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## Analgesic Effect of Alcohol Mediates the Association between Alcohol Intoxication and Deliberate Self-Harm

Suzanne C. Amadi, Mitchell E. Berman, Matthew A. Timmins, Casey R. Guillot , Jennifer R. Fanning, Michael R. Nadorff, and Michael S. McCloskey

### ABSTRACT

We examined whether the analgesic effect of alcohol mediates the association between alcohol and deliberate self-harm (DSH) using data from a larger study on alcohol effects. Men ( $n = 106$ ) and women ( $n = 104$ ) low-risk alcohol drinkers (ages  $M = 26.00$ ,  $SD = 6.98$ ) recruited from the community who had no suicide attempt or episode of deliberate self-harm within the past year were randomly assigned to either a placebo drink condition or a drink calibrated to reach approximately .050%, .075%, or .100% blood alcohol concentration. Notable within-condition BAC variability, as well as overlap between conditions, suggested that BAC would be a more accurate indicator of intoxication compared to condition assignment. Pain tolerance was assessed by increasingly intense 1-s shocks delivered via fingertip electrodes. Self-reported pain associated with the pain tolerance index was also examined. A laboratory task of DSH, the Self-Aggression Paradigm, was then completed, with DSH operationalized as the number of self-administered shocks the participant was led to believe were twice the intensity of his or her pain tolerance and could cause “minor tissue damage that would quickly heal.” A negative binomial parallel mediational model for count data revealed that pain tolerance, but not self-report pain, mediated the effect of alcohol on DSH. As such, the current study provides preliminary experimental evidence that the analgesic effect of alcohol is partially responsible for link between alcohol intoxication and deliberate self-harm.

### KEYWORDS

Alcohol; pain; pain tolerance; self-aggression; self-harm

*Deliberate self-harm* (DSH) is a broad term that describes any self-directed, purposeful act that causes physical injury, with or without suicidal intent (Hawton & James, 2005). DSH behaviors range from *non-suicidal self-injury* (NSSI), which occurs in the absence of the intent to die (e.g., cutting, burning, scratching, head banging, and biting; Klonsky, 2011), to suicidal behavior, which is DSH with an intent to die, regardless of the lethality of the act (Hawton & James, 2005). NSSI and suicidal behavior are often co-occurring behaviors (Grandclerc, De Labrouhe, Spodenkiewicz, Lachal, & Moro, 2016), with less lethal forms of DSH predicting suicidal thoughts and behaviors (Whitlock et al., 2013). However, NSSI and other forms of non-lethal DSH occur far more frequently in the population compared to death by suicide (Centers for Disease Control & Prevention [CDC], National Center for Injury Prevention & Control, 2005–2015). For example, data from the CDC indicate that 495,348 non-fatal DSH

injuries resulting in emergency department visits and 48,344 suicides occurred in the United States in 2018. The number of non-lethal DSH incidents in the general population is likely even greater than reported estimates, as many DSH incidents, including NSSI, do not come to the attention of medical professionals or are not reported (Madge et al., 2008). Thus, examining possible risk factors for non-lethal DSH is critical given that such behaviors are a significant predictor of present and future suicidal thoughts and behavior (Whitlock et al., 2013).

Alcohol use has been suggested as a risk factor for DSH. The association between alcohol use and DSH has been supported by survey data garnered from clinical and non-clinical samples of adolescents and adults. For example, in a study of participants from several European countries, adolescents who reported multiple occasions of alcohol intoxication were at heightened risk to engage in non-lethal DSH—regardless of intent—compared to abstainers (Rossow et al., 2007). Similarly, in an Australian undergraduate sample, those who engaged in NSSI reported more alcohol misuse than those who did not engage in NSSI (Hasking, Momeni, Swannell, & Chia, 2008). In addition, alcohol use at baseline assessment was a predictor of lethal and non-lethal DSH one year later among Finnish adolescents who had endorsed depressive symptoms in outpatient care (Tuisku, Pelkonen, Kiviruusu, Karlsson, & Marttunen, 2012). Moreover, researchers found that alcohol use and misuse were associated with an increased risk of repeated DSH acts, as well as with suicide among adults in England (Ness et al., 2015). Among adults in a United States sample, researchers found that 20% of those who engage in NSSI reported being under the influence of alcohol or another psychoactive substance during an NSSI event (Klonsky, 2011). Therefore, there appears to be a reliable association between alcohol use and DSH across the spectrum of lethality.

Though non-experimental studies of alcohol use and DSH are of value, experimental studies using laboratory analogues of DSH are particularly informative as they allow for causal inferences regarding the effect of alcohol on DSH and help extricate the effects of chronic alcohol misuse from acute alcohol intoxication. The Self-Aggression Paradigm (SAP: Berman & Walley, 2003; McCloskey & Berman, 2003) is one such laboratory task that has been used to examine DSH, including the effects of alcohol on DSH. The SAP cover-task involves a series of competitive reaction-time trials against a (fictitious) opponent, and the participant (and ostensibly the opponent) is provided the opportunity to self-administer a shock on trials in which he or she “loses” (i.e., has the slower reaction-time). Evidence for the validity of the inferences drawn from the SAP include associations with self-report measures of suicidal disposition and self- and other-directed aggressive behavior. Importantly, SAP shock behavior is not accounted for by social desirability or motivation to win on the task (Berman & Walley, 2003; McCloskey & Berman, 2003).

To date, three separate studies using the SAP in non-overlapping samples found that acute alcohol intoxication elicits DSH in the laboratory (Berman, Bradley, Fanning, & McCloskey, 2009; Berman et al., 2017; McCloskey & Berman, 2003). For example, McCloskey and Berman (2003) assigned men who were not alcohol-dependent to either a placebo drink condition or an alcohol drink condition designed to reach a target blood alcohol concentration (BAC) of .10% on average. Participants in the alcohol condition self-administered both higher average shocks and selected an “extreme” shock

option (the highest shock option available and purportedly twice a pre-determined pain tolerance level) more frequently than participants in the placebo condition. Berman and colleagues (2009) found comparable results in regard to the selection of the “extreme” shock option in a veridical control drink condition (i.e., condition in which participants were informed that the control drink did not contain alcohol). In a third study, Berman and colleagues (2017) included both men and women in placebo (.000% BAC), low (.050% BAC), medium (.075% BAC), and high (.100% BAC) alcohol dose groups. DSH was again operationalized as the selection of an “extreme” shock, but with participants additionally being told that the “extreme” shock “may cause minor tissue damage.” Results revealed that men engaged in more DSH compared to women, as men more frequently selected the “extreme” shock. However, both men and women chose the “extreme” shock with greater frequency as a function of increasing BAC level. In a secondary data analysis of Berman et al. (2017), we sought to determine if state dissociation serves as an explanatory mechanism between alcohol intoxication and DSH (Timmins et al., 2020). Although alcohol intoxication produced BAC associated dissociation-like effects, state dissociation did not mediate the effect of alcohol intoxication on DSH.

Along with DSH, alcohol is also associated with increased pain tolerance, which is consistent with alcohol’s anti-nociceptive effects (James, Duthie, Duffy, McKeag, & Rice, 1978; Woodrow & Eltherington, 1988). In one such study, alcohol increased pain tolerance to electrical stimulation in a 0.10 g/dl ethanol condition among healthy participants without a notable personal or family history of alcohol misuse but did not increase pain tolerance to electrical stimulation among participants in a 0.04 g/dl ethanol condition (Perrino et al., 2008). Changes in mood state were also assessed, and the analgesic effect of ethanol was not related to alterations in mood, which suggested an independent analgesic effect of the ethanol. Additional support for the analgesic effect of alcohol on pain response has been demonstrated by authors of a quantitative literature review who found a general pain-dampening effect of alcohol in healthy participants (Horn-Hofmann, Büscher, Lautenbacher, & Wolstein, 2015). Further, a recent meta-analysis of 18 experimental studies found a small to moderate analgesic effect ( $g = 0.35$ ;  $p = .002$ ) of alcohol administration on pain response (i.e., pain threshold and tolerance; Thompson, Oram, Correll, Tsermentseli, & Stubbs, 2017). A moderate to large effect was found for reduction in pain intensity self-ratings after alcohol administration. Moreover, the effects of alcohol appeared to be dose-dependent, as increases in BAC were associated with increases in pain threshold and decreases in self-rated pain intensity (Thompson et al., 2017).

It is important to note that research that involves the administration of alcohol is generally limited to healthy participants due to guidelines endorsed by the National Advisory Council of Alcohol Abuse and Alcoholism for ethical and safety considerations when using human subjects (National Institute on Alcohol Abuse & Alcoholism, n.d.). However, studies using healthy participants are still of importance as they provide useful preliminary data of alcohol’s effect on pain tolerance, which can help identify risk factors that are relevant to those who engage in DSH.

Research to date has also established an association between pain tolerance and DSH. In one study, individuals with borderline personality disorder (BPD) who reported on a

lack of pain during NSSI (their responses on a questionnaire) took more time to release their hand during the cold pressor task compared to both healthy controls and individuals with BPD who reported experiencing pain during NSSI (Russ et al., 1992). In addition, Hooley, Ho, Slater, and Lockshin (2010) found that those who had a history of DSH had higher pain tolerances during a pressure algometer task than those who did not have a history of DSH (Hooley et al., 2010). Also, Hamza, Willoughby, and Armiento (2014) generally replicated this previous finding using a cold pressor task as the painful stimulus. Specifically, the researchers found evidence that participants who engaged in NSSI and endorsed self-punishing motives endured pain longer during the cold pressor task than participants who engaged in NSSI and did not endorse self-punishing motives, as well as longer than participants who did not report any NSSI (Hamza et al., 2014). The latter finding suggested that the relationship between pain and DSH may depend on individual differences in beliefs about pain experiences.

In summary, alcohol consumption is positively associated with DSH both in real-world settings (e.g., Klonsky, 2011; Rossow et al., 2007) and the laboratory (Berman et al., 2009; 2017; McCloskey & Berman, 2003). Additionally, research has established that alcohol intoxication is associated with greater pain tolerance (Perrino et al., 2008), and pain tolerance has been associated with DSH (Hooley et al., 2010). It is therefore reasonable to posit that alcohol's association with DSH might be partially accounted for by alcohol's analgesic properties. The aim of this study was to examine the effect of experimentally manipulated BAC levels on pain tolerance, as well as the potential mediating effect of pain tolerance on a laboratory analogue of DSH. To test the proposed relationships, the variables of interest were retrieved from an archival dataset from a larger study designed to test the effects of alcohol on DSH in men and women (Berman et al., 2017). We predicted that increasing BAC would be positively associated with pain tolerance, which in turn would mediate the relation between BAC and DSH.

## METHOD

### *Participants*

Participants consisted of a community sample of 210 (104 women, 106 men) low-risk alcohol drinkers aged 21 to 54 years-old ( $M = 26.02$ ,  $SD = 6.97$ ) who self-identified as Caucasian (65.6%), African American (24.4%), Hispanic (3.8%), or other ethnicity (6.2%) and were currently located in the Southeastern region of the United States. Participants were recruited to participate in a study advertised as "The effects of alcohol on motor skills." About 43% of participants received up to a high school education or equivalent, 20% received up to an associate degree, 22% received up to a bachelor's degree, and 10% received up to an advanced degree (e.g., master's, doctoral degree). Approximately 76% of participants were never married.

Respondents underwent a prescreening via telephone interview. Potential participants were excluded if they indicated they had never consumed alcohol or endorsed criteria for alcohol dependency. Criteria for alcohol dependency included having any score above 8 on the Alcohol Use Disorders Identification Test (AUDIT; Saunders, Aasland, Babor, De La Fuente, & Grant, 1993). If the participant scored a 7 or 8 on the AUDIT, they completed the Short Michigan Alcoholism Screening Test (SMAST; Selzer,

Vinokur, & van Rooijen, 1975) and were excluded if they scored 3 or higher on the SMAST. Participants were classified as a (“social drinker”) if they used alcohol within the past year but did not report problematic drinking behavior based on the prescreening procedure. Other exclusion criteria included participation in an alcohol or shock study in the same research setting, current medication that would contraindicate alcohol consumption, a neurological condition that would preclude electric shock stimulation, pregnancy (determined via a urine assay on the day of the study), nursing, history of bipolar or psychotic disorder, a history of substance use treatment, and the inability to adhere to a 1-week lead-in protocol proceeding the scheduled session in the laboratory (i.e., no medication that could interact with alcohol for 1-week before the study; no food consumption on the day of the study; no alcohol consumption for 48-h before the study).

Participants invited for further evaluation provided informed consent and completed a health status questionnaire that included a closed and open-ended question about attempts of DSH within the past 12 months. Participants were excluded if they had any suicide attempt and/or engaged in DSH that required medical attention within the past year. This precaution was in place so as not to administer alcohol to an individual with relatively recent marked suicidality. However, no volunteer was eliminated based on this criterion. A total of 4.3% of participants (9 participants) responded “yes” to the question related to having ever attempted to self-harm.

An IQ of 85 or below (i.e., borderline or below average intellectual functioning), as measured by the Wechsler Abbreviated Scale of Intelligence, was exclusionary (though no participant was excluded based on this criterion). Participants were also excluded if, at the appointed session, they had a positive urine toxicological screen for cannabis, opioids, benzodiazepines, methamphetamine, or cocaine, or if they had an expired-breath BAC estimate greater than .000%. Participants received monetary compensation for their participation in the study.

### ***Alcohol Manipulation***

Each participant was assigned to either a placebo (.000% BAC target; 24 women and 26 men), low-alcohol (.050% BAC target; 23 women and 24 men), medium-alcohol (.075% BAC target; 26 women and 29 men), or high-alcohol (.100% BAC target; 31 women and 27 men) dose condition using a pseudo-random number generator. Group composition did not significantly differ as a function of gender,  $\chi^2(3) = .52, p = .91$ .

Participants in all conditions were told that the drink contained alcohol but were blind to group assignment. They were informed that the amount of alcohol they receive would be decided “randomly, like flipping a coin,” and be dependent on height, weight, and gender. They were also told that the amount of alcohol they receive would produce about the same level of intoxication in both them and another participant (their opponent). Additionally, they were told that the most alcohol anyone in the study would receive would be about the same as 1.5 ounces or one “shot” of 100 proof alcohol for every 40 pounds of body weight, enough alcohol to make them legally intoxicated with blood alcohol level to or around .10 before playing the game against another participant.

Participants in the low, medium, and high dose groups were provided two cups containing orange juice and 190-proof (95% ethanol) grain alcohol to achieve a 5:1 orange juice to alcohol mixture. The placebo dose group was given orange juice equal in volume to the orange juice given to those in the medium dose group with a few drops of alcohol added to the surface of the drink and rubbed around the rim of the cup. We used a placebo group rather than a veridical control (that is, participants are informed their drink contains no alcohol) because (a) placebo (McCloskey & Berman, 2003) and veridical (Berman et al., 2009) controls produce similar effects for alcohol-SAP studies, and (b) to provide consistent instructions across the four groups regarding the potential for receiving alcohol. The amount of alcohol per drink was also adjusted for weight and gender to achieve, on average, target BACs before the behavioral tasks (Watson, Watson, & Batt, 1981).

Participants were given 15, 22.5, or 30 min to consume the drink in the low, medium, and high doses, respectively. A 20-min waiting period followed drink completion to reach target BACs during the SAP procedure. BAC was assessed using an expired-breath sample obtained with an Alco-Sensor IV (Intoximeters, Inc., St. Louis, MO) hand-held breathalyzer. Participants in the placebo condition were given 22.5 min (the average consumption time for the low, medium, and high doses) to finish the drink. For each participant, averaged BACs before and after the behavioral tasks were used as a biological index of alcohol intoxication during the SAP.

### ***Pain Tolerance Assessment***

The SAP consists of a pain tolerance procedure, followed by the reaction-time task. Fingertip electrodes were attached to the middle and index fingers on the participant's non-dominant hand, followed by a series of increasing 1-s shocks at 100- $\mu$ A intervals. In the current study, pain tolerance was operationalized as the microampere level at which the participant both indicated the shock was "painful" and voiced an unwillingness to go higher. The pain tolerance protocol had a maximum 2.50 mA limit set to ensure participant safety. If the participant did not indicate pain tolerance by the maximum shock, their pain tolerance was recorded as the maximum intensity. The procedure was repeated for a fictitious opponent, and audiotaped responses of same-sex actors were played over an intercom for the participant to enhance the credibility of the cover task. Self-rated pain associated with the pain tolerance electrical stimulation assessment served as a secondary index of pain experience. Specifically, the participant was asked to respond to the following question: "How painful was the highest shock you took during the threshold procedure?" Responses ranged from 1 = *Not at All* to 8 = *Very Much*.

### ***The Self-Aggression Paradigm***

The SAP task immediately followed the pain tolerance procedure. The SAP is a competitive reaction-time task consisting of 40 trials with predetermined 50% wins and 50% losses. During the SAP, participants competed against a fictitious "opponent" whose behavior was computer-controlled and pre-programmed. No feedback about the opponent's shock selections was provided. When a participant "lost" a trial, they were given the opportunity to select a shock level. The range of shock intensities included 0 to 10,



with 10 being the same intensity as the pain tolerance level. Participants also had the option to select a “20” shock, which was described to the participants as an “extreme” shock. The 0 option was included to increase the ecological validity of the task and give participants the opportunity to not administer a shock. Participants were told that if the extreme shock was selected, a shock twice the pain tolerance shock would be delivered that could produce “minor tissue damage” that would quickly heal. In actuality, the extreme shock option was programmed to deliver a shock equal to the 10 shock. The 9 shock was set at 95% of the 10 shock, 8 at 90%, 7 at 85%, and so forth. DSH was operationalized as the total number of extreme 20 shocks self-administered during the 40 trials. BAC was again measured after participants completed the SAP. After completing the SAP, the participant completed a post-task questionnaire that included the item assessing pain associated with maximum shock received during the pain tolerance assessment. When BAC was below .02%, the participant was dismissed.

## RESULTS

Overall, pre- to post-SAP BAC level ranged from 0.00 to 0.15 ( $M=0.06$ ,  $SD=0.04$ ). Except for the placebo group, BACs within each dose condition showed substantial variability and overlap across groups: .01 to .05 BAC in the low-alcohol group ( $M=0.05$ ,  $SD=0.01$ ); .04 to .011 BAC in the medium-alcohol group ( $M=0.07$ ,  $SD=0.02$ ), and .03 to .015 BAC in the high-alcohol group ( $M=0.07$ ,  $SD=0.02$ ). Given the variability within and overlap across drink conditions, BAC as a continuous independent variable was used to provide a more meaningful approach to examine the relationships of interest. With the inclusion of the placebo group, observed skew was minimal [ $-.14$  ( $SE=.17$ )]. Gender and BAC were not significantly related,  $r_{pbi}=.03$ ,  $p=.67$ .

Participants' pain tolerance ranged from 0.32 mA to 2.52 mA ( $M=1.60$ ,  $SD=0.79$ ). Self-rated pain ranged from 0 through 8 ( $M=4.18$ ,  $SD=2.23$ ). Sixty-five participants selected the extreme shock at some point during the SAP (31.1% of sample). The number of times that each of those participants selected the extreme shock ranged from 1 to 20.

Average BAC was positively correlated with the number of total extreme shocks selected during the SAP ( $r=.25$ ,  $p<.001$ ) and with pain tolerance ( $r=.22$ ,  $p=.001$ ). Average BAC was not significantly correlated with self-rated pain ( $p=.064$ ). Pain tolerance was positively correlated with total extreme shock ( $r=.35$ ,  $p<.001$ ), and self-rated pain was negatively correlated with total extreme shock ( $r=-.16$ ,  $p<.05$ ). Pain tolerance was negatively correlated with self-rated pain ( $r=-.27$ ,  $p<.001$ ).

MPLUS Version 8.2 (Muthén & Muthén, 1998–2017) was used to conduct a parallel mediation analysis with both pain tolerance and self-rated pain as mediators of the relation between alcohol intoxication and the number of extreme shocks selected during the SAP. A negative binomial model for the extreme shock count dependent variable was used based on its Bayesian Information Criteria value. Bootstrapped (10,000) standard errors and maximum likelihood model estimation were used to test the model.

The total effect from BAC to extreme shock was significant,  $b=14.27$ ,  $SE=7.23$ ,  $p=.049$  (95% CI [4.16, 31.49]). When the two mediators were included in the model, the direct effect from BAC to extreme shock was no longer significant,  $b=9.33$ ,  $SE=6.96$ ,  $p=.18$  (95% CI [−0.72, 25.70]). In addition, a significant total indirect effect emerged,

$b = 4.95$ ,  $SE = 1.96$ ,  $p = .01$  (95% CI [1.87, 9.51]), suggesting that one or both mediators account for the difference between the total and direct effect.

Examination of the two specific indirect effects revealed a significant BAC  $\rightarrow$  pain tolerance  $\rightarrow$  total extreme shock indirect effect,  $b = 4.63$ ,  $SE = 1.96$ ,  $p = .02$  (95% CI [1.58, 9.26]). However, the BAC  $\rightarrow$  self-rated pain  $\rightarrow$  total extreme shock indirect effect was not significant,  $b = 0.32$ ,  $SE = 0.65$ ,  $p = .63$  (95% CI [-0.72, 1.92]). Further examination revealed that although pain tolerance and self-rated pain were negatively related,  $b = -0.43$ ,  $SE = 0.12$ ,  $p < .001$  (95% CI [-0.66, -0.19]), only pain tolerance was significantly related to BAC  $b = 4.47$ ,  $SE = 1.37$ ,  $p = .001$  (95% CI [1.78, 7.15]).

## DISCUSSION

Results of this study support the notion that experimentally altered pain tolerance via alcohol administration is causally related to DSH observed under controlled laboratory conditions. Specifically, the analgesic effect of alcohol while intoxicated in part facilitated the use of the “extreme” shock during the SAP task. This finding builds upon previous research on alcohol consumption and SAP performance by uncovering a mechanism that in part explains how alcohol facilitates the risk of engaging in DSH.

Hooley and Franklin (2018) proposed a benefits and barriers model for NSSI that includes physical pain as a barrier to NSSI. Accordingly, physical pain, which is usually avoided, can serve a self-punishing function for individuals who engage in DSH across time. However, the model generally does not account for analgesic effect of alcohol. Given the co-morbidity between alcohol use and DSH, alcohol could potentially facilitate DSH in individuals with a life-history of DSH by decreasing an important barrier to self-harm; that is, the general avoidance of pain. It is important to note that an insufficient number of individuals with a history of DSH were available in the sample to test this notion (4.3%). Building on the results of the current study, we encourage researchers to address this important question by recruiting and oversampling individuals with clinically-meaningful history of DSH and placing addition precautions in place for alcohol administration with this vulnerable population.

There are several caveats worth noting for this study. For safety reasons, there was a limit on the maximum shock intensity used during the pain tolerance procedure. It is possible that some participants may have gone beyond our maximum limit if the limit was not in place. In the current study, 28% of participants reached the pain tolerance maximum limit, including 22 participants in the .100% BAC group. This ceiling effect might have truncated the range of potential tolerance levels observed. Keep in mind that the laboratory study of DSH requires a proxy of real-life DSH that can both help us inform risk for DSH and at the same time protect the safety of research participants.

In addition, high-risk drinkers were excluded from the study. Inclusion of drinkers at risk for alcohol dependence was considered, but not justifiable given the aim of the original study was to first demonstrate an association between alcohol intoxication and DSH in a proof of principle experimental design. Thus, the inclusion of a sufficient number of individuals at risk for alcohol dependence and individuals who actively engage in self-harm behaviors was beyond the scope of this study but worthy of future investigation.

When both pain tolerance and pain self-ratings of the highest shock were included as mediators in the model, a significant indirect effect emerged for pain tolerance but not for self-ratings of the highest shock. Notably, self-ratings of the pain associated with the tolerance electrical stimulation were inversely associated with the pain tolerance index. Thus, participants with higher pain thresholds tended to rate the shock as less painful, which is intuitive. In addition, self-ratings of the pain tolerance shock were inversely associated with extreme shock settings. That is, individuals who rated the threshold shock as more painful tended to make less use of the 20 shock, which is also intuitive. The relation between BAC and pain-ratings, although in the expected direction, were not significant, which could account for the null finding for that indirect path.

Although alcohol's analgesic effect was observed via alterations in pain tolerance, alcohol's deleterious effects on the ability to process information (Guillot, Fanning, Bullock, McCloskey, & Berman, 2010; Peterson, Rothfleisch, Zelazo, & Pihl, 1990; Thompson et al., 2017) might have interfered with the accuracy of pain self-ratings, which were assessed after completing the reaction-time task. It is not possible to determine from these results, however, if self-ratings were inaccurate due to impaired cognition associated with alcohol intoxication. Given the inherent limitation of the one-item measure used to assess pain perception in the current study, researchers should consider the use of multi-item pain self-rating measures tailored to the stimulus characteristics used to elicit pain in future studies. Finally, although electric shock has its advantages when used as a pain threshold index, such as reproducibility and ease of quantification, other approaches provide potentially complementary information (e.g., cold pressor, thermal heat; see Ammerman, Berman, & McCloskey, 2018 for a review). Thus, a multi-modal approach to pain assessment would be useful to consider in future studies.

Another limitation of the study is that the affective aspects of pain perception were not considered in the context of alcohol intoxication. Pain is both a sensory and emotional experience (International Association for the Study of Pain Task Force, 1979). For one individual, pain can be perceived as discomforting (stabbing, shooting, exhausting), but for another individual, pain can be perceived as exhilarating (e.g., tension releasing). In addition, research shows that emotion dysregulation is associated with diminished pain perception and DSH (Franklin, Aaron, Arthur, Shorkey, & Prinstein, 2012). Diminished pain perception in the context of emotion dysregulation could be due to decreased attention on pain sensation and increased attention on the emotional experience of pain. Thus, examination of pain and emotion in the context of alcohol and DSH is an important future area of inquiry.

It is important to note that alcohol intoxication does not invariably lead to self-harm episodes, and that not all incidents of DSH occur when under the influence (see Anestis, Joiner, Hanson, & Gutierrez, 2014 for a discussion of these issues). Thus, given the design limitations of experimental behavioral pharmacological studies of alcohol in humans, extrapolating these findings to extra-laboratory venues should be done with caution. However, the finding that alcohol-related analgesia seemingly plays a role in DSH when intoxicated provides a potentially meaningful framework for understanding why the risk of DSH is heightened when intoxicated.

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## AUTHOR NOTES

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