

Alcohol Dose–Dependent Increases in Smoking Urge in Light Smokers

Andrea C. King and Alyssa M. Epstein

Background: The current study assessed dose-dependent effects of alcohol compared with placebo on ratings of urge to smoke in light smokers.

Methods: Sixteen nonalcoholic social drinker-smokers were tested individually in three separate early evening sessions where they received a placebo (with 1% ethanol as a taste mask), a low-dose (0.4 g/kg) alcoholic beverage, or high-dose (0.8 g/kg) alcoholic beverage administered in random order. Participants refrained from smoking 2 hr before and throughout the entire early evening experimental sessions. Two subfactors of the Brief Questionnaire of Smoking Urges, BQSU; (factor 1, urge to smoke for stimulation; factor 2, urge to smoke to relieve negative mood and withdrawal) were assessed at baseline and again at rising and declining portions of the blood alcohol curve.

Results: Both the high and low doses of alcohol significantly increased BQSU factor 1 scores during the rising and declining blood alcohol concentration (BAC) limbs ($p < 0.05$). Comparisons across doses during both limbs revealed that the high dose significantly increased factor 1 smoking urge compared with the low dose and placebo beverage ($p < 0.05$, high > low = placebo). Alcohol tended to increase factor 2 scores throughout the BAC curve, but levels were not as increased as factor 1 scores. Finally, there was no significant association between participants' smoking levels and smoking urge ratings during the high- and low-dose sessions.

Conclusions: The results support a dose-dependent alcohol-induced increase in smoking urge in cigarette-deprived light smokers. These smoking urge increases were apparent during the rising limb of the BAC and maintained throughout the declining limb. Smoking urge increases were greater for positive reinforcing effects than for negative reinforcing effects.

Key Words: Alcohol, Urge to Smoke, Cigarette Smoking, Light Smokers.

THERE ARE NUMEROUS studies showing a positive association between cigarette smoking and alcohol drinking. Studies of adolescents, college students, and general adult samples indicate that use of either substance increases the risk for the other, and stronger associations are found between these substances at higher use patterns (Chen and Kandel, 1995; Kandel and Yamaguchi, 1993; Sher et al., 1996; Torabi et al., 1993; Zacny, 1990). Persons with alcohol use disorders are more likely to smoke and smoke at heavier levels than the general population (Bien and Burge, 1990; Bobo, 1992; Shiffman and Balabanis, 1995; Toneatto et al., 1995), and heavy social drinkers (i.e.,

persons who consume five or more drinks per occasion at least monthly) tend to have lower odds of success in smoking cessation (Dawson, 2000).

Laboratory paradigms examining the effects of alcohol on cigarette smoking have primarily measured smoking topography, i.e., ad libitum cigarettes smoked, number or intensity of puffs taken, and carbon monoxide levels. Results have shown that compared with placebo, alcohol potentiates subjective rewarding effects of nicotine (Rose et al., 2004), as well as cigarette smoking behavior (Griffiths et al., 1976; Henningfield et al., 1983, 1984; Mintz et al., 1985). This effect may be more pronounced in heavier, nonalcoholic drinkers than lighter drinkers (Henningfield et al., 1984). Alcohol-induced increases in cigarette smoking may also relate to the amount of alcohol consumed (Nil et al., 1984), particularly during the first hour after drinking (Mitchell et al., 1995). These data suggest that craving, presumably leading to smoking behavior (Shiffman et al., 1997), may also be heightened when blood alcohol concentrations (BACs) are increasing, i.e., when alcohol-induced positive subjective effects are prominent (Martin et al., 1993). However, alternative theories suggest that cigarette urge and smoking behavior increase after alcohol consumption to offset the sedative effects of alcohol (for review, see Zacny, 1990), which typically emerge during the latter por-

From the University of Chicago, Pritzker School of Medicine, Department of Psychiatry, Chicago, Illinois (ACK); and the Illinois Institute of Technology, Institute of Psychology, Chicago, Illinois (AME).

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Reprint requests: Andrea C. King, PhD, University of Chicago, Department of Psychiatry, 5841 South Maryland Avenue, Chicago, IL 60637; Fax: 773-702-6454; E-mail: aking@yoda.bsd.uchicago.edu

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tions of the BAC (Martin et al., 1993), especially in heavier social drinkers (King et al., 2002).

Cigarette craving is an important factor involved in nicotine addiction and is thought to be one of the underlying mechanisms involved in concomitant alcohol-smoking behaviors. However, the effect of alcohol on smoking urge in the absence of smoking or *in vivo* cigarette cues, which may exert their own independent effects on craving, has not been systematically examined. The two studies investigating alcohol-induced changes in smoking urge in the absence of cigarette smoking (Burton and Tiffany, 1997; Sayette et al., 2005) incorporated a smoking cue (i.e., exposure to a lit cigarette) shortly after consumption of a single, moderate-high dose of alcohol (approximate three to four drink equivalent). In both studies, alcohol increased participants' subsequent ratings of urge to smoke, although only one of the studies (Sayette et al., 2005) found that alcohol further augmented craving to the *in vivo* cigarette cue compared with the neutral cue. However, the lack of data on cigarette craving after various doses of alcohol consumption in these studies limits conclusions that can be drawn regarding potential linear or nonlinear drug interactions (for review, see Perkins, 1997).

There is also scant information on the effects of alcohol on smoking urge in lighter smokers because existing studies have generally been limited to heavy, nicotine-dependent smokers who smoke 10 or more cigarettes per day. Only one study (Sayette et al., 2005) recruited both heavy smokers (20–40 cigarettes per day) and tobacco chippers (smoking 1–5 cigarettes at least 2 days per week). The latter group was expected to show greater increases in smoking urge after alcohol consumption because alcohol may be a more potent stimulus to trigger cravings in lighter versus heavier, regular smokers (Shiffman et al., 1994). However, the data revealed that both groups showed comparable alcohol-related increases in cigarette craving, with the heavy smokers reporting greater overall cravings. Although the potentiating effects of alcohol on smoking urge may be similar among smoker subtypes, it remains to be determined whether there are differential effects based on alcohol dose, BAC limb, or smoking urge factors (positive versus negative reinforcing effects).

The primary goal of the current study was to examine dose- and BAC limb-dependent effects of alcohol on smoking urge in light smokers. This placebo-controlled paradigm was designed to help elucidate whether increased smoking urge is due to direct pharmacological or priming effects of alcohol or to learned associations (i.e., similar increases in smoking urge across alcohol doses and placebo). Heavy social drinkers were examined to target consumers of alcohol who smoke and binge drink on a frequent basis without adding the confounds of tobacco or alcohol withdrawal on the subjective and behavioral measures. The secondary goal was to investigate the relationship between participants' smoking patterns and acute smoking urge changes at both rising and declining limbs. It was hypoth-

Table 1. Demographic Variables for Smoker-Drinkers

Demographics	
Age (years)	25.8 (0.6)
Education (years)	15.3 (0.3)
Race (% white)	75%
Sex (male/female)	11/5
Smoking behavior	
Smoking occasions/week ^a	5.2 (0.4)
Cigarettes smoked/occasion	6.1 (0.9)
FTND ^b total	1.3 (0.6)
Smoking duration (years)	6.8 (0.8)
Drinking behavior	
Drinking occasions/week	3.4 (0.3)
Alcohol drinks/occasion	7.1 (1.7)
No. of reported binges ^c in past 6 months	61.8 (8.8)

Data are mean (SEM).

^a $n = 7$ reported daily smoking; $n = 9$ reported smoking 3 to 6 days/week.

^b The Fagerström Test for Nicotine Dependence (Heatherton et al., 1991) is a six-item questionnaire assessing levels of nicotine dependence, yielding possible scores from 0 to 10.

^c Binge is defined as five or more drinks/occasion for males and four or more drinks/occasion for females.

esized that there would be an inverse relation between average daily smoking level and alcohol-induced smoking urge.

METHODS

Participants

Participants were 16 nonalcoholic, heavy social drinkers (11 males, 5 females) with light smoking backgrounds. To be eligible, participants had to have reported regular use of cigarettes at least three times weekly with less than 12 cigarettes per smoking day, and weekly moderate to heavy alcohol consumption (10–40 drinks weekly with at least one weekly “binge” episode [≥ 5 drinks per occasion, 4 for females]). The demographic and background characteristics of the sample are listed in Table 1. Participants were recruited through newspapers, word-of-mouth referrals, and local flyers. A brief phone screen by the research assistant determined general participant exclusion for major medical and psychiatric disorders and inclusion for the social drinker-smoker criteria. For approximately half of the participant sample, subjective and performance effects have been previously described in investigations of alcohol response in nonalcoholic heavy social drinkers (King and Byars, 2004; King et al., 2002).

Eligible persons from the phone screens were invited for an in-person screening session that consisted of a brief physical examination by a resident physician, laboratory tests (liver function tests and urine toxicology), and a psychiatric interview and questionnaires to rule out history of psychiatric disorder or alcohol or drug dependence (i.e., *DSM-IV* criteria; American Psychiatric Association, 1994). Female candidates were given pregnancy tests (the results of which were all negative), and they reported no plans to become pregnant or breastfeed during participation.

Procedure

The protocol was approved by the University of Chicago Institutional Review Board. Each subject participated in three laboratory sessions, which were conducted in a comfortable living room-like environment and separated by at least 48 hr. All participants received a placebo session (1% volume of ethanol as a taste mask), a low-alcohol-dose session (0.4 g/kg; 8% volume alcohol; approximate 2 drink equivalent), and a high-alcohol-dose session (0.8 g/kg; 16% volume alcohol; approximate four drink equivalent), in random order. All drinks were prepared with the appropriate dose of 180-proof ethanol mixed with water, artificial sweetener, and sugar-free grape Kool-Aid® (Kraft, Northfield, IL) and divided into two equal-sized portions. To attain similar BAC levels for men and

women, alcohol doses were adjusted for female participants at an approximate 90% volume equivalent to that for men. To reduce alcohol expectancy, the consent form, which was completed before participation, stated that the testing sessions could include administration of a stimulant, sedative, alcohol, or placebo. The participant was instructed to abstain from consuming food or cigarettes for at least 2 hr before the session and to abstain from taking any medications or substances 12 hr before and after the sessions.

The participants arrived for each session at approximately 4:30 PM and were given a low-fat snack (15% daily calories), followed by completion of baseline questionnaires and measures. Participants submitted to a breathalyzer (Alco-Sensor III, Intoximeter, Inc., St. Louis, MO) at the beginning of each session to ensure recent alcohol abstinence. At 5:30 PM, in the presence of the experimenter, the participant consumed the first beverage portion over a 5-min period, followed by a 5-min rest, and then had 5 min to consume the second beverage portion. Participants completed measures for the next approximate 3 hr, which consisted of breath tests and questionnaires. Other tests and physiological measures were taken after their questionnaires at various intervals as part of a larger study (King and Byars, 2004; King et al., 2002). During intervals where no measures were obtained, the participant was allowed to read or watch videotapes. At the end of the session (approximately 9:00 PM), the participant was transported home.

The main dependent measure was the 10-item Brief Questionnaire of Smoking Urges (Cox et al., 2001), a reliable and valid scale of cigarette craving, that consists of two factor-derived subscales. Factor 1 reflects desire to smoke for stimulation or positive reinforcement and includes items such as "I have a desire for a cigarette right now" and "A cigarette would taste good now." Factor 2 reflects urge to smoke to relieve nicotine withdrawal or negative reinforcement and includes items such as "I could control things better right now if I could smoke" and "Smoking would make me feel less depressed." The Brief Questionnaire of Smoking Urges was completed at baseline, 15 min after alcohol consumption (rising BAC), and 105 min after consumption (declining BAC).

Instrumentation for estimating BACs from breath samples used the Alco-Sensor IV (Intoximeter Inc., St. Louis, MO). To minimize experimenter bias, the Breathalyzer was preset to display readings of 0.000 for all measures during testing, with the actual levels downloaded to a computer after the participant completed the study. Breathalyzer readings were obtained regularly throughout the session: baseline and 15, 45, 105, and 165 min after consumption. The first rising BAC measure was obtained 15 min after the completion of the beverages, because breath alcohol samples are only valid after this initial interval after alcohol consumption. Due to one instrumentation error, complete BAC data were available for 16 of the 17 participants.

Statistical Analyses

Smoking urge data were analyzed by repeated-measures ANOVA, with dose and time as the within-subjects factors. Significant main effects and interaction terms were further examined by simple effects tests. To protect against violations of sphericity, Greenhouse-Geisser corrections were used to adjust the degrees of freedom for the F tests. Pearson correlations were computed in exploratory analyses of the associations between participants' smoking levels (average daily number of cigarettes) and smoking urge ratings during the high- and low-dose sessions.

RESULTS

For BAC readings, as expected, alcohol concentrations determined by breath samples showed the highest levels during the high-dose session compared with the low-dose and placebo sessions [dose \times time: $F(8, 112) = 56.01, p < 0.001$]. In terms of capturing rising and declining limbs, Fig. 1 illustrates that during the first time point at 15 min after beverage completion, BAC levels were on the sharply rising

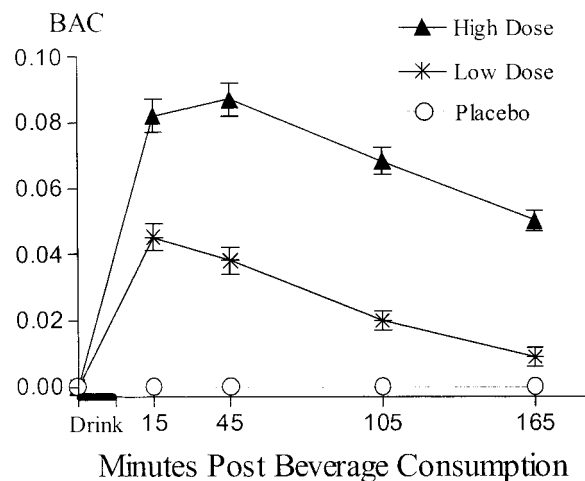


Fig. 1. Blood alcohol curve. Results show the BAC at the predrink baseline and at 15, 45, 105, and 165 min after drink consumption.

ascending limb, and at 105 min, BAC levels were reflective of the slow rate of alcohol elimination during descending BACs.

As shown in Fig. 2, factor 1 scores showed a significant dose by time interaction [dose \times time: $F(4,60) = 4.32, p < 0.005$]. Both the high and low alcohol doses increased factor 1 smoking urge during ascending BACs compared with their respective baselines (simple effects, $p < 0.05$), whereas the placebo beverage did not produce changes in urge over time. Across doses at this rising time point, it was the high dose that produced the greatest increases in smoking urge (simple effects, $p < 0.05$, high $>$ low = placebo). These alcohol dose-related increases in factor 1 craving were maintained through the declining limb (simple effects, $p < 0.05$, high $>$ low = placebo). For factor 2, there was a trend for alcohol to increase craving in a similar pattern as with factor 1: both high and low doses produced factor 2 increases for the rising limb, which was maintained during the falling limb [dose \times time: $F(4, 60) = 2.19, p = 0.08$]. As seen in Fig. 2, the absolute changes for factor 2 were of a much smaller magnitude than for factor 1.

An exploratory analysis of the relation between alcohol-induced increases in factor 1 smoking urge (change from base to rise) and background smoking levels revealed no significant associations at either the high alcohol dose [$r(14) = -0.35, p = \text{not significant (NS)}$] or the low alcohol dose [$r(14) = -0.04, p = \text{NS}$]. There were also no significant associations between baseline smoking levels and increases in factor 2 smoking urge for either the high dose [$r(14) = -0.25, p = \text{NS}$] or the low dose [$r(14) = -0.04, p = \text{NS}$].

DISCUSSION

The findings of the current study revealed that compared with placebo, alcohol dose-dependently increased cigarette craving in smoking-deprived participants. Alcohol increased factor 1 smoking urge (desire to smoke for positive

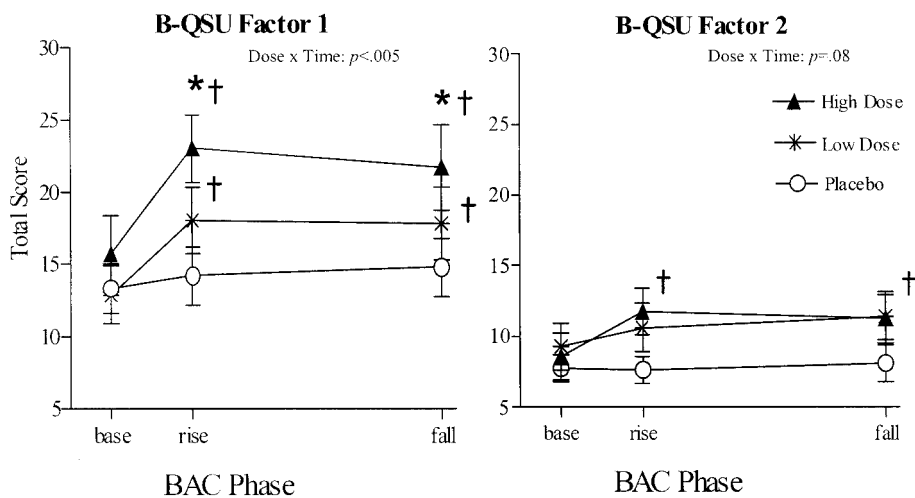


Fig. 2. Brief Questionnaire of Smoking Urges (BQSU factor 1 and factor 2 scores). Base = predrink baseline; rise = 15 min after drink consumption; fall = 105 min after consumption. High alcohol dose = 0.8 g/kg and low dose = 0.4 g/kg. †Significantly greater than baseline at that time ($p < 0.05$); *significantly greater than other doses ($p < 0.05$).

reinforcement) from predrink baseline during the rising limb of the BAC, and this increase was maintained during the latter portion of the BAC. Further, the high alcohol dose rather than the lower dose produced the most elevated smoking urge increases after alcohol consumption. The high alcohol dose also tended to produce modest, nonsignificant increases in factor 2 scores (desire to smoke to relieve negative mood and withdrawal), but to a much lesser extent than for factor 1 (Fig. 2).

Taken together, these findings lend support to the theory that in heavy social drinkers, alcohol produces dose-dependent increases in cigarette craving that emerge when blood alcohol levels are rising. Given the higher magnitude of smoking urge increases in factor 1 compared with factor 2, the data support the hypothesis that alcohol-related desire to smoke is more related to enhancement of positive mood states rather than through negative reinforcement. The findings of the current study argue against the theory that the main role of cigarette smoking during a drinking episode is to offset sedative-type effects or negative reinforcement factors. Even though the latency since the last cigarette was greater during the declining limb than during the rising limb, neither factor 1 nor factor 2 urge ratings continued to increase throughout the BAC curve. However, there are several alternative interpretations of the results, including acute tolerance (i.e., development of tolerance within a session) or possible ceiling effects such that craving levels might be at or near their peak in these nondependent smokers. Future studies examining more specific limb effects with and without smoking cues and equating BAC limbs on time since last cigarette may help to further discern temporal effects in alcohol-induced cigarette craving.

The majority of previous studies examining the effects of alcohol on smoking urge have typically used in vivo smoking cues (Burton and Tiffany, 1997; Sayette et al., 2005) or other measures of smoking topography (Griffiths et al., 1976; Henningfield et al., 1983, 1984; Mintz et al., 1985; Mitchell et al., 1995; Nil et al., 1984). Such smoking cues

produce their own independent effects on smoking urge (Hutchison et al., 1999), thereby making it difficult to isolate the independent effects of alcohol on smoking urge. The current study provided a unique focus on alcohol dose and BAC limb effects of alcohol on smoking urge, although it may be argued that alcohol drinking itself or repeated craving surveys might act as interoceptive smoking cues to participants. To control for alcohol expectancies acting as a cue for the desire to smoke, participants were informed that they could receive any of several categories of substances, including a sedative, a stimulant, alcohol, or a placebo (which included an alcohol taste mask). By the end of each session, approximately one third of participants were incorrect in their estimates of what category of substance they thought they received. Although expectancy or paired-associate effects therefore cannot be completely ruled out as a contributor to the observed effects, they certainly alone cannot explain the main findings of the study, that greater quantities of alcohol produced more heightened increases in smoking urge. Intermediate results observed in the low-alcohol-dose session confirm that these increases occur in a linear, dose-dependent manner. Moreover, higher alcohol doses, which produce more prominent central pharmacological effects, further augmented smoking urge beyond that of the low dose.

By examining regular social drinker-smokers, this study examined a subgroup of interest in terms of chronic, frequent consumption of both substances, while reducing the potential interfering effects of alcohol or tobacco withdrawal in subjective assessments if alcohol or heavy nicotine-dependent participants were examined. It is important to note that there may be important differences in alcohol-smoking interactions based on participants' drinking or smoking phenotype. A recent study showed that placebo beverage increased cigarette craving at 10 min in heavy and light smokers of moderate drinking backgrounds (Sayette et al., 2005). In contrast, in the current study, placebo beverage produced little change in smoking urge across the entire session, even as time since last cigarette

continued to increase. We may speculate that our heavier drinkers, who shown greater sensitivity to subjective alcohol stimulation than lighter drinkers (Holdstock et al., 2000; King et al., 2002), may also show more sensitivity to alcohol-induced cigarette craving than lighter drinkers. In terms of smoking phenotype, within our sample of light smokers, smoking background was not associated with alcohol-induced increases in smoking urge, similar to findings in heavy smokers and chippers (Sayette et al., 2005).

Clinical implications of this laboratory study suggest that smokers desiring to quit should avoid or minimize alcohol drinking and be aware that cigarette cravings during drinking may occur in nonsmoking environments (i.e., not be confined to bar-type settings). Because no amount of cigarette smoking is considered to be "safe," encouraging drinkers at all levels of smoking to reduce their alcohol consumption may in turn reduce frequency or magnitude of cigarette cravings, ostensibly reducing cigarette consumption levels and promoting a healthier lifestyle. Further, although some tobacco chippers may show a fairly consistent smoking pattern (Shiffman et al., 1992), repeated exposure to alcohol may place light-smoking heavier drinkers at risk for development of nicotine dependence. Although these issues are beyond the scope of the current study, future longitudinal studies following various trajectories of social drinker-smokers may help to answer this question.

Some limitations of this study should be mentioned. First, although the purpose of the study was to examine alcohol-induced cigarette craving without the confounds of cigarette smoking, it is possible that there is a disparity between subjective measures of cigarette craving and actual smoking choice. That is, participants may crave cigarettes in a dose-dependent manner, but they may not necessarily choose to smoke during these heightened levels of craving. However, evidence from previous research (Doherty et al., 1995; Shiffman et al., 1997) supports the theory that participant's smoking urge during an early nicotine abstinence phase is strongly correlated with subsequent choice smoking behavior. Another limitation is that our sample was primarily comprised of male participants (69% of the sample) who were not heavy, nicotine-dependent smokers. With two recent exceptions (Burton and Tiffany 1997; Sayette et al., 2005), most studies have been limited to a predominantly male sample, and further research is needed to determine whether there are sex differences in alcohol-induced smoking urge. Studies are also needed to determine whether the results may extend to heavier smokers. Finally, the sample size was modest. Although alcohol dose-dependent increases in factor 1 urge to smoke seem to be robust, with large effect sizes ($d = 0.53$, high dose versus low dose; $d = 0.99$, high dose versus placebo), the small sample precluded more detailed analyses on factors related to sensitivity to alcohol-induced smoking urge (e.g., expanded between-subjects analyses of the influences of smoking or drinking levels).

In sum, regular social drinker-smokers showed alcohol

dose-dependent increases in urge to smoke. The experimental paradigm involving direct "real-time" assessment of smoking urge during predrinking baseline and throughout the BAC may provide important clues on the precipitants and mechanisms underlying the combined use of these two substances. The results support a direct pharmacological or priming mechanism for alcohol-related increases in cigarette craving, which is augmented at higher alcohol doses and emerges during the early rising portion of the BAC curve. Continued examination of these determinants may increase our understanding of vulnerability to comorbid alcohol and tobacco dependencies, as well as guide treatment providers with information to advise their patients attempting to abstain or reduce consumption of alcohol, cigarettes, or both substances concurrently.

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