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Implicit alcohol cognitions in risky drinking nicotine users with and without co-morbid major depressive disorder

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HIGHLIGHTS

- Risky drinking nicotine users with MDD had strongest implicit drinking motivations.
- Risky drinkers without comorbid nicotine and MDD had weakest implicit cognitions.
- There was a main effect of nicotine use on alcohol–approach cognitions.
- Risky drinking nicotine users with MDD were most likely to report drinking to cope.
- Results are consistent with negative reinforcement models of addiction.

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ABSTRACT

Objective: Alcohol consumption, nicotine use, and major depressive disorder (MDD) are highly co-morbid. The negative reinforcement model of addiction would suggest that smokers may consume alcohol to relieve negative affective symptoms, such as those associated with MDD and withdrawal from nicotine. Over time, these behaviors may become so strongly paired together that they automatically activate a desire to use alcohol, even in the absence of conscious or deliberate intention. This study examined implicit alcohol cognitions in 146 risky drinking nicotine users (n = 83) and non-users (n = 63), to help uncover cognitive mechanisms that link drinking, nicotine use, and depression together. We proposed that nicotine users with a history of MDD would have stronger implicit motivations to drink than non-nicotine users without MDD.
Method: Participants were assessed on lifetime MDD (n = 84) or no MDD (n = 62), and then completed an Implicit Association Task designed to test the strength of associations between alcohol pictures and "approach" words.
Results: Regression analyses showed that implicit alcohol–approach attitudes were stronger among risky drinking nicotine users than non-users. Alcohol–approach motivations were also stronger among risky drinking nicotine users compared to non-users with a history of MDD; nicotine use was unrelated to implicit alcohol cognitions for risky drinkers without MDD.
Conclusions: Implicit cognitive processes may be targeted in behavioral and pharmacological treatments in risky drinking nicotine users, particularly those with depression comorbidity.

1. Introduction

Alcohol involvement, major depressive disorder (MDD), and nicotine use have a high degree of co-occurrence (Grant et al., 2004; Hitsman, Borrelli, McChargue, Spring, & Niaura, 2003; Kessler, Chiu, Demler, & Walters, 2005; Lasser et al., 2000). The odds of nicotine dependence are almost 3 times greater among individuals with an alcohol use disorder (AUD) than those without (Hasin, Stinson, Ogburn, & Grant, 2007). Further, the odds of having MDD are 2.5 times greater among individuals with an AUD compared to those without (Grant et al., 2004), and rates of current smoking are nearly 3 times greater among individuals with MDD compared to the general population (Kessler, Chiu, et al., 2005; Kessler, Berglund et al., 2005; Lasser et al., 2000). Heavy drinking and MDD are associated with persistent smoking, greater difficulty quitting, more intense nicotine withdrawal,

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and more severe nicotine dependence (Cook et al., 2012; Weinberger, Desai, & McKee, 2010; Weinberger, Maciejewski, McKee, Reutenauer, & Mazure, 2009).

Although several theories have been purported to explain associations among drinking, nicotine use, and MDD, there is strong theoretical reason to believe that motivation to drink may be increased by nicotine deprivation and symptoms of depression (Eissenberg, 2004; Palfai, Monti, Ostafin, & Hutchison, 2000). Negative reinforcement models of addiction would suggest that negative affect states associated with nicotine craving and withdrawal and symptoms of MDD may be common processes that activate the desire to use alcohol (Brandon, Wetter, & Baker, 1996; Wills & Hirky, 1996).[1] In this manner, nicotine users and those with MDD may engage in drinking behavior because it can lessen negative affective states that are commonly associated with both nicotine withdrawal, nicotine urge reduction, and MDD, such as anxiety, irritability, depressed mood/dysphoria, fatigue, and difficulty concentrating (American Psychological Association, 2013; Bradley et al., 2011; Dani & Harris, 2005; Hughes, Higgins, & Bickel, 1994; Kassel, Stroud, & Paronis, 2003; Rose et al., 2004). Over time, the learned association between alcohol use and the alleviation of these negative affective states may become so strongly paired that the desire to drink becomes automatically and unconsciously triggered when such symptoms or states arise (Cohn et al., 2012; Greenwald, McGhee, & Schwartz, 1998; Ostafin & Palfai, 2006; Palfai et al., 2000; Piasecki et al., 2011).

Previous investigations have tested the reinforcing properties of alcohol in relation to nicotine and depression. In an experimental paradigm, Kirchner and Sayette (2007) found that alcohol increased negative reinforcement expectancies for smoking among light tobacco users (≤5 cigarettes/day), and a more recent observational study of smokers who drink suggested that alcohol and nicotine co-use had a stronger effect on negative reinforcement ratings for drinking relative to those for cigarettes (Piasecki et al., 2011). Further, in this study, problem drinking predicted negative, but not positive, reinforcement ratings from both drinking and smoking (Piasecki et al., 2011). Several other studies have found that alcohol is related to smoking urge reduction, particularly under nicotine deprivation conditions (Palfai et al., 2000; Piasecki, McCarthy, Fiore, & Baker, 2008; Rose et al., 2004). The negative reinforcement model would also suggest that individuals at risk for depression would be more likely to develop alcohol use problems later in life than those with little or no risk (Gilpin & Koob, 2008; McCarty et al., 2009, 2012). One prospective longitudinal study observed this pattern among women, with an MDD episode at age 27 increasing the risk of developing an AUD three years later (McCarty et al., 2009). Another longitudinal analysis among adolescents showed that depressive symptomology was a key predictor of alcohol use risk one year later (McCarty et al., 2012). However, evidence from other epidemiological and clinical studies is mixed. Some studies show that adult alcohol use develops "secondary" to childhood or adolescent depression vulnerability, while others indicate that depression develops in response to chronic drinking (Boden & Fergusson, 2011; Crum et al., 2008).

The Implicit Association Task (IAT) has increasingly been used to study the cognitive mechanisms that underlie drug and alcohol use behavior (Cohn et al., 2012; De Houwer, Custers, & De Clercq, 2006; Ostafin & Palfai, 2006; Tibboel et al., 2011; Wiers, Houben, & de Kraker, 2007), and may help tease apart the mechanisms linking nicotine use, drinking, and depression. The IAT is a performance-based categorization task that measures the strength of associations between two mental concepts (Greenwald et al., 1998). The primary assumption of the IAT is that individuals who exhibit regular substance use will be faster at categorizing stimuli into a category that is highly related to their substance use or frequently paired with substance use, rather than concepts that are hypothesized to be inherently incongruent with their substance use. For alcohol use or smoking, the pairing of "approach" or "pleasant" words with "alcohol" or "cigarette" stimuli has been posited to be a measurement of "desire" or "motivation" to drink or smoke. Results from this type of paradigm in drinking populations show that alcohol–approach associations are stronger for problem drinkers compared to non-problem drinkers and non-drinkers, are related to difficulty controlling alcohol use, are associated with alcohol craving, and predict heavy episodic drinking (Cohn et al., 2012; Ostafin & Palfai, 2006; Palfai & Ostafin, 2003). In relation to smoking, De Houwer et al. (2006) used the same IAT "approach–avoid" paradigm that has been applied in the alcohol field to examine implicit smoking–approach cognitions. They found that smokers were more likely to approach smoking-related stimuli than avoid them and had significantly higher smoking–approach cognitions compared to non-smokers. However, despite the high level of comorbidity among drinking, nicotine use, and depression, no studies have examined implicit reasons for alcohol use in nicotine users with and without co-occurring MDD histories.

In sum, one hypothesis for the high degree of association among nicotine dependence, MDD and heavy alcohol consumption is the strong paired association that develops between drinking and the alleviation of negative affect associated with features of each disorder. Given that alcohol serves to regulate symptoms associated with both MDD and nicotine withdrawal (Brandon, 1994), it is possible that risky drinking nicotine users who have a co-morbid history of MDD would have stronger implicit attitudes to drink, relative to risky drinkers without such comorbidities (Brandon et al., 1996; Wills & Hirky, 1996). As yet, little is known about the implicit drinking motivations that differentiate risky drinking nicotine users with and without MDD. This is unfortunate given that cognitive-behavioral interventions for each behavior (problem drinking, nicotine dependence, MDD) focus on altering maladaptive thought patterns unique to each disorder, but do not often take into account co-morbidities that may impact these thought processes.

The aims of this study were to examine the association of nicotine use to implicit alcohol–approach cognitions in risky drinkers, and to compare differences in implicit alcohol cognitions across risky drinking nicotine users and non-users with and without a history of MDD. We hypothesized that risky drinking nicotine users would exhibit significantly stronger implicit desires to drink relative to non-users. We also hypothesized that depression history would moderate the association between nicotine use and implicit alcohol motivations such that nicotine users with an MDD history would show significantly stronger implicit drinking motivations compared to nicotine users without MDD.

2. Methods

2.1. Participants

Participants were 146 risky drinkers (64% male; n = 94) recruited from two large universities, located in the southeastern and northeastern United States. Inclusion criteria were: 18 years of age or older (M = 28.62, SD = 9.91), consumed alcohol at risky levels (at least 4 standard drinks per occasion for men, 3 for women twice per month in the past 90-days), no cocaine or opiate dependence, and not currently in treatment. Half were single (57%, n = 84), employed full or part-time (47%, n = 69), and White (56%, n = 82).

[1] Other potential mechanisms have been proposed to explain the links among smoking, depression, and alcohol use, including recurrence of a major depressive episode provoked by nicotine abstinence (Borrelli et al., 1996; Glassman, Covey, Stetner, & Rivelli, 2001), alleviation of major depressive episodes by alcohol intake (McCarty et al., 2009), alleviation of alcohol withdrawal symptoms via nicotine ingestion (Lajtha & Sershen, 2010), underlying genetic or neurobiological deficits that impact neurotransmitter systems implicated in the dopaminergic reward/deficit system (Hurley, Taylor, & Tizabi, 2012; Littleton, Barron, Prendergast, & Nixon, 2007), and a general tendency for persons with mental health problems to smoke and engage in other addictive behaviors (Hitsman, Moss, Montoya, & George, 2009; Kalman, Morissette, & George, 2005; Le Strat, Ramoz, & Gorwood, 2010; Mackowick, Lynch, Weinberger, & George, 2012).