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Negative affect is associated with alcohol, but not cigarette use in heavy drinking smokers

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HIGHLIGHTS

- We modeled negative affect, smoking and drinking in heavy drinking smokers.
- Negative affect was related to alcohol use, but not cigarette use.
- Craving was examined as a statistical mediator of this association.
- Craving fully mediated the negative affect–alcohol use association.

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ABSTRACT

Co-use of alcohol and cigarettes is highly prevalent, and heavy drinking smokers represent a large and difficult-to-treat subgroup of smokers. Negative affect, including anxiety and depressive symptomatology, has been associated with both cigarette and alcohol use independently, but less is known about the role of negative affect in heavy drinking smokers. Furthermore, while some studies have shown negative affect to precede substance use, a precise biobehavioral mechanism has not been established. The aims of the present study were twofold. First, to test whether negative affect is associated with alcohol and cigarette use in a large community sample of heavy drinking smokers ($n = 461$). And second, to examine craving as a plausible statistical mediator of the association between negative affect and alcohol and/or cigarette use. Hypothesis testing was conducted using a structural equation modeling approach with cross-sectional data. Analysis revealed a significant main effect of negative affect on alcohol use ($\beta = 0.210, p < 0.05$), but not cigarette use ($\beta = 0.131, p > 0.10$) in this sample. Mediational analysis revealed that alcohol craving was a full statistical mediator of this association ($p < 0.05$), such that there was no direct association between negative affect and alcohol use after accounting for alcohol craving. These results are consistent with a negative reinforcement and relief craving models of alcohol use insofar as the experience of negative affect was associated with increased alcohol use, and the relationship was statistically mediated by alcohol craving, presumably to alleviate negative affect. Further longitudinal or experimental studies are warranted to enhance the causal inferences of this mediated effect.

1. Introduction

Epidemiological studies have suggested a strong correlation between smoking and alcohol use (Anthony & Echeagaray-Wagner, 2000). Specifically, it has been estimated that 20–25% of smokers are also heavy alcohol users (Dawson, 2000; Toll et al., 2012) and this co-occurrence has been associated with poorer outcomes in terms of physical health and smoking cessation quit rates (Hymowitz et al., 1997; Kahler, Spillane, & Metrik, 2010; Toll et al., 2012). In the context of a quit attempt, alcohol use has been associated with increased risk for a smoking lapse, such that heavy drinking smokers are 4 times more likely to experience a smoking lapse in the context of a drinking episode and 8 times more likely to lapse in the context of a heavy drinking episode (Kahler et al., 2010).

Several theories have been proposed to explain the strong co-occurrence of alcohol and cigarette use. For example, participants with a past history of alcohol dependence have reported greater reinforcement from nicotine administration as compared to those who were never alcohol dependent (Hughes, Rose, & Callas, 2000). Alternatively, smokers report less intoxication to standard alcohol doses than non-smokers in spite of similar metabolic elimination rates, a phenotype thought to confer risk for alcohol dependence (Madden, Heath, Starmer, Whitfield, & Martin, 1995). Genetic studies have also identified common underlying genetic liability for both alcohol and nicotine dependence (Kendler, Myers, & Prescott, 2007). Lastly, through

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blockade of the upregulation of $GABA_A$ receptors during alcohol abstinence, nicotine may reduce the severity of alcohol withdrawal (Staley et al., 2005), thus leading to substantial negative reinforcement in heavy drinking populations.

Psychological factors such as negative affectivity may also influence the co-occurrence of alcohol and nicotine misuse. The experience of negative affect, which according to the tripartite model of internalizing disorders represents the common psychological construct linking anxiety and depressive symptomatology (Clark & Watson, 1991), has been associated with both alcohol misuse and smoking behavior independently. It has been estimated that 47% of individuals meeting criteria for a lifetime Alcohol Use Disorder (AUD) also met criteria for at least one other psychiatric disorder (Helzer & Pryzbeck, 1988), and internalizing disorders including anxiety and depression have been consistently associated with alcohol misuse (Greeley & Oei, 1999; Jackson & Sher, 2003; Kessler et al., 1994; Prescott, Aggen, & Kendler, 2000). Some studies have suggested that anxiety disorders (Merikangas, Risch, & Weissman, 1994) and depressive symptoms (Hussong & Chassin, 1994) precede comorbid AUDs suggesting a causal mechanism through which the negative affect leads to escalations in alcohol use.

Negative affect has also been associated with cigarette smoking (Breslau, Novak, & Kessler, 2004a, 2004b; Lerman et al., 1996); however, the direction of this effect remains unclear. In a large twin-study of females, Kendler, Kessler, Neale, Heath, and Eaves (1993) found evidence that the co-occurrence of major depressive disorder and cigarette smoking was not causal, but instead was the result of shared familial risk factors. Relatedly, numerous studies have suggested a bidirectional association between depressive symptomatology and cigarette use (for review see Chaiton, Cohen, O'Loughlin, & Rehm, 2009).

While extant evidence suggests an association between negative affect and alcohol and nicotine use, a precise mechanism underlying this relationship has not yet been identified. One plausible candidate is the experience of craving (defined as a strong desire to use a substance), which has long been recognized as an important construct in the development and maintenance of addictive disorders, (for review see Addolorato, Leggio, Abenavoli, and Gasbarrini (2005)). The import of craving in addiction development and maintenance prompted it's inclusion as a symptom of substance use disorders in DSM-5 (American Psychiatric Association, 2013). Drawing from a cognitive social learning perspective (Marlatt & Gordon, 1985), Wright, Beck, Newman, and Liese (1993) identified several distinct subtypes of craving which included craving in response to lack of pleasure. This conception of craving was later bolstered by Verheul, van den Brink, and Geerlings's (1999) three-pathway model of craving, wherein *relief craving* represents a central feature of alcohol craving. Further, relief craving is consistent with a negative reinforcement model of addiction, whereby drug seeking and drug taking is negatively reinforced via alleviation of negative affect whether it be withdrawal induced or pre-morbid (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004). Thus several influential models of addiction etiology and maintenance have converged to suggest that negative affect may perpetuate relief craving for drugs of abuse in a goal directed manner aimed at alleviating negative affect. Accordingly, craving may be advanced as a plausible mediator of the association between negative affect, vis-à-vis anxiety and depressive symptomatology, and cigarette and/or alcohol misuse. Recently in a large randomized controlled study Glöckner-Rist, Lémenager, and Mann (2013) demonstrated anxiety to be selectively associated with relief craving for alcohol, thus bolstering the empirical rational for examining craving as a plausible mediator of the association between negative affect and alcohol (and potentially cigarette) use.

In order to advance the literature reviewed above and provide clarity on the relationship between negative affect, craving, and cigarette and alcohol use in heavy drinking smokers, the aims of this study are twofold. First, we tested whether negative affect is associated with cigarette use and alcohol use in a large community sample of non-treatment seeking heavy drinking smokers. Based on the literature demonstrating a significant positive association between anxiety and depressive symptomatology and alcohol or cigarette use independently (e.g. Breslau et al., 2004b; Jackson & Sher, 2003; Prescott et al., 2000), we hypothesize a positive association between negative affect and both cigarette and alcohol use. Where a significant association is identified (i.e. between negative affect and smoking or negative affect and drinking) we will conduct exploratory analyses examining craving as a statistical mediator of the association between negative affect and cigarette and/or alcohol use. A tiered analytic technique is employed as a significant association or 'total effect' must be demonstrated prior to examination of possible mediators. As the data in these analyses are purely cross-sectional, we aim to assess craving as a plausible mediator of the relationship between negative affect and cigarette and/or alcohol use through examining craving as a mediator in the statistical sense. Thus, the overall objective of this study is to elucidate the role of negative affect on alcohol and cigarette use among heavy drinking smokers and examine craving as a plausible mediator of these effects.

2. Methods

2.1. Participants

Participants were recruited for a large double blinded placebo controlled laboratory study of Varenicline, Naltrexone, and their combination for heavy drinking smokers (Ray et al., 2014). This laboratory study was not a treatment study for nicotine or alcohol abuse or dependence and all subjects were non-treatment seekers. A community-based sample of heavy drinking, daily smokers was recruited ($N = 461$) via online and print advertisements in the Los Angeles area. Inclusion criteria were: (1) age between 21 and 55; (2) smoking ≥ 10 cigarettes per day; (3) current status of heavy drinking according to the National Institute on Alcohol Abuse and Alcoholism guidelines (NIAAA, 1995): ≥ 14 drinks (7 for women) per week or >4 drinks (3 for women) per occasion at least once per month over the past 12 months. Exclusion criteria were: (1) more than 3 months of smoking abstinence in the past year; (2) self-reported use of illicit drugs (other than marijuana) in the previous 60 days; (3) self-reported lifetime history of psychotic disorders, bipolar disorders, or major depression with suicidal ideation; (4) serious medical condition in the past 6 months (e.g., significant cardiovascular disease; uncontrolled hypertension; hepatic or renal disease). Participants were required to have a Breath Alcohol Concentration (BrAC) of 0.00 g/dl at the assessment visit and a toxicology screen was administered to ensure no drug use.

2.2. Procedures and measures

Interested individuals first completed a telephone interview to assess for major inclusion/exclusion criteria. Eligible callers were then invited to an in-depth face-to-face assessment. Data for this study was collected at this in-person assessment. After the assessment visit, eligible participants completed a physical exam and, if medically eligible, were randomized to one of 4 medication conditions in a 2×2 placebo controlled design. All data for the present study was collected prior to medical screening and medication randomization. After receiving a full explanation of the study procedures and providing written, informed consent, participants completed a series of self-report questionnaires including a demographics questionnaire. The following measures were used for hypothesis testing:

Beck Depression Inventory-II. The Beck Depression Inventory (BDI-II) is the most commonly used assessment of current (past 2 weeks) depressive symptomatology (Beck, Steer, & Brown, 1996b; Sharp & Lipsky, 2002). The self-report measure consists of 21 items each scored on a 0 to 3 Likert scale. Total score on the measure is a simple

sum of item scores. High reliability has been demonstrated for the BDI-II (Beck, Steer, Ball, & Ranieri, 1996a).

Beck Anxiety Inventory. The Beck Anxiety Inventory (BAI) is a widely used measure of state anxiety and captures anxiety symptoms present in the past 2 weeks (Beck & Steer, 1990). The BAI has demonstrated high internal reliability and significant convergent and divergent validity (Fydrich, Dowdall, & Chambless, 1992). The BAI consists of 21 items each scored on a Likert scale from 0 to 3.

Timeline Follow-Back (TLFB) . The 30-day timeline follow-back (TLFB) interview (Sobell, Sobell, Leo, & Cancilla, 1988) was used to record the quantity and frequency of alcohol and cigarette use over the past 30 days. An alcohol binge was defined as consuming ≥5 drinks (≥4 drinks for women) in a given drinking episode in concordance with NIAAA guidelines (NIAAA, 1995). The following indices were derived from the 30-day TLFB: (1) drinks per drinking day (DPDD), (2) percent binge drinking days (Binge), and (3) cigarettes per day (CPD).

Fagerstrom Test for Nicotine Dependence (FTND) . The Fagerstrom Test for Nicotine Dependence (FTND) is a well-validated and widely used 6-item measure of nicotine dependence severity (FTND: Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991).

Penn Alcohol Craving Scale. The Penn Alcohol Craving Scale (PACS) is a 5-item self-report measure of alcohol craving over the past week (Flannery, Volpicelli, & Pettinati, 1999). Each question is scored on a Likert scale from 1 to 7. Flannery et al. (1999) established several indicators of measure validity for the PACS including: internal reliability, predictive validity, construct validity, and discriminant validity.

Obsessive Compulsive Drinking Scale. The Obsessive Compulsive Drinking Scale (OCDS) measures craving as a multidimensional construct including obsessive thoughts about drinking, compulsions to drink alcohol, and interference in daily functioning (Anton, 2000). The OCDS consists of 14 questions each measured on a Likert scale from 1 to 5. Internal reliability, congruent validity, and predictive validity have been demonstrated for the OCDS (Anton, 2000). For the present study a single total composite score was calculated to capture total craving as a single construct.

Wisconsin Inventory of Smoking Dependence Motives (WISDM) . The Wisconsin Inventory of Smoking Dependence Motives (WISDM-68) is a large and well-validated measure of dependence motives across multiple domains (Piper et al., 2004). The WISDM contains 68 items scored on a 7-point Likert scale. The 13 subdomains of the WISDM include automaticity, taste/sensory processes, positive reinforcement and craving, among others. For this study, score on the craving dimension of the WISDM (consisting of 4 items) will be used as an indicator of cigarette craving.

2.3. Data analytic strategy

Hypothesis testing was conducted using a structural equation modeling (SEM) framework in EQS Version 6.2 for Windows (Bentler, 1995). Due to significant positive multivariate kurtosis as indexed by normalized Mardia's coefficient (>20 in all models), the modestly skewed distribution of alcohol use in this sample of heavy drinking smokers, and the existence of numerous univariate outliers (n = 58 as defined by Z-score >4), models were examined using robust standard errors, which are relatively insensitive to all of these properties of our data. Further with regards to outliers, all models were fully replicated with identical substantive effects when all outliers were removed. Statistical model fit was assessed with the Satorra-Bentler scaled χ^2 fit index (Satorra & Bentler, 2001). However, use of the χ^2 likelihood ratio test to assess model fit has been deemed unsatisfactory for numerous reasons (Tanaka, 1993), thus a relative estimate (ratio of χ^2 to degrees of freedom) was also calculated with values <2 indicating reasonable fit (Byrne, 1989). Model fit was also assessed with the robust versions of the comparative fit index (CFI: Bentler, 1990), and the root mean square error of approximation (RMSEA: Browne, Cudeck, Bollen, & Long, 1993) including a 90% confidence interval. Both the CFI and the RMSEA are sensitive to model misspecification and are minimally affected by sample size (Bentler, 1995). The CFI ranges from 0 to 1, with values above 0.90 indicating acceptable fit (Bentler, 1990). RMSEA values less than 0.05 indicate close fit and values less than 0.07 indicate reasonable fit (Steiger, 1990, 2007). Only covariates that significantly predicted outcome variables or significantly altered model fit or structure were retained in the final models presented herein.

In line with the study aims, two structural equation models were constructed in a tiered fashion. The first SEM model examined the effect of negative affect, as indexed by the BDI and the BAI, on cigarette use, indexed by CPD and FTND, and alcohol use, indexed by DPDD and Binge. Sex and age were entered initially as covariates in the model predicting both cigarette and alcohol use. Additionally, an a priori hypothesized covariance between alcohol use residual and cigarette use residual was estimated in this model to account for covariance between alcohol and cigarette use not explained by negative affect. The second SEM model was constructed to test whether craving (as indexed by the PACS and OCDS for alcohol and the WISDM craving subscale for cigarettes) was a significant statistical mediator of the effects identified in the first model. As before, sex, and age were entered as covariates. No covariances were estimated in this second model.

3. Results

3.1. Sample characteristics

Mean, standard deviation, range, and Pearson bivariate correlations for all variables in the structural equation models are presented in Table 1.

3.2. Measurement reliability

With the exception of the FTND, all scales demonstrated high internal reliability. Specifically, unstandardized Cronbach's alphas were computed for each self-report scale and were found to be highly reliable (BAI: $\alpha = 0.92$, BDI: $\alpha = 0.89$, PACS: $\alpha = 0.90$, OCDS: $\alpha = 0.92$, WISDM Craving: $\alpha = 0.84$). In this sample, Cronbach's alpha for the FTND ($\alpha = 0.48$) did not reach the threshold of 0.70 (Cortina, 1993). Multiple other studies have shown the FTND to have poor internal consistency (e.g. Haddock, Lando, Klesges, Talcott, & Renaud, 1999; Heatherton et al., 1991; Payne, Smith, McCracken, McSherry, & Antony, 1994), which may be a result of the small number of items in combination with demonstrated multidimensionality (Haddock et al., 1999; Radzius et al., 2003). However, in spite of sub-par internal reliability, the FTND has demonstrated good test–retest reliability and convergent validity (Pomerleau, Carton, Lutzke, Flessland, & Pomerleau, 1994) and thus FTND score was maintained as a valid and reliable indicator of cigarette use. To examine whether measurement unreliability was biasing results, the models presented below were replicated without inclusion of FTND score (i.e. using CPD as the sole indicator of cigarette use) and the results were identical.

3.3. Effect of negative affect on smoking and drinking

The model assessing the main effect of negative affect on cigarette and alcohol use was found to fit the data well (S-B $\chi 2$ [16] = 25.60, $p = 0.060$; relative $\chi 2 = 1.6$; CFI = 0.980; RMSEA = 0.038, 90% CI: [0.000, 0.065]; Fig. 1). In this model all observed variables loaded significantly on their respective latent variable (βs > 0.65). Additionally,

Table 1
Mean, standard deviation, range and first-order Pearson correlations for all variables included in the models presented.

		Mean	Std. dev.	Min	Max	1	2	3	4	5	6	7	8	9
1	Age	36.10	10.60	21	55	1.000								
2	Male	0.71	0.45	0	1	0.007	1.000							
3	BAI	7.53	8.29	0	52	−0.049	0.016	1.000						
4	BDI	11.39	10.07	0	51	0.000	−0.036	0.496**	1.000					
5	CPD	13.72	7.75	0.03	61.3	0.131**	0.066	0.072	0.044	1.000				
6	FTND	3.87	1.87	0	8	0.198**	0.030	0.094	0.078	0.523**	1.000			
7	DPDD	7.05	4.64	1	37.9	−0.074	0.140**	0.117*	0.151**	0.248**	0.099*	1.000		
8	Binge	0.68	0.33	0	1	−0.118*	0.296**	0.131**	0.129**	0.201**	0.081	0.595**	1.000	
9	PACS	17.44	6.81	5	35	−0.083	0.009	0.433**	0.307**	0.160**	0.172**	0.225**	0.251**	1.000
10	OCDS	31.59	10.13	8	69	0.026	0.046	0.554**	0.359**	0.215**	0.179**	0.245**	0.300**	0.797**

significant covariance was observed between cigarette use and alcohol use residuals ($p < 0.05$). In this model, negative affect was significantly associated with alcohol use ($\beta = 0.210$, $p < 0.05$) after controlling for age ($\beta = -0.118$, $p < 0.05$) and sex ($\beta = 0.304$, $p < 0.05$). A significant main effect of negative affect on cigarette use was not observed ($\beta = 0.131$, $p > 0.10$), after controlling for age ($\beta = 0.212$, $p < 0.05$).

In light of the significant association between negative affect and alcohol use only, a second model was constructed assessing whether craving for alcohol mediated the relationship between negative affect and alcohol use. As negative affect was not significantly associated with cigarette use, cigarette use was entered in as a covariate predictor of alcohol use and craving in this model. No dependent variable covariances were estimated in this model. The mediational model was found to fit the data well (S-B $\chi 2$ [31] = 59.72, $p = 0.002$; relative $\chi 2 = 1.92$; CFI = 0.970; RMSEA = 0.048, 90% CI: [0.029, 0.065]; Fig. 2) and all factor loadings were significant (β's > 0.53). In this model, negative affect was significantly related to craving ($\beta = 0.624$, $p < 0.05$) after controlling for cigarette use ($\beta = 0.181$, $p < 0.05$). Additionally, alcohol craving was significantly associated with alcohol use ($\beta = .323$, $p < 0.05$) after controlling for age, sex and cigarette use (β's = −0.162, 0.287, and 0.195 respectively, p's < 0.05). No direct effect of negative affect on alcohol use was observed ($\beta = -0.049$, $p > 0.10$). Indirect path analyses revealed a significant mediated effect of negative affect on alcohol use, as mediated by alcohol craving (indirect effect B = 0.616, SE = .175, $p < 0.05$). These results indicated a full statistical mediation effect, insofar as the association between negative affect and alcohol use was no longer statistically significant after accounting for the effect of alcohol craving.

4. Discussion

Heavy drinking smokers represent a large and difficult to treat subgroup representing approximately 20–25% of all smokers (Dawson, 2000; Toll et al., 2012) and co-use of cigarette and alcohol is associated with poorer clinical outcomes (Hymowitz et al., 1997; Kahler et al., 2010; Toll et al., 2012). Several factors ranging from pharmacological interaction (i.e., cross-reinforcement) to common genetic risk factors have been proposed to explain the high prevalence of alcohol and cigarette use co-occurrence. Negative affect including anxiety and depressive symptomatology has been associated with both alcohol use and cigarette use independently (Greeley & Oei, 1999; Kessler et al., 1994; Prescott et al., 2000) and thus may represent a shared psychological risk factor for alcohol and cigarette misuse. In light of the existing literature on negative reinforcement models addiction etiology (e.g. Baker et al., 2004), the present study tested whether greater negative affect was associated with alcohol and cigarette use in a large community sample of heavy drinking smokers. Analyses revealed that negative affect was positively associated with the magnitude of alcohol use in this population of heavy drinking smokers, who represent a unique subpopulation of drinkers associated with more detrimental health outcomes (N. S. Miller & Gold, 1998).

While a similar association between negative affect and cigarette use was hypothesized, negative affect was not significantly associated with cigarette use in this sample of heavy drinking daily smokers after controlling for age (removal of age as a covariate did not alter the significance of this effect [data not shown]). It appears that, while negative affect is predictive of smoking behavior in the general population, or

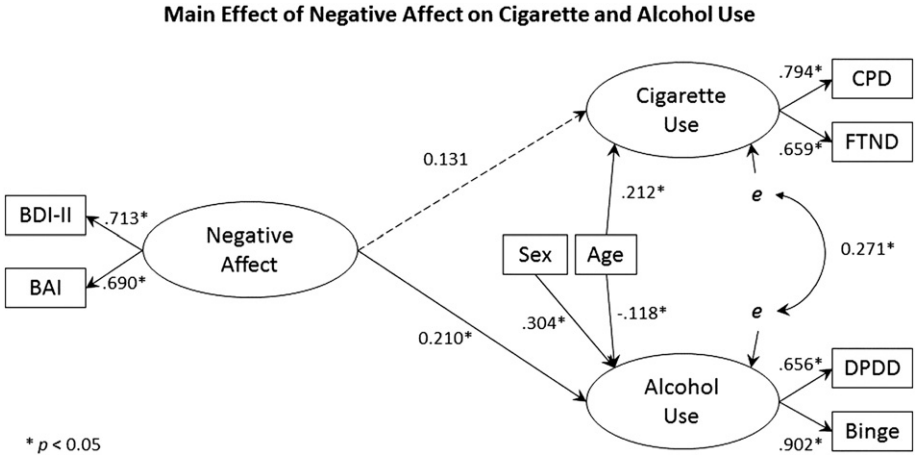

Fig. 1. Structural equation model testing the effect of negative affect [indexed by Beck Depression Inventory (BDI-II) and Beck Anxiety Inventory (BAI)] on alcohol use [indexed by Drinks per Drinking Day (DPDD) and percent Binge drinking days (Binge)] and cigarette use [indexed by average Cigarettes per Day (CPD) and the Fagerstrom Test for Nicotine Dependence (FTND)], controlling for sex and age. Standardized path coefficients are shown. Non-significant estimated paths are shown as a dotted line. Residual covariances are represented with a double sided arrow.

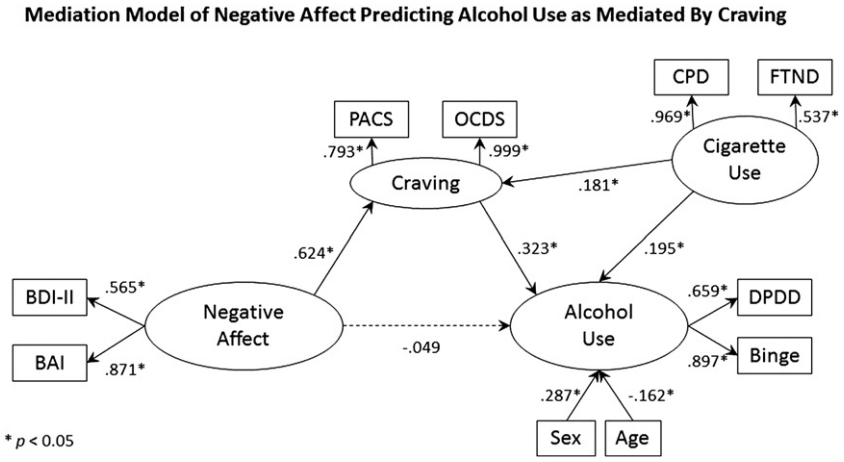

Fig. 2. Structural equation model examining the effect of negative affect on alcohol use as statistically mediated by alcohol craving [indexed by the Penn Alcohol Craving Scale (PACS) and the Obsessive Compulsive Drinking Scale (OCDS)], after controlling for sex, age and cigarette use. Standardized path coefficients are shown. Non-significant estimated paths are shown as a dotted line. Indirect path analysis revealed that alcohol craving was a full statistical mediator of the association between negative affect and alcohol use (indirect effect B = 0.616, SE = .175, $p < 0.05$).

smoking status (Breslau et al., 2004a, 2004b), among regular smokers who are also heavy drinkers, negative affect may not predict magnitude of cigarette use or nicotine dependence. In their review of the tripartite model of anxiety and depression and cigarette use, Ameringer and Leventhal (2010) found evidence that all three dimensions of the tripartite model (i.e. negative affect, anhedonia and low positive affect, and anxious arousal) predicted smoking status, but found little to no support for the claim that tripartite dimensions predicted smoking heaviness or severity of nicotine dependence, particularly among non-treatment seekers. The results of the present study are in line with this review in that the negative affect was not associated with smoking heaviness (CPD) and severity of nicotine dependence (FTND) among non-treatment seeking regular smokers.

These results suggest that treatments targeting anxiety and depressive symptomatology in heavy drinking smokers might reduce the magnitude of their alcohol use, but may not reduce their smoking directly. As alcohol use is known to increase the risk of smoking lapses, however, reductions in alcohol use, via targeting negative affect, may significantly reduce the likelihood of a smoking lapse during a quit attempt. In other words, amelioration of negative affect in heavy drinking smokers may have indirect effects on smoking cessation success by reducing alcohol use, therefore disrupting the association between alcohol and cigarette use. One study with alcohol dependent smokers found past history of mood disorder to moderate the effect of intensive smoking intervention, which incorporated CBT skills for mood management, such that those patients with a mood disorder history greatly benefit from intensive treatment (Carmody et al., 2012). Further studies in smoking cessation trials are warranted to understand the role of mood management in targeting the complex interplay between negative affect, smoking and drinking in this hard-to-treat sample of heavy drinking smokers.

The second exploratory aim of this study was to examine craving as a statistical mediator of the relationship between negative affect and cigarette and/or alcohol use. As a main effect of negative affect on cigarette use was not observed, craving was tested as a statistical mediator of the association between negative affect and alcohol use only. As hypothesized based on negative reinforcement and relief craving models of alcoholism etiology (Baker et al., 2004; Verheul et al., 1999) analysis of this mediational path revealed a full statistical mediation such that negative affect was only associated with alcohol use, insofar as it was associated with increased craving for alcohol. In other words, after accounting for the effect of craving, negative affect was not independently associated with alcohol use. These results provide additional support for relief craving models and suggest that psychological interventions that target anxiety and depressive symptomatology may reduce alcohol use through reducing the experience of craving for alcohol among heavy drinking smokers. Critically for the generalizability of this finding to a population of daily smokers, this effect was robust to controlling for age, sex and cigarette use. These analyses advance a plausible mechanism through which negative affect leads to increased alcohol use in heavy drinking smokers, namely craving for alcohol. These findings are consistent with a negative reinforcement model of alcohol use whereby the experience of negative affect, was predictive of craving for alcohol, presumably to alleviate this negative affect (Baker et al., 2004; McCarthy, Curtin, Piper, & Baker, 2010).

As smoking lapses are much more likely to occur in the context of a drinking episode (Kahler et al., 2010), long-term smoking cessation would appear to be more difficult to maintain in heavy drinking smokers as compared to light or moderate drinkers. Understanding the psychological correlates of drinking and smoking in this subgroup could help tailor treatment development including targeting negative affect. The results of this study suggest that for heavy drinking smokers, alcohol-specific interventions may be augmented by addressing anxiety and depressive symptomatology, which can directly reduce craving for alcohol and ultimately reduce problematic alcohol use in daily smokers. While state-of-the art treatment manuals of alcohol (and substance) use disorders recommend an assessment of the functional relationship between negative affect and substance use (e.g. Barlow, 2014; W. R. Miller & Arciniega, 2004), the results of the present study underscore the importance of both assessing and addressing negative affect in any treatment of AUDs.

The study should be considered in light of its strengths and weaknesses. Strengths of the study include the use of well-validated indicators of all constructs assessed. Furthermore, the study benefits from a large sample of heavy-drinking smokers providing strong statistical power. The structural equation modeling approach represents a significant strength in that it allows for the examination of several constructs while minimizing the influence of random measurement error and reduces the number of independent statistical tests, reducing the possibility of Type I error. Conversely, the cross sectional nature of the study represents a significant limitation. While mediational models often attempt to establish causal pathways, it has been argued that cross-sectional covariance structural models, such as the ones presented herein are unable to establish temporal precedence, a necessary step in determining causation (MacKinnon & Fairchild, 2009). As such we aimed to advance craving as a plausible mediator of the relationship between negative affect and drinking by demonstrating that alcohol craving was a mediator in the statistical sense. Relatedly, the implied direction of mechanistic associations in cross-sectional mediational

models is statistically arbitrary, such that reversing the direction of one path does not alter the statistical conclusions reached. In the present study this concern is mitigated somewhat by the full mediated effect, which does not allow for the mechanistic path to be reversed in a meaningful way (i.e. negative affect cannot mediate the relationship between craving and alcohol use and alcohol use cannot mediate the relationship between negative affect and craving). However, as stated above, further experimental or longitudinal research is required to establish craving for alcohol as a causal mediator of the association between negative affect and alcohol misuse in heavy drinking smokers. Additional limitations include the retrospective self-report nature of the data analyzed that may be prone to recall bias. Lastly, even with well validated measures, cross sectional self-report craving scores may not adequately capture the degree of variability in intensity and frequency of craving.

5. Conclusion

On balance, this study extends the literature on the association between negative affect and alcohol and cigarette use by demonstrating an effect of negative affect on alcohol use but not cigarette use in heavy drinking smokers. Additionally, the results of these exploratory mediational analyses advance craving for alcohol as a plausible mediator of the effect of negative affect on alcohol use requiring further empirical examination. These results are congruent with the existing literature establishing a significant positive relationship between anxiety and depressive symptomatology and alcohol use (Greeley & Oei, 1999; Jackson & Sher, 2003; Kessler et al., 1994; Prescott et al., 2000). Further research is needed to replicate and extend these results. In particular, longitudinal investigations and studies of treatment-seeking heavy drinking smokers are needed to ascertain the clinical implications of these findings as well as its causal chain.

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Contributors

Lara A. Ray is the graduate mentor to Spencer Bujarski and was the Principle Investigator for the parent study from which data was culled.

Conflict of Interest

No conflicts declared.

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