCPS-CPI = Graded Chronic Pain Scale–Characteristic Pain Intensity subscale; AUDIT-Haz = Alcohol Use Disorders Identification Test–Hazardous Use Cutoff; AUDIT-C = AUDIT–Consumption subscale; AUDIT-H = AUDIT–Harmful Use subscale; AUDIT-D = AUDIT–Dependence subscale. In all models, path *a* represents the association between the independent variable and the mediator; path *b* represents the associations between the mediator and the dependent variable, controlling for the independent variable; path *c* represents the total effect of the model (direct effect + indirect effect); and path *c'* represents the direct effect of the independent variable on the dependent variable.

\*\**p* < .01.

First, the study design is cross-sectional, and prospective research is needed to examine causal pathways and clarify the temporal relationship between emotion dysregulation, pain intensity, and hazardous alcohol use. Second, this study relied exclusively on self-report measures and is therefore limited by potential responding inaccuracies (Del Boca & Darkes, 2003). Future investigations would likely benefit from using experimental and/or ecological momentary assessment of emotion dysregulation, pain intensity, and alcohol use. Future research should also use more comprehensive measures of chronic pain and medical record checks to minimize potential inaccuracies and method variance associated with the use of self-report measures (Williams et al., 1989). Third, the current sample was limited to individuals who endorsed chronic musculoskeletal pain. Thus, the extent to which these findings may generalize to individuals with other chronically painful conditions (e.g., neuropathic pain) remains unclear. Fourth, generalizability may be further limited by composition of the study sample, which was limited to past-month alcohol users. Indeed, future research will be needed to determine whether these findings generalize to lighter drinkers with pain and individuals who are older and less well educated. Finally, although we hypothesize that pain-related negative affect also plays a mechanistic role in relations between emotion dysregulation, pain intensity, and hazardous alcohol use, we were unable to test these relationships with the data available from the parent study. Future work should test a serial mediation model to explore the contribution of negative affect to these relationships among individuals with chronic pain.

In summary, these findings suggest that emotion dysregulation may contribute to hazardous alcohol use among individuals with chronic pain, perhaps indirectly via pain amplification. Emotion dysregulation warrants consideration as a potential transdiagnostic vulnerability factor in comorbid chronic pain and hazardous drinking. Future research is needed to corroborate these findings and elucidate causal/temporal pathways between emotion dysregulation, the experience of pain, and hazardous drinking.

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