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Abstract

Objective: Non-experimental survey and field research support the notion that alcohol use may be associated with deliberate self-harm (DSH) across the spectrum of lethality, from non-suicidal self-injury (NSSI) through suicide. Non-experimental studies, however, provide limited information about potential causal relationships between alcohol consumption and DSH. Two previous experiments showed that a relatively high-dose of alcohol increases the likelihood of engaging in DSH in the laboratory, defined by the self-administration of a "painful" shock (*the Self-Aggression Paradigm: SAP*; Berman & Walley, 2003; McCloskey & Berman, 2003). In this study, we examined whether (a) lower doses of alcohol also elicit DSH, (b) this effect occurs for women as well as men, and (c) individual differences in past non-suicidal self-injury (NSSI) moderate alcohol's effects on DSH. **Method:** Non-alcohol dependent men and women (N = 210) were assigned either to .00%, .05%, .075%, or .100% blood alcohol concentration (BAC) drink conditions and completed a self-rating scale of NSSI (*the Deliberate Self-Harm Inventory: DSHI; Gratz, 2001*). As in previous SAP studies, DSH was operationalized by shock setting behavior during a competitive reaction-time game. **Results:** Overall, a greater proportion of participants in the .075% and .100% (but not .050%) alcohol conditions self-selected a "painful" shock to administer compared to participants in the placebo condition. NSSI predicted self-administration of painful shocks, but did not moderate the alcohol effect. **Conclusions:** Results provide experimental evidence to support the notion that interventions for self-harm should include processes to monitor and limit alcohol intake.

Keywords: deliberate self-harm, NSSI, alcohol, dose-response, self-aggression paradigm

Public health significance: Results highlight the importance of addressing alcohol use patterns in interventions intended to reduce the risk of deliberate self-harm.

Effects of Alcohol Dose on Deliberate Self-Harm in Men and Women

Deliberate attempts to cause physical harm or injury to oneself, irrespective of lethal intent, fall under the broad umbrella of deliberate self-harm (DSH; Muehlenkamp, Claes, Havertape, & Plener, 2012). DSH includes a wide range of complex and multi-determined behaviors, from non-suicidal self-injury (NSSI), which is not motivated by a desire to cause death (e.g., skin cutting, burning of the skin, head banging (Klonsky, 2011; Muehlenkamp et al., 2012; Swannell, Martin, Page, Hasking, & St John, 2014) through death by suicide (Muehlenkamp et al., 2012).

Although suicide was the tenth leading cause of death (42,773) in 2014, it remains a relatively rare event in the population (age adjusted rate of about 13 per 100,000). In contrast, the age adjusted rate for non-fatal occurrences of DSH in the United States in 2014 was more than ten times as great (152 per 100,000; Centers for Disease Control and Prevention, National Center for Injury Prevention and Control, 2005). These data likely underestimate the extent of non-fatal DSH, as they include only individuals who came to the attention of medical personnel. It is important to note that these data do not consider the motivation for the act; that is, whether a desire to die was present at the time.

NSSI is also not uncommon in the population (Klonsky, 2011; Muehlenkamp et al., 2012; Swannell et al., 2014). Two separate quantitative reviews of NSSI in adolescents revealed similar overall life-time prevalence rates of 18% (Muehlenkamp et al., 2012) and 17.2% (Swannell et al., 2014), respectively. Recent estimates suggest that lifetime prevalence rates for adults are around 5-6% (Klonsky, 2011; Swannell et al., 2014).

Non-lethal and lethal DSH differ in methods used, clinical correlates, functions, and developmental trajectory (Andover & Gibb, 2010; Boxer, 2010; Liotta, Mento, & Settineri, 2015; Nock, Joiner, Gordon, Lloyd-Richardson, & Prinstein, 2006). Despite these differences, non-lethal DSH, including NSSI, and suicidal behaviors often co-occur within individuals (Victor & Klonsky, 2014). Indeed, NSSI has been shown to predict future suicide ideation and attempts above other established risk-factors (Guan, Fox, & Prinstein, 2012) and individuals who engage in NSSI often do so while feeling suicidal (Klonsky, 2011). Given the frequency with which non-lethal DSH (including NSSI) occurs in the population, and its relation to suicide, identifying causal risk factors for non-lethal DSH seems as important as identifying those for suicidality.

Alcohol misuse has long been associated with DSH across the spectrum of lethality. For example, moderate to heavy drinking patterns were associated with higher levels of DSH in a large cross-national sample of adolescents (Rossow et al., 2007). Similar results were found for a non-clinical community sample of university students. Specifically, participants who engaged in moderate-severe non-suicidal DSH reported more risky drinking compared to those who did not (Hasking, Momeni, Swannell, & Chia, 2008). In depressed adolescents receiving outpatient treatment, alcohol use patterns predicted both deliberate self-harm and suicidality at one year follow up (Tuisku, Pelkonen, Kiviruusu, Karlsson, & Marttunen, 2012).

The relation between NSSI and alcohol use has also been examined. Whitlock and colleagues, using a large cross-university student sample, found that 18% of students who engaged in NSSI reported doing so while under the influence, with men four times as likely to report intoxication as a precipitating factor (Whitlock et al., 2011). Klonsky (2011), in a random sample of adults (N = 439) interviewed by telephone, found that 20% respondents who engaged

in NSSI during their lifetime admitted to being under the influence of alcohol or drugs at the time, and 15% received treatment for substance misuse. Given that DSH is multi-determined, it is not surprising that alcohol use is a modest correlate of DSH and is neither necessary nor sufficient for DSH to occur.

As can be seen, evidence for a link between alcohol intoxication and DSH is complex and comes almost exclusively from non-experimental studies employing self-report. Unpacking the acute effects of alcohol intoxication from chronic alcohol misuse on non-lethal DSH using cross-sectional designs is not possible (Hufford, 2001). Indeed, the co-occurrence of DSH and acute alcohol intoxication could be accounted for by the indirect effects of chronic alcohol misuse. Specifically, long-term social, medical, and financial problems associated with chronic alcohol misuse could lead to both self-harm and alcohol ingestion as a strategy to manage negative affect. Importantly, the results of non-experimental studies, though suggestive, do not help to establish that ingestion of alcohol had a *causal* effect on DSH. Specifically, the temporal relation between alcohol use and self-harm might be in the opposite direction, with the decision to engage in self-harm preceding ingestion of alcohol (Hayward, Zubrick, & Silburn, 1992; Hufford, 2001). A pressing concern for the field, therefore, is to understand when and for whom alcohol intoxication confers risk for DSH (Kaplan, Giesbrecht, et al., 2013).

Of course, DSH with lethal intent cannot be studied experimentally in the laboratory. However, non-suicidal DSH can be studied under controlled laboratory conditions, providing one means for better understanding the link between alcohol intoxication and self-harm. The Self-Aggression Paradigm (SAP) is a laboratory analog of non-suicidal DSH that has been used to explore variables associated with DSH (Berman, Bradley, Fanning, & McCloskey, 2009; Berman, Jones, & McCloskey, 2005; Berman & Walley, 2003; McCloskey, Ben-Zeev, Lee,

Berman, & Coccaro, 2009; McCloskey & Berman, 2003; McCloskey, Look, Chen, Pajoumand, & Berman, 2012).

Experiments using the SAP have shown that men with no history of alcohol dependence who consumed a dose of alcohol targeting a .100% BAC were more likely to engage in deliberate self-harm, defined as the selection of an ostensibly "painful" shock to self-administer, compared to men who consumed a placebo (McCloskey & Berman, 2003) or a veridical non-alcoholic drink (Berman, Bradley, et al., 2009). Although suggestive of a causal link, these studies did not address whether the effects of alcohol on DSH are dose dependent or if this effect emerges at lower doses. If "turning up" the dose increases the likelihood of DSH, such findings would provide strong complementary evidence that alcohol intoxication is a causal risk factor for self-harm. Examination of various doses of alcohol would also identify the level of alcohol intoxication that confers risk for DSH.

Previous SAP studies explored the effects of alcohol on DSH in men only. Although several reviews have examined the role of gender in alcohol intoxication and suicide (Anestis, Joiner, Hanson, & Gutierrez, 2014; Kaplan, Mcfarland, et al., 2013; Norström & Rossow, 2016), the role of gender in alcohol-related non-suicidal DSH has not been adequately examined (but see Whitlock et al., 2011). Studying the effects of alcohol on self-harm using the SAP allows for alcohol dose to be adjusted for gender differences and body mass index. Given that alcohol intoxication increases other-directed aggressive responding in a dose-dependent fashion in both men and women (Duke, Giancola, Morris, Holt, & Gunn, 2011), and that alcohol is associated with self-harm in both men and women in field, we expected to find the effects of alcohol on self-harm to emerge for both men and women when alcohol dose is equated experimentally.

Given the imperfect relation between alcohol and DSH, the expression of DSH in intoxicated individuals certainly involves moderator variables. One potential variable is past self-harm behaviors. With respect to behaviors intended to harm others, the expression of other directed aggressive behavior in the laboratory (including the effects of pharmacological manipulations) depends to a degree on past aggressive acts (Berman, McCloskey, Fanning, Schumacher, & Coccaro, 2009). Similarly, a history of self-harm might predispose individuals to the engage in DSH when intoxicated.

Our first aim was to determine if self-harm behavior increases as a function of dose, and if this effect emerges for both men and women. A second aim was to determine if past NSSI moderates the effects of alcohol on SAP self-harm. Participants (N = 210) were assigned to consume either a placebo control drink or an alcoholic drink apportioned to produce an average BAC of either .050%, .075%, or .100% and then completed a laboratory task designed to assess non-suicidal DSH. We expected that the effects of alcohol on a laboratory analogue of DSH would be dose dependent, with a positive association between level intoxication and SAP shock selections. We also expected that alcohol effects would be dependent on history of NSSI such that as the severity of past NSSI increases so would the likelihood of selecting a "painful" shock when intoxicated.

Method

Participants

Healthy social drinkers (N = 210; 104 women; 21 through 55 years old) were recruited from the community for a study on "the effects of alcohol on motor skills." Potential participants were screened via telephone. Alcohol dependence was identified by scores greater than 8 on the Alcohol Use Disorders Identification Test (AUDIT; Saunders, Aasland, Babor, De La Fuente, &

Grant, 1993); participants scoring in the borderline range on the AUDIT (a score of 8 through 9) were also administered the Short Michigan Alcohol Screening Test (SMAST; Selzer, Vinokur, & Van Rooijen, 1975) and were excluded with a score of 3 or more.

Other exclusions on the telephone screen were: Prior participation in alcohol- or shock-related research in our lab; having never consumed alcohol; currently prescribed medication that precludes alcohol consumption; pregnancy or nursing; current mood or psychotic disorder or other severe psychological problem requiring treatment; a significant medical condition such as kidney or liver problems; a history of medical problems due to alcohol use; or the inability to participate in a one-week medication free lead-in period. Participants were asked to refrain from alcohol use for 48 hours before the study, and to not eat anything before the scheduled appointment.

On the alcohol administration day, urine toxicological screening (cannabis, opioid, benzodiazepine, methamphetamine, cocaine) and an expired-breath BAC assay were conducted. A positive result (including BAC > .000%) was exclusionary A health-questionnaire was also administered and reviewed with the participant by doctoral students in a clinical psychology program. Those with a history of treatment for alcohol or drug use were excluded, as well as those who have had a suicide attempt or self-injury requiring medical attention in the previous 12 months.

Self-identified race and ethnicity of the sample was 65.2% Caucasian, 24.8% African American, 3.8% Hispanic, and 6.2% “Other.” The University of Southern Mississippi Human Subjects Protection Review Committee approved the study procedures and consent process.

Measures

The Deliberate Self-Harm Inventory (DSHI; Gratz, 2001). The DSHI is a 17-item self-rating scale designed to assess non-lethal self-harm conceptualized as "… the deliberate, direct destruction or alteration of body tissue without conscious suicidal intent, but resulting in injury severe enough for tissue damage (e.g., scarring) to occur" (*p*. 255). Respondents are asked, "Have you ever intentionally (i.e., on purpose)" engaged in a series of self-harm behaviors (e.g., "Cutting?": "Burned yourself with a lighter or a match?"; "Carving words into skin?"). "Yes" was coded as "1." "No" was coded as "0." A total score (from 0 through 17) measures extent of self-harm behaviors. The DSHI has acceptable internal consistency (α = .81 in the present sample). Evidence for the validity of the DSHI is reported elsewhere (Gratz, 2001). 21.4% of participants endorsed one or more items (scores ranged from *0* through *10*; M = 0.47; SD = 1.38). The DSHI scores were $\log_{10}$-transformed to adjust for positive skew (Gratz, 2001). The DSHI was embedded in a battery of self-report measures assessing a wide range of personality and behavioral variables.

Behavioral analog of self-harm. The Self-Aggression Paradigm (SAP; Berman & Walley, 2003; McCloskey & Berman, 2003) was administered on a separate day from the DSHI, with the DSHI being completed first in about half the cases. During the SAP, the participant is provided with the opportunity to self-administer electric shock throughout a series of reaction-time trials with a fictitious opponent. Intensity of self-administered shock on a given trial is completely under the control of the participant, with 12 possible shock intensities: "0" (no shock on that trial), "1" through "10" (with "10" being equivalent to a pain threshold determined before the task), and "20" (which the participant is told is twice as intense as the "10" pain threshold and could produce "minor tissue damage." The "20" shock, if selected, produced the same shock intensity as the "10" shock.

DSH was defined in two ways: (1) selection of the "20" shock at least once; and (2) the total number of times the "20" shock was selected. Validity for the SAP is supported by positive associations with both self-ratings of suicidal thoughts and behaviors and a history of non-suicidal self-injury (Berman et al., 2005; Berman & Walley, 2003; McCloskey et al., 2012). Discriminant validity for the SAP is supported by lack of associations with self-ratings of anxiety or performance or desire to win on a reaction-time cover task (Berman & Walley, 2003). In contrast to self-ratings of self-harm, the SAP does not appear to be contaminated by social desirability (Berman & Walley, 2003)—an advantage compared to self-ratings of self-harm.

External validity for the SAP is supported by the notion that the laboratory correlates of SAP behavior parallel those observed for extra-laboratory ("real-world") self-injurious behaviors, including compromised 5-HT functioning (McCloskey et al., 2009), benzodiazepine consumption (Berman et al., 2005), alcohol intoxication (Berman, Bradley, et al., 2009; McCloskey & Berman, 2003), history of depression (McCloskey, Gollan, & Berman, 2008), and model effects (Berman & Walley, 2003; Sloan, Berman, Zeigler-Hill, & Bullock, 2009). In the absence of pre-disposing conditions (e.g., social influences, suicidal history, alcohol intoxication) the 20 shock is rarely selected. Stability of SAP behavior is supported by relatively consistent shock selections across blocks of trials in the absence of experimental influences (Berman & Walley, 2003; McCloskey & Berman, 2003).

Procedure

Participants were randomly assigned to one of four alcohol drink groups: Placebo (.000% BAC; 24 women and 26 men), "low dose" (.050% BAC; 23 women and 24 men), "medium dose" (.075% BAC; 26 women and 29 men), and "high dose" (.100% BAC; 31 women and 27 men). Participants in the low, medium, and high dose conditions were given a mixture of chilled

orange juice and 190-proof grain alcohol (95% ethanol) divided between two cups. Orange juice was added to achieve a 5:1 orange juice to alcohol mixture. The volume of the alcohol drinks was based on an equation incorporating weight and gender in order to achieve a target BAC (Watson, Watson, & Batt, 1981). All participants, including those in the placebo condition, were told that the drink could contain alcohol, but no additional information was provided about the drink. For the placebo condition, participants were administered a drink of chilled orange juice approximately equivalent in volume to a typical .075% BAC medium dose with a few drops of alcohol floated on top of the drink and rubbed around the rim of the cups.

Participants were given 15, 22.5, or 30 minutes to consume the drink in the low, medium, and high doses, respectively. A 20-minute waiting period followed drink completion to reach target BACs during the SAP procedure. Participants in the placebo condition were given 22.5 minutes (the average consumption time for the low, medium, and high does) to finish the drink. After completion of the drinking phase, an expired-breath sample was obtained using an Alco-Sensor IV (Intoximeters, Inc., St. Louis, MO) hand-held breathalyzer, and the SAP procedure was initiated.

The participant ("Subject A") was seated in front of a computer keyboard and monitor, following which the researcher provided the instructions for the reaction-time task via intercom from another room. The participant was led to believe (via audiotaped responses played over an intercom) that there was another "subject" of the same gender (a faux "Subject B") in the adjoining room who would be competing with the participant in the reaction-time task using identical equipment.

Before the reaction-time trials, a "pain threshold" was determined. A fingertip electrode was attached to the middle and index fingers of the participant's non-dominant hand, and a series

of shock intensities increasing at 100-microampere intervals was administered until the participant reported that the shock became "painful." This procedure was then repeated for "Subject B." This pain threshold procedure adjusts for individual differences in tolerance to shock stimulation and the acute antinociceptive dose effects of alcohol (Campbell, Taylor, & Tizabi, 2006) so as there are no systematic differences in pain expectations associated with the "20" shock.

Next, the researcher informed the participant (and ostensibly "Subject B") that the task consisted of a series of competitive reaction-time trials in which a "release" signal would be provided simultaneously to the participant and "Subject B" to determine "who was faster" on each trial. On trials the participant "lost" (i.e., was slower to release the space-bar compared to the "opponent"), he or she received a signal to select the intensity of shock to receive. After a brief delay, a message that the shock level selected was being delivered was presented on the computer screen, followed almost immediately by a 1-sec shock. On trials the participant "won," the "opponent" ostensibly self-administered a shock in the same way. The participant was not privy to the level of shock "the opponent" selected. The SAP consisted of 40 trials, including 20 trials in which the participant lost (and thus selected a shock to receive), with the frequency and pattern of wins (50%) and losses being pre-scripted.

Shock levels were selected by pressing 1 of 12 buttons on a computer keyboard, representing shock levels of "0" through "10" and "20." The participant was free to select any of these shock levels on a given "loss" trial. The "10" shock was equivalent to the shock level judged "painful." The "9" shock was set at 95% of this maximum, 8 at 90%, 7 at 85%, and so forth.

The participant was informed that selecting the 20 would administer a "painful" shock, twice the intensity of the pain threshold (10 shock), and that this level of shock, if selected, could produce "minor tissue damage." Thus, participants were led to believe that selecting the 20 shock would produce a very painful level of electrical stimulation. Although intentionality of self-harm cannot be directly observed, it is reasonable to infer that the participant is cognizant that the selection of the 20 shock could potentially result in (albeit modest) harm. In actuality, selecting the 20 delivered a shock that was the same intensity as the 10 shock.

The participant was also told that if a 0 was selected, no shock would be administered on losing trials. The 0 option was provided to increase the ecological validity of the task, and to allow the participant to opt out of receiving a shock after the threshold procedure. The participant was led to believe that the "opponent" received the same amount of alcohol as he or she did. We provided no other information about the role of shock in the task (that is, the participant was not told that the purpose of the competitive reaction-time task was to assess non-suicidal DSH), and both "subjects" were told before the task that neither would know the other's shock selections.

Subjective effects of alcohol were assessed post-task. The participant rated, from *1* through *10*, the number of "shots" of 100 proof vodka it would take to equal the amount of alcohol received. The participant also rated how intoxicated he or she felt immediately before the reaction-time task from *1* (*not at all*) through *8* (*very much*).

Finally, the participant rated the following post-task: (1) The degree of pain associated with the upper threshold (equal to the 10 shock); (2) how painful the 20 shock was if used; (3) the expected pain from the 20 if not used; and (3) the degree of tissue damage believed to be caused by the 20. All items were rated on a scale from *1* through *8* with higher ratings being

associated with higher levels of pain or tissue damage. BAC readings were obtained until the participant's BAC decreased to below .02%, at which point the participant was dismissed.

Results

Analyses were conducted two-tailed at the .05 level of significance. Follow up post-hoc mean comparisons for significant ANOVA *F*-tests used the Tukey's *HSD* procedure.

AUDIT scores. The mean AUDIT total score from the telephone screen for the sample was 4.15 (*SD* = 2.27). AUDIT scores did not differ as a as a function of alcohol group, $F(3, 202) = 0.22$, $p = .88$. Men produced higher AUDIT scores ($M = 4.82$; $SD = 2.13$) compared to women ($M = 3.47$; $SD = 2.21$), $F(1, 202) = 19.35$, $p < .001$, but no drink by gender interaction emerged for this analysis ($p = .81$).

BAC as a function of alcohol condition. The drink administration procedure appeared to produce the targeted BACs during the SAP task on average. A 4 (Alcohol Group) x 2 (Gender) x 2 (Before versus After the SAP) mixed-model ANOVA revealed a main effect for Alcohol Group. Placebo ($M = .000\%$, $SE = .000\%$), low ($M = .051\%$, $SE = .002\%$), medium ($M = .073\%$, $SE = .002\%$), and high ($M = .095\%$, $SE = .002\%$) alcohol group BACs all differed at $p < .05$. An Alcohol Group by Before versus After the SAP interaction emerged, $F(3, 202) = 5.78$, $p = .001$. BACs before and after the SAP did not significantly differ for the placebo or low-dose conditions. However, BACs after the SAP were slightly higher ($M = .076\%$, $SD = .016\%$) compared to before the SAP ($M = .070\%$, $SD = .020\%$) in the medium-dose condition, $F(3, 202) = 11.85$, $p = .001$. BACs after the SAP were also slightly higher ($M = .101\%$, $SD = .021\%$) compared to before the SAP ($M = .089\%$, $SD = .029\%$) in the high-dose condition, $F(3, 202) = 15.81$, $p < .001$.

Perceived intoxication. Alcohol group by gender ANOVAs were conducted to determine if (1) perceived amount of alcohol consumed ("number of shots consumed"), and (2) subjective intoxication ("… how intoxicated did you feel right before the reaction-time task?") differed as a function of alcohol group or gender. An alcohol effect was found for "shots," $F(3, 202) = 36.24$, $p < .001$. High-dose (4.42 shots) differed from medium-dose (3.62 shots), low-dose (2.93 shots), and placebo (1.36 shots). Medium- and low-dose did not differ from each other, but both differed from placebo. Note that 87.1% of the participants in the placebo condition reported receiving at least one "shot." No gender main effect or dose by gender interaction emerged. An alcohol effect was found for intoxication, $F(3, 202) = 33.22$, $p < .001$. High ($M = 5.49$, $SD = 1.95$) and medium-dose ($M = 4.90$, $SD = 1.90$) did not differ from each other, but both differed from low-dose ($M = 3.56$, $SD = 2.04$), and placebo ($M = 2.21$, $SD = 1.61$). Perceived intoxication differed by gender, $F(1, 202) = 5.66$, $p = .018$, with women ($M = 4.48$, $SD = 2.36$) reporting greater intoxication compared to men ($M = 3.77$, $SD = 2.12$).

Any use of the 20 shock. A three-way (Alcohol Group × Gender × 20 shock) frequency analysis was conducted using a hierarchical log-linear model to determine if the proportion of participants who selected a 20 at least once (i.e., used 20 shock versus did not use 20 shock) differed as a function of alcohol group and gender. Stepwise deletion starting with the highest order three-way association, followed by the three two-way and first-order effects, was used to examine all first order and higher associations (see Table 1 for cell counts and observed percentages).

Standardized residuals were examined, and no outliers were found. The chi-square model fit between expected and observed frequencies was good, $\chi^2(6) = 9.69$, $p = .14$. Three of sixteen cells (18.75%) had an expected frequency (EF) of less than 5 (women who did not select the 20

in the BAC .000, .050, and .075 cells). One of these cells did have an EF < 1 (BAC .000). Given that fewer than 20% of cells had an expected frequency less than 5, however, beginning this analysis with a three-way effect including gender (rather than collapsing across the gender) was deemed appropriate.

Parameter estimates (and partial chi-square associations; see Table 2) revealed a first-order association for use of the 20. Specifically, 31.0% of the participants overall used the 20 option at least once, but 69.0% did not, $Z = 4.80$, $p < .001$. This first-order effect was limited by second-order associations. First, more men (47.2%) used the 20 shock at least once compared to women (14.4%), $Z = 4.03$, $p < .001$. Second, alcohol dose and 20 use were associated, $Z = 2.58$, $p = .01$. As can be seen in Table 1, significantly fewer *total* participants in the BAC = .000% condition (10.0%) used the 20 compared to those in the BAC = .075% group (38.2%) and the BAC = .100% group (46.6%). Odds ratios for active dose conditions versus placebo overall were *OR* = 2.55, 3.82, and 4.66 for BAC = .050%, .075%, and .100%, respectively. Alcohol dose was associated with use of the 20 in both men and women, with significant placebo versus high-dose (BAC = .10%) differences emerging for both women (0% versus 25.8%) and men (19.2% versus 70.4%).

Number of 20 shocks used. BAC during the SAP was estimated by taking the average BAC before and after the SAP; thus, providing a dimensional biomarker of intoxication during the task. Most participants (83%) who selected the 20 at least once administered that shock level multiple times and use ranged from *0* through *20* ($M = 2.86$, $SD = 5.80$). Total number of 20s selected was associated with the DSHI ($r = .23$, $p = .001$), gender ($r_{pb} = .32$, $p < .001$; men coded 1, women 0), and BAC ($r = .19$, $p < .01$). No significant associations emerged among the DSHI, gender, or BAC.

Moderated regression using the PROCESS macro for SPSS (Hayes, 2013) was employed to examine the relation between BAC and number of 20s selected, and whether DSHI scores or gender moderated the effect of alcohol on SAP behavior. Interaction terms was created by multiplying the relevant first order variables after centering for ease of interpretation. The overall model was significant, $R^2 = .19$, $F(5, 204) = 8.57$, $p < .001$. The first order effects all uniquely contributed to the model: BAC: $b = 27.76$ ($SE = 9.01$); $t = 3.08$, $p = .002$ ($LLCI = 10.00$, $ULCI = 45.53$); DSHI: $b = 5.04$ ($SE = 2.33$); $t = 2.16$, $p = .03$ ($LLCI = 0.45$, $ULCI = 9.62$); and Gender: $b = 3.56$ ($SE = 0.73$); $t = 4.90$, $p < .001$ ($LLCI = 2.13$, $ULCI = 4.99$). The interaction between DSHI scores and BAC, however, was not significant, $b = 60.73$ ($SE = 53.18$); $t = 1.14$, $p = .25$ ($LLCI = -44.13$, $ULCI = 165.59$). No significant gender by BAC interaction was found, $b = 30.20$ ($SE = 17.87$); $t = 1.69$, $p = .09$ ($LLCI = -5.03$, $ULCI = 65.43$).

Pain threshold check. Four separate drink group by gender ANOVAS were conducted on: (1) ratings of the pain associated with the 10 shock (pain threshold); (2) the painfulness of the 20 for participants who used this shock; (3) the expected painfulness of the 20 for those who did not use this shock; and (4) the tissue damage anticipated to be caused by the 20 shock. Women rated the 20 as potentially causing more tissue damage ($M = 3.96$, $SD = 2.66$) compared to men ($M = 2.97$, $SD = 2.26$), $F(1, 202) = 8.34$, $p < .01$. No other significant effects emerged from these analyses (all ps $> .10$).

Discussion

A substantial literature supports the notion that alcohol intoxication is related to deliberate self-harm. However, the results of field and laboratory studies, including the present findings, suggest that alcohol is neither necessary nor sufficient for a self-harm episode to occur. However, the results of this study suggest that the effects of alcohol on self-harm are dose

dependent, and increase as a function of BAC. Although alcohol ingestion was not necessary to engage in self-harm (about 10% of the participants who did not receive alcohol selected the 20 shock), this risk increased as a function of dose assignment with about 47% of participants in the high-dose alcohol group (target BAC = .100%) using the 20 shock at least once. Results indicate that the effects of a low-dose of alcohol on DSH were not significantly different than placebo, but medium- and high-doses produced similar effects and both differed from placebo.
In addition, the number of 20s used fit a linear model, with individual differences in BAC positively associated with self-harm. Taken together, these results show that the tendency to initiate and perseverate in an analog task of non-suicidal DSH increased as a function of intoxication. Thus, these results provide complementary evidence to field studies suggesting that alcohol might be an important risk factor for self-harm, but only at levels of intoxication that would produce marked cognitive impairment (Guillot, Fanning, Bullock, McCloskey, & Berman, 2010).

Previous studies demonstrated that a high-dose of alcohol increased risk of DSH compared to no-alcohol drink conditions. However, these findings were limited by a single high dose and the inclusion of men only. The dose-response design of the present study provides evidence of a causal relation between alcohol and self-harm at multiple levels of alcohol intoxication. Specifically, “dialing up” alcohol exposure was accompanied by a commensurate increase in risk of self-harm, providing additional evidence for a causal relation. In addition, this effect was found for both men and women. That is, women also appear to be susceptible to the facilitative effect of alcohol on non-suicidal self-injury, despite engaging in lower levels of non-suicidal DSH overall compared to men. This finding is similar to findings for other-directed

aggression in which a positive linear effect has been reported for alcohol and aggression in both men and women (Duke et al., 2011).

Considering that not all self-harm events will involve alcohol, it was reasonable to assume that there are individual differences in how alcohol affects DSH behaviors. We examined whether alcohol presents a greater risk for individuals with a history of NSSI. Results indicated that the alcohol effect did not increase as a function of past NSSI. It is worth noting as a limitation that NSSI behaviors were relatively limited in this sample. We cannot rule out that NSSI might be a moderator of alcohol-associated DSH in clinical samples, or samples with a more pronounced NSSI history. In addition, the relation between alcohol intoxication and self-harm may be different in individuals with a history of alcohol misuse or dependence. Exclusion of individuals with alcohol use problems limits the generalizability of the current findings to this population, and offers an important direction for future research.

Both field studies and the present findings suggest that alcohol intoxication is neither necessary nor sufficient to elicit self-harm. It is therefore worthwhile to discuss the value of studies on alcohol and DSH. An examination of the role of alcohol in fatal motor vehicle crashes found that of the 20,871 drivers who died in a motor vehicle accident in 2013 only 36% had a detectable BAC level (National Highway Traffic Safety Administration, 2014), which is similar to the BAC rate in suicide decedents (Anestis et al., 2014). However, the fatal motor vehicle accident rate was six times as great for those who had a BAC > .08% compared to those who had BAC < .08%. This finding indicates that although alcohol is not present in the modal motor vehicle accident, it is, nevertheless, a factor in accident fatalities (as it is for suicides). Experimental studies using laboratory analogues (driving simulators) have provided strong complementary evidence for a causal link between alcohol consumption and impaired driving

that is dose dependent (Downey et al., 2013). Thus, field and laboratory studies suggest that while alcohol is neither necessary nor sufficient for a motor vehicle fatality to occur, it plays an important role. Similarly, the results of field, and now three separate laboratory studies, suggest that although alcohol intoxication is neither necessary nor sufficient for DSH to occur, alcohol might pay a role in the expression of DSH. Therefore, it is reasonable to suggest that the assessment and minimization of alcohol misuse should be considered in programs designed to limit DSH behavior.

The purpose of this study was to fill an important gap in the literature by examining the risk conferred by increasing levels of intoxication on DSH studied in the laboratory as a function past NSSI. The design used in the present study (confirmed by several outcomes), controlled for the antinociceptive of alcohol so that alcohol-related differences in pain experience would not account for the SAP behavior. The present study was not designed to test potential mechanisms for alcohol-related self-harm. However, acute alcohol intoxication is associated with dose-dependent attenuation in fear (Moberg, Weber, & Curtin, 2011), tendency to behave aggressively (Duke et al., 2011), and impairment across a range of cognitive tasks associated with judgment and executive functions (Guillot et al., 2010). Alcohol intoxication also dampens amygdala response to threating stimuli (Sripada, Angstadt, McNamara, King, & Phan, 2011) and impairs one's ability to reflect on self-relevant information associated with deliberate self-harm (Berman, Bradley, et al., 2009). It is reasonable, therefore, to posit that the acute effects of alcohol on fear and cognition increase the likelihood of engaging in self-harm. Future studies should therefore focus on the cognitive-affective processes underlying this relation.

DSH, irrespective of lethality, is difficult to study prospectively (Van Orden et al., 2010). Laboratory tasks such as the SAP provide the ability to test theories and identify causal risk

factors associated with self-harm with a high degree of internal validity that can be used to complement data from field and survey studies. Extrapolating these findings to extra-laboratory venues involving lethal forms of DSH should be done with caution, however, given that the SAP is best conceptualized as an analog of non-lethal self-harm. It is worthwhile to note, however, that engaging in non-lethal self-harm behavior outside the laboratory is not trivial and is one of the strongest predictors of later suicide attempts (Guan et al., 2012; Yen et al., 2003).

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Table 1

Count and Percentage of Participants Who Self-administered the 20 Shock

		Alcohol Group			
Gender		.000	.05	.075	.100
Women	No 20 Shock	24	19	23	23
		100%	82.6%	88.5%	74.2%
	Used 20 Shock	0_a	$4_{a,b}$	$3_{a,b}$	8_b
		0.0%	**17.4%**	**11.5%**	**25.8%**
Men	No 20 Shock	21	16	11	8
		80.8%	66.7%	37.9%	29.6%
	Used 20 Shock	5_a	$8_{a,b}$	$18_{b,c}$	19_c
		19.2%	**33.3%**	**62.1%**	**70.4%**
Total	No 20 Shock	45	35	34	31
		90.0%	74.5%	61.8%	53.4%
	Used 20 Shock	5_a	$12_{a,b}$	21_b	27_b
		10.0%	**25.5%**	**38.2%**	**46.6%**

Note. Each subscript letter denotes a subset of alcohol dose conditions that do not differ significantly from each other at the .05 level (Bonferonni corrected tests).

Table 2

Significance Tests for Hierarchical Model of Alcohol Group and Gender (N = 210)

Effect	*df*	Partial Association χ^2
First order effects:		
20	1	31.26*
Drink condition	3	1.39
Gender	1	0.02
Second order effects:		
Drink condition × 20	3	25.50*
Drink condition × gender	3	5.34
Gender × 20	1	32.26*

*$p < .001$