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Evaluating the influence of at-risk alcohol use on factors associated with smoking cessation: Combining laboratory and ecological momentary assessment *

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Abstract

Objective—Most smokers want to quit but most cessation attempts end in failure. Alcohol consumption is associated with smoking behavior and relapse. We examined the associations between severity of drinking and psychological processes during a cessation attempt in the laboratory and during a quit attempt.

Methods—Smokers ($N=209$) enrolled in a smoking cessation study were followed from 2 weeks pre-quit through 4 weeks post-quit. Participants scoring 0–7 and 8–15 on the Alcohol Use Disorders Identification Test (AUDIT) were classified as low-risk and high-risk drinkers, respectively. Participants attended one pre-quit laboratory session before which they were required to abstain from smoking and another pre-quit session before which they smoked normally. Craving was assessed in the laboratory with the Questionnaire for Smoking Urges (QSU). A subsample of the participants also completed a 1-week ecological momentary assessment (EMA) study starting on the quit day. During EMA, craving for cigarettes was assessed, and attentional bias was assessed using a smoking Stroop task ($n=119$).

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Author Disclosures

Nothing declared.

Contributors

Ms. Sells was the lead investigator for analysis and manuscript development. Dr. Waters was the principal investigator of the protocol and was involved in all aspects of study design and data collection as well as data analysis and manuscript development. Dr. MacLean assisted with manuscript development. All authors have approved the final article.

Conflict of Interest

No conflict declared.

Results—High (vs. low) risk participants reported greater abstinence-induced increases in craving in the laboratory, and also exhibited greater attentional bias on the smoking Stroop task during EMA.

Conclusions—High-risk drinkers exhibited a stronger increase in desire to smoke in abstinence and greater attentional bias to smoking cues early in a quit attempt, both of which may motivate continued smoking behaviors. High-risk drinkers may require more intensive or different smoking cessation interventions.

Keywords

Attentional Bias; Smoking; Craving; Alcohol; Hazardous Drinking

1. Introduction

Cigarette smoking remains the single most preventable cause of morbidity and mortality in the U.S. (Centers for Disease Control and Prevention [CDC], 2012). Smoking cessation rates remain low (Agaku et al., 2014) with systematic reviews highlighting relapse as the modal outcome (Piasecki, 2006). The covariation of alcohol consumption and cigarette smoking is well documented (Falk et al., 2006) and excessive use of alcohol is a risk factor for poor smoking treatment outcome (Cook et al., 2012; Kahler et al., 2009; McKee et al., 2006; Shiffman et al., 1996). Cross-sectional studies have demonstrated that heavy drinkers are more likely to be a smoker, smoke more heavily, and have more difficulty quitting (Dawson, 2000; Toll et al., 2012). Conversely, current smokers, compared to never smokers, have over three times the odds of reporting hazardous drinking (McKee et al., 2007). Laboratory studies have suggested that an individual is more likely to crave or smoke cigarettes after drinking alcohol (Barrett et al., 2013; McKee et al., 2006; Sayette, 2002). Research employing ecological momentary assessment (EMA), in which behavior is repeatedly sampled within an individual's natural environment (Shiffman, 2014), has demonstrated that alcohol use often triggers smoking (Shiffman et al., 2002; Shiffman et al., 1997) and relapse (Shiffman et al., 1996). Furthermore, an EMA study in daily smokers suggested a reciprocal relationship between alcohol and cigarettes with co-use predicting higher subsequent craving for both substances (Piasecki et al., 2011). Event-level reports within the same individual also reveal that drinking alcohol (vs. non-drinking) is associated with a three-fold increase in the number of cigarettes smoked (Witkiewitz et al., 2012).

Collectively, research establishes reciprocal associations between alcohol and cigarette use; however, few studies have explored mechanisms that underlie this relationship. EMA research has suggested that self-reported alcohol use is associated with increased pleasure from recent smoking and vice versa (Piasecki et al., 2006; 2011). Co-use may result in increased salience of both smoking and alcohol cues (e.g., cross-cue reactivity; Erblich et al., 2009; Tomko et al., 2016) that may be associated with a concurrent increased tendency to attend to drug specific stimuli ("attentional bias") (Field et al., 2005).

Although laboratory studies have examined the effect of acute alcohol administration on craving (e.g., McKee et al., 2006; Tomko et al., 2016) and attentional bias to cigarettes (Field et al., 2005), we are not aware of a study examining the association between self-

reported risky alcohol use and smoking-related processes, either in the lab or using EMA. We hypothesized that, across all participants, cigarette craving would be higher in smoking abstinence and this effect would be stronger in high (vs. low) risk drinkers in the laboratory. We also predicted that during a 7-day EMA protocol, high (vs. low) risk drinkers would exhibit greater attentional bias to smoking cues and self-reported cigarette craving early in a quit attempt.

2. Materials and Method

2.1. Participants

The study is a secondary data analysis from a multisite study conducted at the University of Texas M. D. Anderson Cancer Center in Houston, Texas ($n=150$) and at the Uniformed Services University of the Health Sciences in Bethesda, MD ($n=118$) examining cognitive processes in smoking cessation (for further detail see Waters et al., 2014). Briefly, smokers planning to quit without the use of pharmacotherapy attended up to six laboratory visits: a screening visit, two pre-quit laboratory sessions (“Week -2”, “Week -1”), a quit day visit (“Week 0”), a post-quit visit (“Week +1”) and a visit at end of treatment (“Week +4”). At one pre-quit laboratory session participants were required to remain abstinent for twelve hours prior to the session (“abstinent” session), and at the other participants were required to smoke regularly before the session (“non-abstinent” session). Odd-numbered participants completed the non-abstinent session at Week -2 and the abstinent session at Week -1, and vice versa for even-numbered participants. If a participant reported smoking on the day of the abstinent session or had high CO levels (> 10 ppm), the session was rescheduled. Some participants volunteered to participate in a 7-day EMA study that started on the quit day. On quit-day (Week 0), participants were instructed to try to maintain abstinence after waking up; they attended the quit-day session later that day.

The Institutional Review Boards at both sites approved study procedures.

2.2. Treatment

All participants received a standardized self-help manual using a standard relapse prevention/coping skills approach, brief (15–20 minute) individualized smoking cessation counseling from a licensed counselor based on approaches described in Treating Tobacco Use and Dependence Clinical Practice Guideline (Fiore et al., 2008) at each visit, and two brief telephone counseling sessions between Week 0 and Week +4.

2.3. Measures

The Alcohol Use Disorders Identification Test (AUDIT, Bohn et al., 1995) and Fagerström Test of Nicotine Dependence (FTND, Heatherton et al., 1991) were administered at screening. Participants scoring 0–7 on the AUDIT were designated “low-risk” drinkers. Participants scoring 8–15 were designated as “high-risk”. Participants scoring 16 or more (“hazardous”) were excluded from the study, as assessment of cognition may be less reliable in these individuals. A score of 8 is a clinical cut-off commonly used to identify those at risk for habitual alcohol use and has excellent sensitivity and acceptable specificity (Allen et al.,

1997). Craving was assessed during lab visits using the 10-item Questionnaire of Smoking Urges (QSUBrief, Cox et al., 2001).

2.4. EMA Assessments

On quit day, participants had the opportunity to volunteer in the 7-day EMA study using a personal digital assistant (PDA; HP iPAQ Pocket PC) (see Waters et al., 2014). Briefly, participants completed three types of assessments: random assessments (RAs; scheduled for four times/day; median completion rate of presented RAs = 76.00%) and two participant-initiated assessments (when experiencing a temptation, and first lapse to smoking). Each assessment included questions assessing craving (“I am craving a cigarette”; 1=strongly disagree to 7=strongly agree), drinking in past two hours (“No consumption”, “A little”, or “A lot”; dichotomized as No consumption vs. Any drinking), and recency of smoking (“Just smoked/smoking now”, “5–30 minutes”, “31 minutes to 2 hrs”, “greater than 2 hours”). At every other assessment, a smoking Stroop task was administered to evaluate naturalistic attentional bias to smoking cues. Participants were instructed to indicate the color (red, green, blue) of individual words presented on the PDA screen by pressing one of three buttons on the PDA. Each list contained a block of 33 smoking words and a block of 33 neutral words, presented in a counterbalanced order. The smoking Stroop effect was computed as median reaction time (RT) on smoking words minus median RT on neutral words. Therefore, a greater Stroop effect corresponds to a greater attentional bias for smoking stimuli.

2.5. Data Analysis

Overall, 268 participants were recruited. Analysis of laboratory data included those who completed both pre-quit visits ($N=209$) (Table 1); 119 participants contributed Stroop data during EMA. There were no differences on demographic/smoking variables assessed at orientation between study subsamples except that the sample who completed both lab visits were older than non-completers (Supplementary Materials¹).

Analysis of laboratory data used multifactor ANCOVA, with one between-person factor, Group (high vs. low-risk), and one within-person factor, Session (abstinent vs. non-abstinent). ANCOVA with one between-person factor, Group (high vs. low-risk) and one within person factor, Visit (non-abstinent session vs. Quit day), was also used to analyze craving ratings at nonabstinent session and quit day. Analysis of EMA data used linear mixed models (LMM) with SAS PROC MIXED (continuous outcomes: craving, smoking Stroop) or SAS PROC GLIMMIX (binary outcome: presence/absence of drinking in past 2 hours, assessed during EMA). Age was included as a covariate in all ANCOVAs and LMMs.

3. Results

High and low-risk participants did not differ in demographic or smoking variables except age (Table 1), or in number of RAs ($p = .43$) completed. As expected, high (vs. low) risk

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participants reported drinking at a higher proportion of assessments during EMA (18.4% vs. 3.9% respectively), $F(1, 2320) = 21.44, p < .0001$.

ANCOVA conducted on craving ratings in the laboratory revealed a main effect of Session, $F(1, 206) = 15.37, p < .001$, indicating that craving was higher in the abstinent (vs. non-abstinent) session, and a Group x Session interaction, $F(1, 206) = 5.01, p = .03$, indicating that abstinence-induced increase in craving was greater in high (vs. low) risk participants (Fig. 1). When comparing craving at the non-abstinent session to quit-day (Fig. 1), a separate 2-factor ANCOVA revealed a Group x Visit interaction, $F(1, 193) = 6.70, p = .01$, indicating that the effect of Visit (2 levels: non-abstinent vs. quit-day) was greater in high (vs. low) risk participants. Craving ratings were higher on quit-day (vs. non-abstinent session) in high-risk participants ($p = .02$) but not in low-risk participants ($p = .07$) (Fig. 1).

High (vs. low) risk participants exhibited greater attentional bias on the smoking Stroop task during EMA (Means = 36.62 ms vs. 12.24 ms respectively), $F(1, 1166) = 7.14, p = .008$, an effect that persisted when controlling for presence of recent smoking and drinking. However, unlike the laboratory data, high-risk participants did not report higher craving ratings during EMA ($p > .10$). The effect of Group on these outcomes was not moderated by recent smoking or drinking reported with EMA (p 's $> .10$).

4. Discussion

High-risk (vs. low-risk) drinkers reported greater abstinence-induced increases in craving during smoking abstinence. This was true when craving was assessed after overnight abstinence prior to a quit attempt and when craving was assessed on the first day of a quit attempt. Our findings therefore support reciprocal influences of alcohol and tobacco on craving in human laboratory studies that explicitly manipulate alcohol and cigarette administration (Verplaetse and McKee, 2016). In the current study, however, alcohol was not administered in the laboratory. Rather, high-risk drinkers were defined as endorsing more problems associated with alcohol consumption. This suggests the reciprocal relationship between alcohol and abstinence-induced cigarette craving may not solely be attributable to intoxicating effects of alcohol. Rather, those who self-report high-risk drinking may also report greater increases in cigarette craving during deprivation than low-risk counterparts as a result of cross-cue reactivity developed through frequent pairings of alcohol and cigarettes.

High-risk (vs. low-risk) drinkers did not report greater craving for cigarettes during EMA, but they did demonstrate a greater attentional bias for smoking cues. The greater attentional bias in high-risk drinkers persisted when controlling for recent smoking or drinking, and was not moderated by these variables. Thus, elevated attentional bias in high-risk drinkers is unlikely to be solely due to an acute effect of alcohol.

In the current study, high-risk drinkers also reported more instances of drinking early in a quit attempt, suggesting that high-risk drinkers have increased opportunity for co-use in daily life and potentially greater cross-cue reactivity for smoking and alcohol stimuli. An EMA investigation of adult smokers evaluating cue-reactivity to smoking, alcohol, and stress cues reported that recent alcohol use was associated with increased tonic (non-cue-elicited)

cigarette craving, but not cue-elicited craving, however frequency of alcohol use during EMA was associated with increased smoking cue-reactivity (Tomko et al., 2016). Thus, heaviness of alcohol use may be associated with craving when increased by deprivation (current study) or when provoked by smoking cues (Tomko et al., 2016).

Several limitations should be considered. Due to the small sample of high-risk drinkers, the study was underpowered to examine relationships between drinking risk and relapse, or to examine drinking risk as a moderator variable. Future studies are needed to establish the generalizability of our findings. Analyses focused on examining a between-person association between at-risk drinking predicting smoking-related variables; the opposing relationship of smoking-related variables predicting drinking cannot be assumed. Additionally, within-person associations could not be examined in the laboratory because alcohol was not manipulated as an independent variable, and the low number of reports of drinking in the low-risk group during EMA prohibited reliable within-person models of drinking and attentional bias in the field. We did not assess craving for alcohol, and could not examine the differential roles for cigarette and alcohol craving. As with all EMA studies, reactivity may influence mean responses, although patterns of association may be less affected (Shiffman, 2015). Finally, the current study did not recruit individuals with alcohol use disorders, which may be necessary to establish interventions.

Despite these limitations, our preliminary findings using laboratory and EMA methods suggest high-risk drinkers exhibit greater abstinence-induced craving in the laboratory and greater attentional bias early in a quit attempt. These results offer initial evidence that greater abstinence-induced cigarette craving combined with greater attention to smoking cues during a quit attempt may increase the probability of a relapse in high-risk drinkers. If confirmed, the data suggest interventions targeted at reducing craving or attention to drug stimuli could be useful for high-risk drinkers. Although recent reports question the effectiveness of interventions that train smokers to attend away from smoking stimuli (Begh et al., 2015; but see Kakoschke et al. 2017), attentional retraining administered in the field may have more utility (Cox et al., 2014). Future research would benefit from evaluating craving and attentional bias for both smoking and alcohol in the laboratory and field to explore possible mechanisms that drive co-use and treatment failure.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- High-risk drinkers reported greater abstinence-induced increases in craving.
- High-risk drinkers exhibited greater attentional bias to Stroop smoking words.
- High-risk drinkers may have an increased risk of smoking relapse during abstinence.
- High-risk drinkers may benefit from different types of smoking cessation treatment.

Figure 1. Mean QSU Ratings (± 1 Standard Error) at the non-abstinent session, abstinent session, and quit-day. The change (increase) in craving from the non-abstinent to abstinent sessions is significantly greater in the High (vs. Low) AUDIT group, $p < .05$.

Table 1

Summary Statistics for Low AUDIT and High AUDIT groups

	Whole Sample (<i>N</i> =209)	Low AUDIT (<i>n</i> =188)	High AUDIT (<i>n</i> =21)	χ^2	<i>p</i>
Demographic and Smoking Data					
Age	43.93 (11.81)	44.66 (11.51)	37.43 (12.71)	2.71	.008
Sex (%)				1.38	.23
Male	54.55	53.19	66.67		
Female	45.45	46.81	33.33		
Race (%)				0.95	.62
White	54.07	53.19	61.90		
Black	38.28	39.36	28.57		
Other	7.66	7.45	9.52		
Years of Education	14.19 (2.31)	14.18 (2.33)	14.29 (2.15)	-0.20	.84
Baseline Cigarettes Per Day	19.60 (8.87)	19.85 (9.09)	17.33 (6.32)	1.24	.22
FTND	5.20 (2.02)	5.29 (2.02)	4.43 (1.94)	1.86	.07
CO (Orientation)	21.32 (10.30)	21.45 (10.24)	20.14 (10.97)	0.55	.58
Cotinine (ng/ml)	381.12 (219.90)	387.54 (225.39)	324.25 (156.16)	1.25	.21

Note: Data shown are for participants who completed at least one laboratory assessment. Data are Mean (SD) (continuous variables) or percentages (categorical variables). Data are broken down by Low (AUDIT 7) and High (AUDIT 8) risk drinking at baseline (see text for details). FTND = Fagerstrom Test for Nicotine Dependence; CO = Carbon monoxide