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The Effect of Laboratory Manipulations of Negative Affect on Alcohol Craving and Use: A Meta-analysis

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Abstract

Scientific and lay theories propose that negative affect plays a causal role in problematic alcohol use. Despite this common belief, supporting experimental evidence has been mixed. Thus, the goals of this study were to a) meta-analytically quantify the degree to which experimentally manipulated negative affect influenced alcohol use and craving in the laboratory, b) examine whether the size of this effect depended on key manipulation characteristics (i.e., self-relevance of the stressor, timing of the end of the stressor, and strength of negative affect induction) or sample characteristics (i.e., substance use history). Across 41 studies (N = 2,403), we found small-to-medium effects for more use (d_{av} = .31, 95% CI [.11, .50]) and craving (d_{av} = .39, 95% CI [.04, .74]) following a negative affect manipulation than a control manipulation. We also found a significant increase in craving from pre- to post- affect induction (d_{av} = .36, 95% CI [.14, .58]). This suggests the mixed results from the prior literature were likely due to statistically-underpowered studies. The moderator hypotheses received weak support, with few significant results in the predicted direction. Our meta-analysis provides clarity about a previously inconclusive set of results and highlights the need for more ecologically valid manipulations of affect in future work.

Keywords

Substance Use; Negative Affect; Alcohol; Meta-Analysis; AUD

Given the numerous short-term and long-term consequences of alcohol use and misuse, research identifying causal factors that can inform targets for intervention is warranted. Many sources of evidence suggest that negative affect plays a causal role in the development of problematic alcohol use (Baker et al., 2004; Hull, 1981; Volpicelli, 1987; Stasiewicz & Maisto, 1993). For example, meta-analytic results reveal a positive association between trait

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negative affect and alcohol use disorders (Kotov et al., 2010). Further research provides evidence that participants expect alcohol to reduce negative affect (e.g., Kassel, Jackson, & Unrod, 2000), and alcohol users who report drinking to cope with negative affect report greater alcohol use (Patrick, Schulenberg, O'Malley, Johnston, & Bachman, 2011). In addition, there is empirical evidence that negative affect plays an impedimentary role in relapse (e.g., Cooney et al., 1997), and empirically-supported treatments often focus interventions on disrupting the association between negative affect and alcohol use (e.g., Stasiewicz et a., 2013).

Despite this broad support, evidence from well-controlled laboratory studies that manipulate negative affect and then measure either alcohol use (e.g., amount of alcohol consumed in a sham taste test) or self-reported craving, is mixed. Some studies have found a significant effect of negative affect manipulations on alcohol and use and craving (e.g., Higgins & Marlatt, 1975; Tucker et al., 1980), whereas others have either found no significant effect or the opposite effect (e.g., Bacon, Cranford, & Blumenthal, 2015; Larsen et al., 2013). There are at least three possible ways to interpret mixed results. First, it could be there is no true effect, and significant results are false-positives. Second, it could be that these studies are statistically underpowered to detect the size of the true effect. Third, systematic methodological differences between studies increase or decrease the size of the effect, such that in some situations and/or for some people, negative affect may have a stronger effect on alcohol use and craving. A meta-analysis combining the effects across studies and testing key moderators informed by theory could help support or refute each of these interpretations. In particular, a meta-analysis of laboratory-based studies offers the opportunity for the strongest test of the effect of negative affect on alcohol use and craving, despite limitations of laboratory-based studies (e.g., limited ecological validity).

In terms of the potential moderators of effects, several contextual characteristics of stress or drinking situations have been proposed to influence whether negative affect will lead to alcohol use and craving. For instance, Hull's (1981) self-awareness model predicts alcohol use should occur for stressors that induce negative self-evaluation. This is based on the assumption that alcohol has tension-reducing effects by decreasing the ability to encode information needed for self-awareness. Thus, this theory would predict that mixed results might be a function of some studies using stressors that increase negative self-evaluation (e.g., giving a speech about personal flaws) and other studies using stressors that are irrelevant to the self (e.g., viewing unpleasant, disgusting pictures). Although this theory was developed to understand alcohol use, it can be extended to alcohol craving, meaning that people should crave alcohol when negative self-evaluation is high (versus low).[1]

Another aspect of the drinking context that may be important is the timing of the stressor in relation to the opportunity to consume alcohol. Volpicelli's (1987) review of animal and human research determined that alcohol use does not increase in anticipation of or during a stressor. The review indicated that it was only after a stressor that alcohol use increased. This

[1]In some of the research that followed the model's proposal, Hull and colleagues (Hull & Young, 1983; Hull, Young, & Jouriles, 1986) identified trait self-consciousness as an important moderator, with stronger effects of self-evaluation on drinking among people high in trait self-consciousness. We return to this point in the discussion.

theory would predict that the reason for mixed results in the prior literature is that some studies induced negative affect by having participants anticipate an upcoming stressor (e.g., a speech), whereas other studies measured alcohol use after the end of the stressor (e.g., a stressful imagery script).

Another important consideration may be the strength or potency of the manipulation; that is, its ability to elicit negative affect. Manipulations that elicit high levels of negative affect may be more likely to lead to alcohol use and craving than those that only increase negative affect slightly. Thus, the mixed results from the literature may partly be a function of the relative ability of existing manipulations to successfully increase negative affect. Somewhat in line with this, a meta-analysis of laboratory tobacco research found that studies that elicited more negative affect post-induction had larger effects on tobacco craving (Heckman et al., 2013). This question has not been examined in regard to alcohol use and may elucidate factors contributing to the inconsistency of findings in the alcohol literature.

In addition to contextual characteristics of the stressor, theories have identified individual differences that might explain for whom negative affect is more likely to lead to alcohol use. Theories have identified different motivations for substance use during different phases of the addiction process (Solomon, 1980; Koob, 2009). One set of theories predicts that heavier (e.g., dependent) users will have a stronger link between negative affect and substance use. This is proposed to be a function of the dissipation of the ‘high’ that occurs with repeated frequent use and, increase of unpleasant withdrawal symptoms when the substance is not in the system. Additionally, continued substance use enhances the connections between negative affective stress responses and drug withdrawal (Fox et al., 2005; Sinha et al., 2000), leading dependent users to be more likely to use during all periods of negative affect, and not just withdrawal-related negative affect. Another theory, based on the tobacco literature, suggests the opposite position (e.g., Shiffman et al., 2002). That is, negative affect should be less linked to use among heavy (versus social users). This is proposed to be a function of the fact that compared to other strong cues to use (e.g., morning coffee, end of work), negative affect is not as important of a cue for heavy users. Thus, mixed results highlight the need to examine important sample characteristics as a means to identify precursors to alcohol use and craving across different populations. It should be noted that a handful of other studies have looked at other individual differences such as family history of alcoholism and self-consciousness (Gord & Söderpalm, 2011; Hull & Young, 1983); however, there were too few studies to consider these factors in a meta-analysis.

Current Study

The overall goal of this study was to quantify the degree to which experimental manipulations of negative affect influence alcohol use and craving. We focused on studies involving laboratory inductions of negative affect, instead of naturalistic experiences of negative affect and substance use. This was done in order to conduct the strongest test of the theory, much like one conducts efficacy trials in treatment research before seeking to understand the effect in more real-world conditions. Given our interest in identifying factors that maintain substance use, we focused on use among current users, which included social/recreational users or people with current problems with alcohol use.

Our first goal was to estimate the effect size of manipulated negative affect on alcohol use and craving. Based on theory (e.g., Baker et al., 2004; Hull 1981) we predicted that the effect would be significantly different from zero. Based on similar meta-analyses looking at the effect of experimental affect manipulations on tobacco use and craving (Heckman et al., 2012, 2015), we predicted that negative affect manipulations would have small to medium size effects on alcohol use and craving. Our secondary goal was to clarify the mixed findings in the literature by testing key moderators derived from theories of negative affect and alcohol use. First, in line with Hull (1981), we predicted that the effect would be larger for negative affect manipulations considered self-relevant (e.g., speech about personal flaws) relative to self-irrelevant stressors (e.g., threat of shock). Second, consistent with Volpicelli (1987), we predicted that the effect of negative affect on drinking would be larger for studies that assessed use or craving after the stressor was over, compared to studies that measured use or craving during the anticipation of an upcoming stressor. Third, we looked at the relative strength of the induction, assuming that inductions that lead to a greater increase in negative affect would have larger effects. Finally, we examined whether the substance use history of the sample moderated effects. Based on contradictory theories (Baker et al., 2004; Shiffman et al., 2002), we did not have a specific prediction about substance use history as a moderator.

Method

Literature Search

Figure 1 shows the diagram of our literature search. In order to identify relevant publications, we searched PsychINFO and Pubmed using combinations of the following search terms: negative affect, negative emotion, and stress with substance use, alcohol, and craving. During the search, the title and abstract were reviewed, and articles were excluded if they did not report data or did not manipulate negative affect (i.e., correlational studies). We also examined the reference list of key review articles in the area. Combining these searchers yielded 325 articles. At this stage, the method sections for articles were examined for the main inclusion criteria: a laboratory study that manipulated negative affect and then measured alcohol craving and/or use. This stage of the process left 49 articles. Of these, 9 did not include sufficient data needed to calculate effect sizes. In one case we were able to obtain the data from the corresponding authors. In all other cases, we either received no response or the necessary data were no longer available. We ultimately retained 41 studies with a total samples size of 2,403. Due to varying levels of missing data for certain analyses, the exact number of studies varies for any given effect size.

Study Coding and Data Extraction

Data were extracted by two undergraduate students and the first two authors. All data were checked by a different coder and discrepancies were adjusted by consensus. To calculate effect sizes, we extracted the M and SD for craving and use. Our primary effect size compared alcohol use or craving in response to the negative affect induction relative to the neutral induction (hereafter referred to as *post-induction* use or craving). The majority of these studies were between-subject designs comparing participants exposed to different negative or neutral affect manipulations. There were some studies that exposed the same

participants to different affect manipulations in separate sessions (e.g., Cooney, et al., 1997; Miller, Hersen, Eisler, & Hilsman, 1974). To facilitate comparisons across within- and between-subject effects, we use d_{av} as our measure of effect size for within-subject effects (Cumming, 2012). This standardizes on the average of the two standard deviations (i.e., pre, post), making it more comparable to *d* from a between-subject design, relative to other methods that adjust for the correlation between repeated measures (e.g., d_{rm}). All effects were coded such that positive values indicated an greater use or craving in the negative affect condition. We interpreted effect sizes in line with Cohen's (1992) recommendations (small: *d* =.2, medium: *d* =.5, and large: *d* =.8).

For a subset of craving studies that had the relevant data (*k* = 14), we also compared changes in craving pre-induction to post-induction in the negative affect condition only (hereafter referred to as *pre-post induction* craving). Whereas the *post-induction* effect captures situation-dependent alcohol craving and use following negative affect, the *pre-post induction* captures within-person change in craving due to negative affect.

To characterize the studies, we recorded the year of publication, total sample size, percentage of the sample who were women, racial and ethnic breakdown of the sample, mean age of the sample, and type of sample (undergraduate/community/mixed). We also coded several moderator variables. To test Hull's theory, we rated the self-relevance of the negative affect induction of the study on a 1 (*not at all self-relevant*) to 4 (*very self-relevant*) scale. A priori, we determined exemplars of the different scale anchors: 1–standardized negative images (e.g., snakes); 2–Personalized imagery script about a stressful event; 3–a speech about a news article or non-self-topic; 4–a speech about personal flaws.[2] To code the timing of the stressor in relation to use, we coded whether the affect induction involved anticipation of an upcoming stressor (e.g., a speech) or was in response to the termination of the stressor (e.g., following negative image exposure). To test whether the strength of the negative affect induction was related to the effect size (cf. Heckman et al., 2012), we coded the *M* and *SD* of self-reported negative affect before and after the induction for participants in the negative affect condition. We used this information in two ways. Similar to Heckman et al.'s (2013) tobacco meta-analysis, we used the raw scores for post-induction ratings only (not change from pre to post). We expanded on Heckman's methods, however, by also calculating the size of the *change* in negative affect from pre-to post-induction. We tested addiction models that implicate negative affect in substance use at later stages of the addiction process (e.g., Koob, 2009) by coding whether the participants had an alcohol use disorder or not. For studies that had subsamples with and without alcohol use disorders, we calculated separate effect sizes for each subsample.

Data Analytic Plan

Given the broad array of methods used across studies, we expected heterogeneity in the effect sizes and therefore used a random effects model to calculate meta-analyzed effect size. Effect sizes were weighted by the inverse of the variance. For within-subject effects, we

[2]The Trier Social Stress Test was a commonly-used stressor across studies. It was coded as a "3" or "4" depending on the topic of the speech. When the topic was a non-personal topic (e.g., controversial issue) it was coded as 3, when it was a personal topic (e.g., personal qualifications) it was coded as a 4.

calculated the variance based on the formula provided by Dunlap, Cortina, Vaslow, & Burke's (1996), which factors in the correlation between repeated measures. Given that the correlations between repeated measures of craving and/or use were generally not reported, we use .8, which is considered a conservative estimate (Rosenthal, 1991). Heterogeneity in the effect sizes across studies was tested with the Q-statistic, which tests the null of no heterogeneity of effect sizes across studies. We also report I^2, which characterizes the total heterogeneity divided by the total variability. Higgins, Thompson, Deeks, and Altman (2003) recommend interpreting I^2 values of 25%, 50%, and 75% as small, medium, and large amounts of heterogeneity respectively. All models were run using the Metafor package in R (Viechtbauer, 2010).

For our main effect size calculation, we calculated the overall effects of negative affect on post-induction alcohol use and both post-induction and pre-post induction craving. We then tested our moderating hypotheses with meta-regression. Each moderation hypothesis was tested by adding the moderator (e.g., self-awareness of the study manipulation) as a predictor of effect size. For categorical moderators (e.g., timing of the stressor), we followed up significant effects by examining the effect size for the different categories. For continuous moderators (e.g., self-awareness), we used estimated model-based effect sizes for different points on the scale.

To address publication bias, we used selection methods (Vevea & Hedges 1995; Vevea & Woods, 2005), which a recent simulation showed outperformed other strategies for detecting and correcting for publication bias (McShane, Bockenhold, & Hasen, 2016). Selection methods contain two components: a data model and a selection model. The data model defines results assuming no publication bias exists, whereas the selection model defines the selection process (e.g., only significant results get published; significant results are more likely to be published). Together, these two models allow for a sensitivity analysis to see how the meta-analyzed effect might change under different selection structures.

We fit three different selection models using the weightfunct in R (Coburn & Vevea, 2016). The first estimated parameters for the selection function (Vevea & Hedges, 1995). We specified the selection function to only include significant results in the same direction. An advantage of this approach is that it allows for the estimation of standard errors and confidence intervals. This model provides a likelihood test, which tests whether the model with the selection parameter is a better fit than one without the selection parameter. Significant results are a sign of publication bias. A disadvantage of this approach is that it generally requires large samples ($k > 100$). The second and third models were based on Vevea and Woods (2005), who proposed a model where the selection parameters are fixed, obviating the need for large samples, but not allowing for the estimation of standard errors and confidence intervals. We used predefined weights that signify moderate and severe bias selection models (see Vevea & Woods for weights). Together the three different models provided a robustness test for our overall effects.

Results

Study Characteristics

The average sample size was 50.06 (see Table 1 for aggregated data and Table 2 for individual studies). Using a between-subject design (assuming an equal number of participants per group), individual studies on average had .10 power to detect small effects (d = .20), .41 power to detect medium effects (d = .50) and .79 power to detect large effects (d = .80). In a within-subject design, this would give .28, .93, and, .99 power to detect small, medium, and large effects. Thus, the studies were individually underpowered for small effects in both within- and between-subject designs and underpowered for medium effects for between-subject designs.

The demographics of the studies roughly followed the general population in the United States (United States Census Bureau, 2017). Participants were generally White and in their early-to-mid 30's (in 2012, the median age was 37). Studies tended to include more men than women and in relation to census data, had an underrepresentation of Hispanic/Latinx (17.6 versus 7.21). Perhaps more striking is the fact that only 51% of studies reported race/ethnicity and many studies only reported the percentage of Caucasians. The majority of the studies (63%) recruited from the community as a whole, with a minority (27%) using exclusively undergraduates. Three studies explicitly reported a mix of undergraduates and community members. See Table 1 for summary statistics and Table 2 for individual study data.

Overall Effects

In terms of alcohol use following a negative affect induction, there was a significant post-induction effect that was small-to-medium in size, (d_{av} = .31, 95% CI [.11, .50], k = 21), with a large amount of heterogeneity (I^2 = 71.54, p < .001). The effect was in the expected direction, indicating that the negative affect conditions produced more alcohol consumption than the control conditions. The post-induction effect was significant for craving and in the same direction and of similar magnitude (d_{av} = .39, 95% CI [.04, .74], k = 14). There was significant heterogeneity in the effect (I^2 > 80). Similar effects were found when looking at changes in craving from pre- to post-induction in the negative affect condition (d_{av} = .36, 95% CI [.14, .58], k = 14), again with significant heterogeneity (p's < .001) that was large in size (I^2 > 80). Taken together, these results are consistent with our predictions and suggest that the effects of negative affect inductions on alcohol use and craving fall between small to medium in size. The large amount of heterogeneity across studies warranted further investigation of moderators.

Moderators

Self-relevance—The self-relevance of the stressor was not significantly related to post-induction use (b = .01, 95% [−.18, .20], p = .929, k = 21) or post-induction craving (b = .26, 95% [−.17, .71], p = .241, k = 14). In support of hypotheses, however, self-relevance was related to larger pre-to-post induction increases in alcohol craving, (b = .175, 95% [.005, .345], p = .043, k = 14). To understand this effect, we estimated, based on the model, the effect size at low self-relevance (i.e., rating of 1 on 4-point scale) and high self-relevance of

the manipulation (i.e., rating of 4). For inductions with low self-relevance, the effect was not significant, (d_{av} = .06, 95% CI [−.28, .41]), whereas inductions high in self-relevance had a significant and large effect (d_{av} = .59, 95% CI [.29, .89]).

Timing—Although not statistically significant (b = .26, 95% [−.21, .74], p = .286, k = 21), studies that used anticipation as the negative affect induction had smaller post-induction use effect sizes (d_{av} = .11, 95% CI [−.31, .53], k = 5) compared to studies that allowed for alcohol use after the stressor was over (d_{av} = .37, 95% CI [.14, .60], k = 16). Moreover, the confidence interval for studies that measured alcohol use during the anticipation phase contained zero, whereas the other studies did not. These results provide weak evidence that alcohol use increases after a stressful event has ended but not during anticipation (Volpicelli, 1987).

Strength of negative affect manipulation—Next, we examined whether the strength of the negative affect manipulation influenced the size of effects. Contrary to predictions, neither self-reported negative affect post-induction nor increase in negative affect from pre-to-post induction was related to effect size for post-induction alcohol use or craving (see Table 3). This may be due, in part, to the fact that so few studies reported enough information about the negative affective experiences of participants in response to the induction to be included in these analyses. We did find one significant effect, such that post-induction negative affect, but not changes in negative affect pre to post induction, was associated with larger pre to post changes in craving (b = .02, 95% CI [.01, .05]).

As a supplement, we examined whether, on average, the manipulations used in these studies actually increased negative affect substantially. Thus, we calculated the meta-analyzed effect size for the increase in negative from pre to post in the negative affect condition. Across the 22 studies that had enough data, the effect of the induction on increases in negative affect across participants was significant and large in size (d_{av} = 1.13, 95% CI [.73, 1.53], k = 21), with a high amount of heterogeneity, I^2 = 96.75. These results show that, in general, the manipulations were successful in increasing negative affect.

Substance use history—Alcohol use disorder status was not significantly related to any of the effect sizes (see Table 3).

Publication Bias

Table 4 displays the results for the publication bias analysis. Following Vevea and Woods (2005), we interpreted the results from these analyses by looking at instability in the effect size across the different models. Large reductions when incorporating more severe selection bias models are indicative of publication bias. We made two comparisons to determine whether a reduction was large: a) the percentage change for the adjusted effect in relation to the original effect and b) the adjusted effect sizes to the 95% CI for the unadjusted effect.

The percentage decrease was generally small (M = 23%; min = 10%, max = 40%). In two cases, the adjusted model had a larger effect size than the unadjusted, which suggests the opposite of publication bias. These results are likely explained by the fact that the Vevea and Hedges (1995) model assumes studies are mostly reporting significant results in the same

direction, whereas we have identified and included nonsignificant results in our analyses. Aside from the two cases of effect size increase, all adjusted estimates were within the confidence intervals of the unadjusted effect. This suggests that there is little evidence of publication bias, which is not surprising given that there have been published results with null findings.

Discussion

The goal of this meta-analysis was to quantify the degree to which experimentally manipulated negative affect influences alcohol use and craving in the laboratory. We found small-to-medium effects, such that after a negative affect manipulation, participants were more likely to consume and crave alcohol than after a control manipulation. It is therefore likely that the mixed results from the prior literature were due to statistically underpowered studies. Our secondary goal was to examine whether the size of this effect depended on key manipulation characteristics (e.g., self-relevance and strength of negative affect induction) or sample characteristics (e.g., alcohol use disorder status). The moderator analyses generally failed to support our hypotheses, except for some consistent effects of self-relevance on craving effect sizes, despite significant heterogeneity of the effect sizes across studies.

Effect of Negative Affect Inductions on Substance Use and Craving

The overall finding that negative affect increases alcohol use and craving supports theories implicating negative affect as a maintenance factor of alcohol use (e.g., Baker et al., 2004; Koob, 2009; Stasiewicz & Maito, 1993). The effect sizes were similar when comparing post-induction between negative affect and neutral/control conditions (between-group) and pre-post change in craving in the negative affect condition (within-individuals), suggesting that this effect occurs both *between* situations and *within* people. The results were also similar across craving and alcohol use. This may be unsurprising, as many theories suggest that substance craving and substance use are strongly causally linked (e.g., Baker et al., 2004); however, other theories suggest that craving only occurs when substance use is blocked (Gass et al., 2014; Tiffany, 1990), which may imply different associations.

There are two ways to interpret the size of the effect. One the one hand, the effect size may be smaller than expected. Lay theories in particular, and psychological theories to a lesser extent (e.g., Baker et al., 2004), place a heavy emphasis on the association between negative affect and alcohol use, and this is reflected in the focus on negative affect and stress in various empirically supported treatments for substance use disorders. Thus, under the highly controlled laboratory conditions, it may be expected that the effect would be medium-to-large (e.g., $d = .60 - .80$). On the other hand, the size of the effect may be as expected or even larger than expected. The stressors used in the laboratory tend to be artificial and quite different (e.g., less complex, shorter duration) from the types of situations that elicit negative affect outside of the lab. Moreover, many aspects of laboratory alcohol studies reduce the likelihood that participants will consume alcohol (e.g., time of day, observer reactance), which would reduce to ability to find an effect. Thus, the fact that these laboratory studies detected effects of small to medium size can be interpreted as a support for theories that implicate negative affect in alcohol use.

Research using ecological momentary assessment, which measures affect and alcohol use across time in participants' natural environments, may help reconcile these interpretations.; however, somewhat similar to the laboratory studies, ecological momentary assessment studies have found mixed associations between prior negative affect and later alcohol use, with most studies finding null results (e.g., Dvorak et al., 2016; Treloar et al., 2015). These findings highlight that there are multiple factors that influence alcohol use; despite this, the meta-analysis detected a significant and arguably meaningful effect.

Moderator Analyses

We focused on several factors gleaned from prior theory to explain heterogeneity of the effect size across studies. First, we found that affect manipulations rated higher (versus lower) in self-relevance had a relatively stronger effect on within-person changes in craving. Although Hull's model (1981) was initially conceived for alcohol use and not craving, it makes logical sense that if, as the theory proposes, alcohol is reinforcing by reducing self-awareness, cravings to drink could be stronger in situations that increase their self-awareness. Our results show that this, in fact, occurs and provides a useful extension of Hull's model beyond alcohol consumption. It is unclear why the self-relevance of the stressor was not related to the effect size for alcohol use, although actual consumption may be a higher-threshold behavior that many participants avoid engaging in the lab (relative to endorsing craving). It is also possible that individual-level moderators influence the extent to which stressors that are more (versus less) self-relevant lead to drinking. In particular, prior research has found that self-relevant stressors lead to drinking among people high in trait self-consciousness (Hull & Young, 1983; Hull, Young, & Jouriles, 1986). Well-designed, well-powered studies in this area are needed to better understand the mechanisms by which self-relevance influences the association between negative affect and alcohol use.

Second, we found weak evidence for the moderating effect of anticipation vs. completion of the stressor prior to assessing drinking. That is, the effect size of negative affect on alcohol use was only significantly different from zero when stressors were completed prior to assessing alcohol use (but not when alcohol use was assessed during anticipation of a stressor). These results are somewhat in line with Volpicelli (1987), who reviewed animal studies showing increased alcohol consumption only after stressors had completed. The small number of studies using anticipation as a negative induction makes these results difficult to interpret. This fact highlights the need for further research.

Third, we sought to examine whether more powerful inductions of negative affect would have a stronger effect on craving and substance use. We found that mean post-induction negative affective responses were associated with a larger increase in within-subject craving; however, we did not find any other evidence in support of our hypothesis. This is somewhat surprising given that there was significant heterogeneity in the intensity of the affect manipulations and the effect size. The fact that few studies reported enough information to be included may have affected the ability to detect an effect. This suggests that future studies in the area should adequately report the effects of their manipulation on negative affective responses of the participants (e.g., *M*'s, *SD*'s tests statistics) to allow future researchers to more effectively evaluate the internal validity of the manipulations.

Finally, we did not find any evidence that the effect of negative affect was stronger or weaker for people with an alcohol use disorder vs. those who did not have a disorder. This likely suggests that the association between negative affect and substance use may not differ as a function of alcohol dependence. Rather, individuals who are dependent may be more likely to experience negative affect, which, in combination with other vulnerability factors (e.g., reduced self-control), may lead to more frequent use. This fits with prior research showing the individuals with alcohol use disorders are higher (versus lower) in trait negative affect (e.g., Kotov et al., 2010).

Recommendations for Future Studies

Based on these results, we make three recommendations for future studies in this area. First, given that the average size of the effect was small to medium, between-subject manipulations of negative affect should include a sample size of at least 260 to ensure power of 0.80, and within-subject designs should have at least 67 participants to adequately power their studies. Failing to do so increases the risk of continued mixed results in the literature and potential for both false-positive and false-negative results. Second, even when we included moderators in our analyses, there was a high amount of heterogeneity of effect size. This suggests that more standardization may be necessary in negative affect-alcohol use studies. Collaboration among multiple labs and using integrative data analysis (i.e., analysis of multiple data sets as one) may help identify sources of heterogeneity and therefore inform effective strategies for reducing their influence. Third, to properly estimate the effect of negative affect on drinking behavior in the real world, future studies, even in the laboratory, can seek to increase the ecological validity of the settings (e.g., bar lab) and the manipulations used (e.g., provocation by another person). Finally, most of the theories in this area have neglected to examine cultural and social identity factors, which may explain heterogeneity across studies. Previous research has identified different rates of substance use by gender (e.g., Wilsnack et al., 2009), racial and ethnic group (e.g., African Americans; Zapolski, Pederson, McCarthy, & Smith, 2013) and sexual minority status (e.g., McCabe et al., 2009), which may imply different contextual maintenance factors.

Limitations

Several limitations should be taken into account when considering our results. Although laboratory studies on the link between negative affect and substance use have been conducted for decades, we still had a fairly modest sample size of studies. More large-scale studies are needed to build further confidence in our results. Nonetheless, our meta-analysis provides initial clarity about a set of difficult-to-interpret mixed results and provides several avenues for future research, including providing guidelines for sample size. Second, our analyses, like those of prior studies, examine negative affect in general. Negative affect, however, is made up of several distinct feeling states (e.g., anger, fear, anxiety), which may have unique associations with alcohol use and craving. The majority of the negative affect inductions used in the reviewed studies likely targeted several emotions, making it difficult for us to capture that nuance in our moderator analyses. Future studies may wish to test the effects of certain negative affect states against others (e.g., anxiety vs anger).

A key limitation highlighted by the current meta-analysis is the lack of tests of ecological validity in the analyzed studies. Although our results show it is possible for negative affect to increase substance use and craving, they do not establish that this occurs outside of the laboratory. More ecologically-valid studies are needed to triangulate these findings. It is important to note, however, that existing ecological momentary assessment studies uncover results that are consistent with those of this meta-analysis (with small to medium effect sizes; Treloar et al., 2015). One compromise in balancing internal and external validity concerns would be to manipulate the presence of negative affect cues in the context of ecological momentary assessment studies, similar to drug cue reactivity studies in naturalistic environments (i.e., cue reactivity with ecological momentary assessment; Wray, Godleski, & Tiffany, 2011). Importantly, incorporating knowledge obtained from contemporary aversive learning theories and research (e.g., conditioned inhibition; serial conditioning; Laude & Fillmore, 2015) can lead to the development of studies and manipulations that take into account the complexities of human stressors and the effects of combined stimuli as they occur in the real world. Despite these limitations, this meta-analysis consolidates and provides some clarity to laboratory studies looking at the effect of negative affect on alcohol use and craving.

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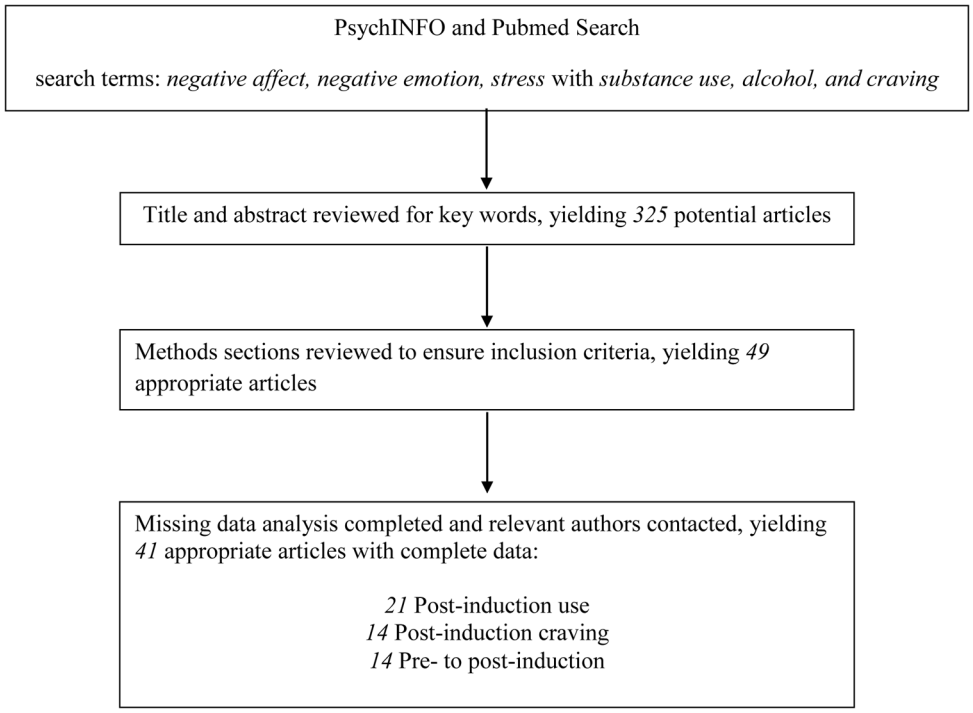

Figure 1.
Diagram of Study Search Strategy

Table 1

Aggregate Study Characteristics

	k	*M*	*SD*	*Min-Max*
Publication Year	41	2000	14.36	1973–2016
N	41	50.06	32.81	8–146
Mean age	31	32.11	9.59	19.3–49.5
% Women	40	36.56	28.46	0–100
Race/Ethnicity				
% Caucasian/White	22	74.04	17.95	22–95
% African American/Black	16	18.40	16.80	1–65
% Asian/Asian American	5	5.68	6.35	1–17
% Hispanic/Latinx	9	7.21	6.99	1–21.4
% Native American	2	3.2	2.54	1.4–5
% Mixed/Bi-racial	1	2.4	--	--
% Other	7	4.22	5.31	0–16

Note. k = number of studies reporting the information.

Table 2

Study Level Characteristics

Author (Year)	*N*	Sample Type	Affect Induction	Outcome	Alcohol use Disorder	Other Disorder(s)
1. Abrams et al (2002)	45	Community	Short Dialogues With other Participants	Use	None	Social Anxiety Disorder
2. Bacon & Thomas (2013)	40	Community	Trier Social Stress Test	Use	Alcohol Dependence	Social Anxiety Disorder
3. Bacon et al. (2015)	40	Undergraduate	Cyberball Social Exclusion	Use	None	None
4. Birch et al. (2004)	86	Undergraduate	10 minutes of Music	Craving	None	None
5. Brady et al. (2006) *	35	Community	Cold-pressor	Craving	Alcohol Dependence	None
5. Brady et al. (2006) *	28	Community	Cold-pressor	Craving	Alcohol Dependence	PTSD
6. Caselli et al. (2013) *	29	Community	Rumination	Craving	None	None
6. Caselli et al. (2013) *	26	Community	Rumination	Craving	None	None
6. Caselli et al. (2013) *	26	Community–Treatment Seeking	Rumination	Craving	Alcohol Dependence	None
7. Coffey et al., (2006)	12	Community–Treatment Seeking	Trauma Imagery Script	Craving	Alcohol Dependence	PTSD
8. Cooney et al. (1997)	50	In Patient	Personalized Imagery Script	Craving	Alcohol Dependence	None
9. Corcoran & Parker (1991)	69	Undergraduate	Preparation for A Speech about Body Flaws	Use	None	None
10. de Wit et al. (2003)	37	Community	Trier Social Stress Test	Use	None	None
11. Fox et al. (2013)	26	Community	Personalized Imagery Script	Craving	None	None
12. Higgins & Marlatt (1973) *	20	Community	Anticipation of Painful Shocks	Use	None	None
13. Higgins & Marlatt (1973) *	20	Community	Anticipation of Painful Shocks	Use	“Alcoholics”	None
14. Higgins & Marlatt (1975)	64	Undergraduate	Anticipation of Evaluation	Use	None	None
15. Higley et al. (2011)	28	Community	Stressful Imagery Script	Craving	Alcohol Dependence	None
16. Hull & Young (1983)	120	Community	Failure Feedback	Use	None	None
17. Jansma et al. (2000)	40	Inpatient Addiction Center	Average of Failure and Sad Music	Use	Alcohol Dependence	None
18. Kidorf & Lang (1999)	84	Undergraduate	Preparation for A Speech about Body Flaws	Use	None	None
19. Larsen et al. (2013)	106	Undergraduate	Trier Social Stress Test	Use	None	None
20. Marlatt et al (1975)	20	Undergraduate	Insulting Confederate	Use	None	None
21. Mason et al. (2008)	47	Community	Unpleasant IAPS Images	Craving	Alcohol Dependence	None
22. McGrath et al. (2016)	100	Mix	Preparing for Speech About Physical Appearance	Use	None	None
23. McNair (1996)	60	Undergraduate	Preparing for Speech About Physical Apparence	Use	None	None
24. Miller et al. (1974) *	8	Community	Role Play Requiring Assertiveness	Use	None	None

Author (Year)	*N*	Sample Type	Affect Induction	Outcome	Alcohol use Disorder	Other Disorder(s)
24. Miller et al. (1974) *	8	Community	Role Play Requiring Assertiveness	Use	Alcohol Abuse or Dependence	None
25. Noel & Lisman (1980) *	38	Undergraduate	Unsolvable Anagrams	Use	None	None
25. Noel & Lisman (1980) *	48	Undergraduate	Unsolvable Anagrams	Use	None	None
26. Nosen et al. (2012)	108	Community–Treatment Seeking	Trauma Imagery Script	Craving	Alcohol Dependence	PTSD
27. Phil et al. (1994)	27	Community	Electric Shock	Use	None	None
28. Phil & Yankofsky (1979)	40	Undergraduate	Intelligence Feedback	Use	None	None
29. Ralevski et al. (2016)	25	Community–Treatment Seeking	Trauma Imagery Script	Craving	Alcohol Dependence	PTSD
30. Ray (2011)	64	Community	Stressful Imagery Script	Craving	None	None
31. Rubonnis (1994)	57	Inpatient	Personalized Imagery Script	Craving	Alcohol Abuse or Dependence	None
32. Sinha et al (2009) *	28	Community–Treatment Seeking	Personalized Imagery Script	Craving	Alcohol Dependence	None
32. Sinha et al (2009) *	28	Community	Personalized Imagery Script	Craving	Alcohol Dependence	None
33. Steinberg et al. (2011) *	24	Community	Uncontrollable Noise	Craving	Alcohol Dependence	None
33. Steinberg et al. (2011) *	28	Community	Uncontrollable Noise	Craving	None	None
34. Thomas (2010)	129	Undergraduate	Worry	Craving	None	None
35. Thomas et al. (2011)	79	Community	Trier Social Stress Test	Use	Alcohol Dependence	None
36. Thomas et al. (2014)	82	Community	Trier Social Stress Test	Use	None	None
37. Tucker et al. (1980)	40	Undergraduate	Intellectual Performance Stress	Use	None	None
38. Vinci et a. (2014)	39	Undergraduate	Unpleasant IAPS Images and Music	Craving	None	None
39. Wardell et al. (2012)	146	Undergraduate	Unpleasant IAPS Images and Music	Use	None	None
40. Wolfe & Maisto (2000)	60	Undergraduate	Increased Salience of Self-discrepancy	Use	None	None
41. Zack et al. (2006)	69	Undergraduate	Read & Create Synonyms for Negative Words	Use	None	None

Note.

*Multiple Samples From the Same Paper With the Same Author and Year; PTSD = Posttraumatic Stress Disorder; Treatment Seeking = Currently in Treatment for Alcohol Use; IAPS = International Affective Picture System.

Table 3

Results of Moderator Analyses for Negative Affect Inductions on Alcohol Use and Craving

	k	*b*	95% CI	R^2
Self-relevance				
Post-induction Use	21	.01	[−.18, .20]	0
Post-induction Craving	14	.26	[−.17, .71]	3.08
Pre-Post Induction Craving	14	.18	[.01, .34]	24.26
Timing				
Post-induction Use	21	.26	[−.21, .74]	0
Post-induction Negative Affect				
Post-induction Use	7	−.001	[−.02, .02]	0
Post-induction Craving	8	.02	[−.02, .06]	0
Pre-Post Induction Craving	8	.03	[.01, .05]	65.24
Negative Affect Change				
Post-induction Use	8	−.05	[−.28, .17]	0
Post-induction Craving	8	−.08	[−.45, .30]	0
Pre-Post Induction Craving	9	.07	[−.15, .29]	0
Alcohol Use Disorder				
Post-induction Use	21	−.22	[−.78, .34]	0
Post-induction Craving	14	.51	[−.21, 1.23]	2.74
Pre-Post Induction Craving	14	.27	[−.17, .71]	2.53

Note. k = the number of samples included in the analysis; Self-relevance = self-relevance of the stressor/negative affect induction of the study; R^2 = amount of heterogeneity accounted for.

Table 4

The Effect Size and Standard Error From Publication Bias Analyses

	1	2	3	4
Post-induction Use	.30 (.114)	.56	.26	.21
Post-induction Craving	.39 (.179)	.26	.24	.18
Pre-Post Induction Craving	.36 (.112)	.57	.33	.26

Note. 1 = unadjusted; 2 = Vevea & Hedges (1995); 3 = Vevea & Woods (2004) Moderate Selection Bias; 4 = Vevea & Woods Severe Selection Bias.