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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