

# The Effects of Alcohol on Cigarette Craving in Heavy Smokers and Tobacco Chippers

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The authors examined the effects of alcohol consumption on cigarette craving in heavy smokers and tobacco chippers ( $n = 138$ ) who were instructed not to smoke for 12 hr. Participants were exposed to both smoking cues (a lit cigarette) and control cues. Half received a moderate dose of alcohol, adjusted for gender, and half received a placebo. Results indicated that alcohol consumption produced an increase in urge-to-smoke ratings before smoking cue exposure. Moreover, during cue exposure, alcohol consumption produced a sharper increase in urge ratings than did the placebo. In addition, during smoking cue exposure, alcohol increased the likelihood of displaying facial expressions associated with positive affect. These findings suggest that alcohol consumption influences both the magnitude and the emotional valence of cigarette cravings.

*Keywords:* cigarette craving, alcohol intoxication, tobacco chippers

Both clinical and experimental research point to an association between alcohol consumption and smoking (see Fertig & Allen, 1995). Given the observed relation between craving and smoking relapse (e.g., Killen & Fortmann, 1997; Shiffman et al., 1997), there has been increased interest in determining the effects of alcohol on cigarette craving, or urge (Tiffany, 1990). Only a few studies have examined the link between alcohol use and cigarette craving (Glautier, Clements, White, Taylor, & Stolerman, 1996; Mintz, Boyd, Rose, Charuvastra, & Jarvik, 1985; Nil, Buzzi, & Bättig, 1984). These studies have found enhanced smoking motivation during intoxication, but methodological concerns (e.g., lack of smoking cues) limited their implications (see Burton & Tiffany, 1997). Recently, Burton and Tiffany (1997) published a smoking cue exposure study testing the effects of alcohol on craving. During cue exposure, alcohol produced a generalized increase in reported urge to smoke (Burton & Tiffany, 1997).

We aimed to replicate this finding and extend the research in several different ways. In addition to testing the effects of alcohol consumption—as well as just the belief that one has consumed alcohol—on cigarette cravings, we analyzed facial expressions displayed during cue exposure to investigate the effects of alcohol

on the affective valence of cigarette urges. To examine the importance of nicotine dependence in understanding the effects of alcohol consumption on cigarette urge, we recruited heavy smokers and light smoking tobacco chippers.

## Facial Expression

Little is known about how emotion influences the link between alcohol consumption and cigarette craving. This partly is due to diverse opinions about the general role of emotion (or affect) in craving. For instance, Tiffany (1992) posited that negative affect (e.g., frustration) is the emotional state linked to craving, whereas others have suggested that cravings may be experienced both positively and negatively (Baker, Morse, & Sherman, 1987). Tests of the association between craving and affect are hampered by undue reliance on self-report instruments. Although these measures provide valuable information not available by means of other response domains, they are vulnerable to a range of biases and inaccuracies (see Sayette et al., 2000).

We used Ekman and Friesen's (1978) Facial Action Coding System (FACS) to examine the affective tone of cravings. FACS is an anatomically based system that permits coding of action units (AUs) that can be combined to describe all possible visible facial movements. Although facial expressions serve multiple purposes, it is clear that many are related to subjective affective experience, with specific AUs differentially reflecting emotional experience (Ekman & Rosenberg, 2005). Although labor intensive, FACS offers an unobtrusive, comprehensive, and reliable measure of facial movement (Sayette, Cohn, Wertz, Perrott, & Parrott, 2001). The terms *positive AUs* and *negative AUs* are used herein to refer to AUs related to affective valence.

Data obtained using FACS have identified perceived smoking opportunity as a factor affecting the emotional tone of craving (e.g., Sayette, Wertz, et al., 2003). Alcohol consumption may be another factor to influence the emotional tone of cigarette cravings. There is evidence that alcohol consumption influences emotional experience. While blood alcohol concentrations (BACs) are rising,

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for example, individuals tend to report experiencing positive affect (e.g., Lukas & Mendelson, 1988; Martin, Earleywine, Musty, Perrine, & Swift, 1993). In this study, we used FACS to test whether alcohol consumption, measured on the rising limb of the BAC curve, might affect the emotional valence of cigarette craving.

### Tobacco Chippers

According to the *Diagnostic and Statistical Manual of Mental Disorders*, craving is "likely to be experienced by most if not all individuals with substance dependence" (American Psychiatric Association, 1994, p. 176). Nondependent users, known as *tobacco chippers* (TCs; Shiffman, Paty, Kassel, Gnys, & Zettler-Siegal, 1994), however, can experience craving without withdrawal. On average, TCs smoke several days a week and approximately four cigarettes on any given day (Shiffman et al., 1994). They smoke normally, absorb normal amounts of nicotine, eliminate nicotine normally, and develop nicotine tolerance (Shiffman, Fischer, Zettler-Siegal, & Benowitz, 1990), yet do not show signs of dependence (Shiffman et al., 1994). The most frequent environmental cue for smoking among TCs is alcohol consumption (Shiffman et al., 1994). Thus, it is unclear whether alcohol consumption should differentially enhance smoking craving in heavy smokers (HSs) and TCs. To our knowledge, this study is the first to contrast the effects of alcohol on smoking cravings in HSs and TCs.

We used self-reported urge and an observational measure of facial expression to test the effects of alcohol consumption on cigarette craving. Heavy smokers and TCs, who drank either alcohol or a placebo, were exposed to smoking cues following a 12-hr period of smoking abstinence. We predicted that merely drinking what one believed was an alcoholic beverage would increase cigarette cravings in nicotine-deprived smokers. In addition, we examined whether actual alcohol consumption would augment both generalized and smoking-cue-elicited craving responses. These effects were predicted to appear in both HSs and TCs, although it was not clear whether this drink effect would differ for the two groups. We also used FACS to analyze expressions displayed during cue exposure to examine the effects of alcohol consumption on the affective valence of craving responses. We hypothesized that, during smoking-cue-exposure, participants drinking alcohol (while blood alcohol levels were rising) would be more likely to evince facial expressions related to positive affect and less likely to display expressions associated with negative affect than would participants drinking a placebo.

### Method

#### Participants

Smokers of ages 21 through 35 ( $n = 138$ ) were recruited through newspaper and radio advertisements for a study paying \$80. The sample was 82% Caucasian, 11% African American, and 7% Hispanic or Asian American. TCs ( $n = 68$ ) had to report smoking at least 2 days per week. On smoking days, they had to average 1 to 5 cigarettes per day. Heavy smokers ( $n = 70$ ) had to average between 20 and 40 cigarettes per day. Both TCs and HSs had to report smoking at these rates for at least 24 continuous months (Shiffman et al., 1994). Gender was balanced across the groups (33 female TCs, 36 female HSs, 35 male TCs, 34 male HSs). Participants were excluded if they reported a medical condition that ethi-

cally contraindicated nicotine or alcohol (e.g., alcohol abuse) or if they were illiterate. All participants provided informed consent. Heavy smokers and TCs had to have initial carbon monoxide (CO) levels (following 12 hr of smoking abstinence) that did not exceed 20 parts per million and 15 parts per million, respectively.

#### Experimental Design

The study had a mixed factorial design, with 12-hr smoking-abstinent HSs and TCs randomly assigned to alcohol or placebo conditions. Participants were informed that they could not smoke during the study. All participants were exposed to control and smoking cues. Participants' own cigarettes served as their smoking cue to increase magnitude of reactivity. A small roll of electrical tape served as the control cue, as it was of similar size and weight to a cigarette yet unlikely to be associated with smoking cues (Sayette & Hufford, 1994; Sayette, Wertz, et al., 2003). Urge ratings and facial coding were assessed during both smoking cue and control cue exposure. (Several other measures were administered only after smoking cue exposure and are not addressed here. A measure of cognitive resource allocation was administered, but because of space constraints this also is not addressed.)

As in prior research (e.g., Sayette, Martin, Hull, Wertz, & Perrott, 2003), the order of cues was fixed, with control cues preceding smoking cues. We did not counterbalance because urge ratings following drug cues tend to remain high, making it hard to interpret effects of subsequent control exposure (Sayette, Martin et al., 2003). That is, once nicotine-deprived smokers are exposed to smoking cues, it is unlikely that their urges will drop back to pre-cue exposure levels without their actually smoking. Thus, the subsequent control cue exposure assessment occurs while they are still in a fairly high state of craving. (Studies that have elicited a strong urge to smoke during smoking cue exposure and have counterbalanced cues often have not tested for [or at least reported] order effects. Those that have provided such data seem to have observed order effects of the sort noted above [e.g., Hutchison, Niaura, & Swift, 1999; Rickard-Figueroa & Zeichner, 1985].)

#### Predrink Assessment

To assess individual differences that might affect craving, we obtained data on age, gender, ethnicity, marital status, and income. Smoking history and patterns also were assessed. Drinking patterns were assessed using a quantity/frequency/variability index, which recorded how often participants consumed various quantities of alcoholic beverages. The 20-item positive and negative subscales of the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) were administered to confirm that mood was similar in the two drink conditions.

#### Craving-Related Measures

**Reported urge to smoke.** Reported urge to smoke was assessed with a rating scale that ranged from 0 (*absolutely no urge to smoke at all*) to 100 (*strongest urge to smoke I've ever experienced*; Sayette, Martin, Wertz, Shiffman, & Perrott, 2001). A measure of magnitude estimation was also included (see Sayette, Martin, Wertz, et al., 2001). The magnitude-estimation data provided findings similar to the traditional rating scale, so only the rating scale data are presented.

**Facial coding.** Facial expressions were coded by an FACS-certified coder during several periods throughout the experiment (see below) using exhaustive FACS. Exhaustive FACS requires coding all visible movements of the face using a frame-by-frame analysis of videotape. We coded the same intervals used in our most recent FACS study (Sayette, Wertz, et al., 2003). First, AUs were coded during two intervals associated with control cue exposure: (a) the initial 5 s during which participants viewed the control cue and (b) the initial 5 s during which they touched and held the

control cue. These two periods allowed an assessment of participants' responses to a novel cue that was not smoking related. FACS also was coded during three fixed-order intervals related to smoking cue exposure: (a) the initial 5 s that participants viewed smoking cues (*Look*), (b) the initial 5 s that they touched the cigarette (*Touch*), and (c) the first 10 s that they held the lit cigarette (*Hold*).

### Procedure

**Screening and instructions.** Participants responding to advertisements underwent a telephone interview aimed at excluding individuals who did not meet the selection criteria. Eligible smokers were asked to attend a 5- to 7-hr laboratory session. All participants were asked to refrain from smoking for at least 12 hr, to avoid eating for at least 4 hr, and to refrain from drinking alcohol or using recreational drugs for 24 hr. They were told that breath samples would be obtained to ensure abstinence. Participants also were told to bring a pack of their preferred brand of cigarettes.

**Laboratory setup.** Participants underwent cue exposure manipulations while seated in a comfortable chair behind a desk. On the desk were an intercom and a mouse button; the latter was used in the cognitive resource allocation task. Facing the desk was a wall-mounted video camera. Participants were told that the camera and intercom facilitated communication and helped the investigator determine that instructions were understood throughout the study.

**Baseline assessment.** Experimental sessions began between 11:30 a.m. and 3:00 p.m. (Drinking began about an hour into the session.) On arrival, participants provided written informed consent. To check compliance with abstinence instructions, they reported the last time they smoked and drank, and CO and BAC readings were recorded. (During each BAC assessment throughout the experiment, they also were asked to rate their level of subjective intoxication [SI] on a scale that ranged from 0 [*not at all intoxicated*] to 100 [*the most intoxicated that I have ever been*].) Participants presented their pack of cigarettes and lighter to the experimenter.

Participants next completed the urge rating scale (Urge 1) followed by a demographics form, the PANAS, and the quantity/frequency/variability index (Calahan, Cisin, & Crossley, 1969). After these forms were completed, Urge 2 was recorded.

**Drink administration.** The drink procedure was similar to that used in prior studies (e.g., Sayette, Martin, Perrott, Wertz, & Hufford, 2001). A researcher brought a tray containing chilled bottles of vodka and cranberry juice cocktail into the participant's room. The alcohol drink was 1 part vodka and 3.5 parts juice. In the alcohol group, the vodka bottle had 100-proof vodka; in the placebo group, the vodka bottle contained flattened tonic water. In the latter group, the glass was smeared with vodka to enhance credibility of the placebo. Total beverage was isovolemic in the two drink conditions. Previous studies revealed that this procedure allowed successful execution of the placebo manipulation (Sayette, Martin, Perrott, et al., 2001), the aim of which was to lead participants to believe they had drunk alcohol (Martin & Sayette, 1993).

Beginning at Time 0, alcohol participants received one third of a gender-adjusted dose of alcohol (men: 0.82 g/kg, women: 0.74 g/kg) to drink evenly over 10 min. After 10 min, Urge 3 was recorded (to examine effects of alcohol cues—including drinking what appears to be alcohol—prior to intoxication on cigarette urge). At 10 min and 20 min, they received the middle and final thirds of the beverage, respectively, and were asked to drink it evenly over the 10-min intervals. Immediately after the final third was finished (30 min), they rinsed their mouths with water.

**Cue exposure.** Cue exposure was conducted using methods described in Sayette, Martin, Wertz, et al. (2001). During control cue exposure, a tray holding an inverted plastic bowl was placed on the desk. Participants responded to two auditory tones (related to the cognitive resource allocation task), then lifted the bowl, which revealed a roll of tape. After picking up the tape, they responded to two more tones and then rated their urge to smoke (Urge 4, about 33 min). Two minutes later, the experimenter replaced the tray and bowl with a second tray and bowl. Following two

more tones, participants provided Urge 5 (about 36 min), then lifted the bowl to reveal their cigarettes, an ashtray, and a lighter. They were asked to remove a cigarette from the pack and light it without putting it in their mouths. They then held the cigarette and looked at it while responding to two more tones. Next, they rated their urge to smoke (Urge 6; about 37 min) and extinguished the cigarette. They completed the two forms assessing smoking expectancies (not discussed here), followed by BAC No. 2 (about 60 min). After a behavioral choice task (not discussed here), all participants were permitted to smoke.

Postsmoking BAC (No. 3) was recorded approximately 77 min following the start of the drink. Blood alcohol concentration levels were recorded at about 10-min intervals until it was clear that they were dropping (i.e., three consecutively decreasing BACs). Participants then ate lunch and were allowed to read, work, or listen to music. When BACs fell below .04%, alcohol participants completed a postexperimental questionnaire asking them to describe the study's purpose and to estimate the amount of vodka (in ounces) they had consumed and their level of intoxication (INT; on a scale that ranged from 0 [*not intoxicated at all*] to 14 [*the most intoxicated I have ever been*]) during the experiment (Sayette & Wilson, 1991). (Placebo participants completed this form after BAC No. 3.) Participants next were debriefed, and when BACs dropped below .025% they were paid and permitted to leave.

## Results

### Baseline and Demographic Comparisons

Smoking Group  $\times$  Drink analyses revealed no differences on income, marital status, age, ethnic makeup, or drinking practices. Heavy smokers received fewer years of formal education, and reported fewer previous quit attempts, than did TCs,  $F_s(1, 134) > 4.6$ ,  $ps < .04$ . Relevant participant characteristics by smoking group are presented in Table 1.

Table 1  
Participant Characteristics by Smoking Group

Characteristic	Tobacco chippers		Heavy smokers	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Age	23.1	3.3	24.0	3.7
Years formal education	14.8	1.1	13.8	1.7*
Years smoking	5.1	3.0	8.0	4.3**
Cigarettes /smoking day	3.8	1.4	23.9	4.9**
Quit	6.7	2.5	5.8	2.7*
CO	5.8	3.4	13.0	4.8**
Drinking patterns				
QFV—often	5.7	0.9	5.6	1.1
QFV—one or two	2.3	1.5	2.5	1.5
QFV—three or four	2.2	1.1	2.3	1.2
QFV—five or six	2.8	1.3	2.6	1.2

*Note.* Quit = number of previous quit attempts; CO = carbon monoxide level at outset of study; QFV = quantity, frequency, variability; QFV—often = how often participants consumed alcohol (rated on an 11-point scale ranging from 1 = *three or more times/day* to 11 = *never*); QFV—one or two = how often participants consumed one or two drinks on a drinking day (rated on a 5-point ranging from 1 = *nearly every time* to 5 = *never*); QFV—three or four = how often participants consumed three or four drinks on a drinking day (rated on a 5-point ranging from 1 = *nearly every time* to 5 = *never*); QFV—five or six = how often participants consumed five or six drinks on a drinking day (rated on a 5-point scale ranging from 1 = *nearly every time* to 5 = *never*).

\*  $p < .05$ . \*\*  $p < .0001$ .

As shown in Table 1, TCs reported smoking fewer cigarettes per day and fewer years of smoking than did HSs,  $F_s(1, 134) > 22$ ,  $p_s < .0001$ . Heavy smokers also recorded higher CO levels than did TCs,  $F(1, 133) = 121$ ,  $p < .0001$ . On the PANAS, HSs reported feeling less positive ( $M = 22.7$ ,  $SD = 7.0$ ) than TCs ( $M = 25.2$ ,  $SD = 7.4$ ),  $F(1, 131) = 4.0$ ,  $p < .05$ . Heavy smokers also reported feeling more negative ( $M = 13.7$ ,  $SD = 4.5$ ) than TCs ( $M = 11.7$ ,  $SD = 2.5$ ),  $F(1, 134) = 10.0$ ,  $p < .01$ . Participants in the two drink conditions did not differ on any of these variables for either TCs or HSs.

### BACs and the Effectiveness of Placebo Manipulation

Alcohol participants had mean BACs of .071% ( $SD = .013$ ) after smoking cue exposure measures (Time 60 min) and .073% ( $SD = .012$ ) after smoking (Time 77 min). Heavy smokers and TCs recorded similar BACs throughout the experiment.

Three measures examined the effectiveness of placebo manipulations. Drink condition did not significantly affect the number of ounces of vodka that participants reported consuming on the postexperimental questionnaire. All participants reported drinking at least some vodka. The remaining two measures (SI and INT) both revealed greater effects for alcohol than placebo conditions ( $F_s > 83$ ,  $p_s < .0001$ ). Alcohol participants reported a higher level of intoxication on the postexperimental INT item ( $M = 5.7$ ,  $SD = 2.3$ ) than did placebo participants ( $M = 2.1$ ,  $SD = 1.6$ ). Similarly, alcohol participants reported higher levels of intoxication on the postdrink SI measure ( $M = 36.3$ ,  $SD = 20.3$ ) than did those who drank the placebo ( $M = 11.0$ ,  $SD = 11.2$ ). There also was a main effect of group,  $F(1, 128) = 4.1$ ,  $p < .05$ , on this item, with TCs reporting more intoxication ( $M = 26.54$ ,  $SD = 22.6$ ) than HSs ( $M = 20.86$ ,  $SD = 18.4$ ). In sum, although participants who drank the placebo felt less intoxicated than those who drank alcohol, the placebo led them to believe that they had drunk similar amounts of vodka and were somewhat intoxicated, thus meeting the modest goals of an alcohol placebo manipulation (see Martin & Sayette, 1993).

### Effects of Beverage Consumption on Craving-Related Responses

*Urge rating scale.* Ratings were recorded at six points. To see whether urge ratings changed during the experiment, we performed an overall 2 (group)  $\times$  2 (drink)  $\times$  2 (gender)  $\times$  6 (urge) repeated measures analysis of variance (ANOVA). There was a main effect of group,  $F(1, 127) = 46.42$ ,  $p < .0001$ , and of time,  $F(5, 635) = 145.28$ ,  $p < .0001$ . There also was a significant Drink  $\times$  Time interaction,  $F(5, 635) = 7.72$ ,  $p < .0001$ , showing that drink type differentially affected urge ratings over the course of the experiment. There were neither main effects nor interactions involving gender. Mean urge ratings throughout the study are presented in Table 2.

We conducted a set of planned repeated measures contrasts involving drink and time that tested several hypotheses. If alcohol consumption is a cue for smoking, then consumption of what one believes is an alcoholic beverage ought to increase cigarette craving. That is, holding and drinking what one believes is alcohol should enhance cigarette cravings. Because placebo and alcohol beverages are likely to be perceived similarly during the first 10 min of drinking, urge ratings should increase similarly from baseline to Urge 3 for both drink conditions. We tested this hypothesis using a 2 (drink)  $\times$  2 (time) repeated measures ANOVA with Urge 1 and Urge 3 as the repeated measure. A main effect of time emerged,  $F(1, 134) = 41.11$ ,  $p < .0001$ . The Drink  $\times$  Time interaction did not approach significance ( $F < 0.1$ ). These data suggest that a belief that one has begun to consume alcohol increases smoking urge ratings.

We next hypothesized that, as the alcohol begins to be absorbed and intoxication levels start to differ between alcohol and placebo groups, urge ratings should increase more for participants consuming alcohol than for those drinking a placebo. This drink effect was expected to begin to show up at Urge 4 (when drinking was completed) and continue through Urge 6. We tested this hypothesis using a series of three 2 (drink)  $\times$  2 (time) repeated measures ANOVAs with Urges 1 and 4, Urges 1 and 5, and Urges 1 and 6 serving as the repeated measures variables. For Urges 1 and 4, there was a main effect of time,  $F(1, 136) = 41.11$ ,  $p < .0001$ , and

Table 2  
Mean Self-Reported Urge Ratings Among Tobacco Chippers and Heavy Smokers in the Alcohol and Placebo Conditions

Rating	Tobacco chippers				Heavy smokers			
	Alcohol		Placebo		Alcohol		Placebo	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Urge rating scale (Time 1)	14.6	20.3 <sub>a</sub>	20.7	16.1 <sub>a</sub>	41.3	27.7 <sub>b</sub>	47.1	24.4 <sub>b</sub>
Urge rating scale (Time 2)	15.4	20.5 <sub>a</sub>	20.4	16.0 <sub>a</sub>	43.6	26.0 <sub>b</sub>	47.3	23.7 <sub>b</sub>
Urge rating scale (Time 3)	20.4	23.3 <sub>a</sub>	28.3	19.7 <sub>a</sub>	47.5	30.0 <sub>b</sub>	53.7	23.9 <sub>b</sub>
Urge rating scale (Time 4)	36.0	25.8 <sub>a</sub>	32.9	21.4 <sub>a</sub>	59.4	27.3 <sub>b</sub>	59.4	23.8 <sub>b</sub>
Urge rating scale (Time 5)	36.9	26.6 <sub>a</sub>	33.6	22.0 <sub>a</sub>	60.9	24.7 <sub>b</sub>	59.9	24.4 <sub>b</sub>
Urge rating scale (Time 6)	53.4	29.9 <sub>a</sub>	44.2	27.5 <sub>a</sub>	77.0	23.9 <sub>b</sub>	72.0	24.3 <sub>b</sub>
Change in urge (Time 6 minus Time 1)	38.9	25.6 <sub>a</sub>	23.6	21.3 <sub>b</sub>	35.7	23.9 <sub>a</sub>	24.9	20.4 <sub>b</sub>

Note. Time 1 = baseline; Time 2 = postinitial questionnaires; Time 3 = 10 min after beginning beverage consumption; Time 4 = control cue exposure; Time 5 = presmoking cue baseline; Time 6 = smoking cue exposure. Values in each row with nonoverlapping subscripts are significantly different from one another ( $p < .05$ ).

a Time  $\times$  Drink interaction,  $F(1, 136) = 6.77, p < .02$ . As seen in Table 2, participants who consumed alcohol showed a sharper rise in urge from Time 1 to Time 4 than did those who drank the placebo. When we examined Urges 1 and 5, we found the same pattern: There was a main effect of time,  $F(1, 136) = 252.40, p < .0001$ , and a Time  $\times$  Drink interaction,  $F(1, 136) = 11.32, p < .01$ . Likewise, when we examined Urges 1 and 6, we observed that there was a main effect of time,  $F(1, 136) = 133.16, p < .0001$ , and a Time  $\times$  Drink interaction,  $F(1, 136) = 7.70, p < .01$ .

It has been suggested that drug-cue-specific urge is best assessed by contrasting urge during drug cue exposure with urge during a control cue exposure (Carter & Tiffany, 1999). Accordingly, we conducted an analysis of covariance with drink as the independent variable, Urge 6 as the dependent variable, and Urge 4 as the covariate. There was a main effect of drink,  $F(1, 135) = 4.24, p < .05$ , with participants who drank alcohol reporting stronger urges during smoking cue exposure, after adjusting for control cue exposure urges, than did those who drank the placebo. These data suggest that alcohol consumption selectively increased urge ratings in response to a smoking cue. (As in our prior work [Sayette, Wertz, et al., 2003], there were not enough instances of discrete emotions to use more specific emotion categories.)

**Facial expression.** Specific AUs and AU combinations were classified as positive AUs or negative AUs based on a review of the FACS literature (Sayette & Parrott, 1999). Reliability was tested using comparison coding by a second FACS-certified coder of a random sample of 58 coding periods in the study. These periods included samples for the different time intervals noted above. Kappa coefficients showed that positive AUs (.92) and negative AUs (.75) were coded reliably. Because FACS data were dichotomous (presence or absence of particular AUs), we analyzed the data using logistic regression (PROC CAT MOD; SAS Institute Inc., 2000).

**Positive AUs during control exposure.** There is no true baseline period for coding facial expression (Sayette, Wertz, et al., 2003). We coded facial reactivity during control cue exposure for the first 5 s that participants looked at the tape and the first 5 s that they touched and held the tape. In general, it was uncommon for participants to express positive AUs during control cue exposure. A repeated measures analysis with drink, group, and gender as between-subjects variables, and control cue exposure coding interval as a repeated measures variable, was conducted. The lone effect was a Drink  $\times$  Time interaction,  $\chi^2(1, N = 138) = 9.0, p < .01$ . Intoxicated smokers were more likely (19%) than placebo participants (8%) to manifest positive AUs during the first coding interval,  $\chi^2(1, N = 138) = 5.2, p < .03$ . This beverage effect was absent during the second control period, in which 10% of participants expressed positive AUs.

**Negative AUs during control exposure.** A repeated measures analysis similar to that used for positive AUs during control exposure (described above) revealed no significant effects.

**Positive AUs during smoking cue exposure.** We conducted a repeated measures analysis with drink, group, and gender as between-subjects variables, and the presence or absence of a positive AU during the three smoking cue coding periods as the repeated measures variable. A significant effect of drink appeared,  $\chi^2(1, N = 138) = 3.9, p < .05$ , indicating that participants who drank alcohol were more likely to express a positive AU during smoking cue exposure than were placebo participants (see Figure

1). A marginally significant effect of gender appeared,  $\chi^2(1, N = 138) = 3.6, p < .06$ , with women being more likely than men to express positive AUs across the three coding intervals. In addition, there was a main effect of time,  $\chi^2(2, N = 138) = 11.8, p < .01$ , indicating that the likelihood of expressing positive AUs declined over time, with 49% of participants displaying positive AUs during Look, 36% during Touch, and 34% during Hold.

**Negative AUs during smoking cue exposure.** We next tested whether the alcohol group was less likely to evince negative AUs than the placebo group. A repeated measures analysis revealed a marginally significant effect of drink,  $\chi^2(1, N = 138) = 3.3, p < .07$ , with intoxicated smokers tending to be less likely than sober smokers to express negative AUs (see Figure 2). There was a marginally significant effect of gender,  $\chi^2(1, N = 138) = 2.8, p < .10$ , with women more likely than men to express a negative AU across the three coding periods. A significant time effect,  $\chi^2(2, N = 138) = 8.6, p < .02$ , showed that the chances of expressing negative AUs increased over time (from 22% during Look, to 25% during Touch, and 35% during Hold).

**Correlations between facial expression and urge rating.** Two correlations were computed to examine potential associations between reported urge and facial expression. The urge rating during smoking cue exposure (Urge 6) was positively correlated with likelihood of evincing a positive AU during the three smoking cue exposure periods,  $r(138) = .22, p < .02$ , but uncorrelated with likelihood of evincing a negative AU ( $r = -.03$ ).

## Discussion

Our major findings are that alcohol consumption increased urge-to-smoke ratings and influenced the emotional valence of cravings. That is, abstinent smokers who drank alcohol experienced cravings that seemed to differ both quantitatively and qualitatively from those who drank a placebo. Quantitatively, smokers who drank alcohol reported stronger urges during smoking cue

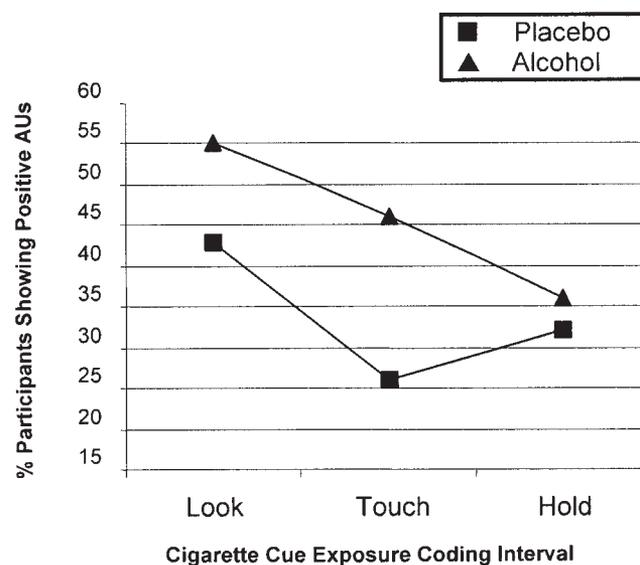


Figure 1. Percentage of alcohol and placebo participants expressing positive-affect-related action units (AUs) during smoking cue exposure.

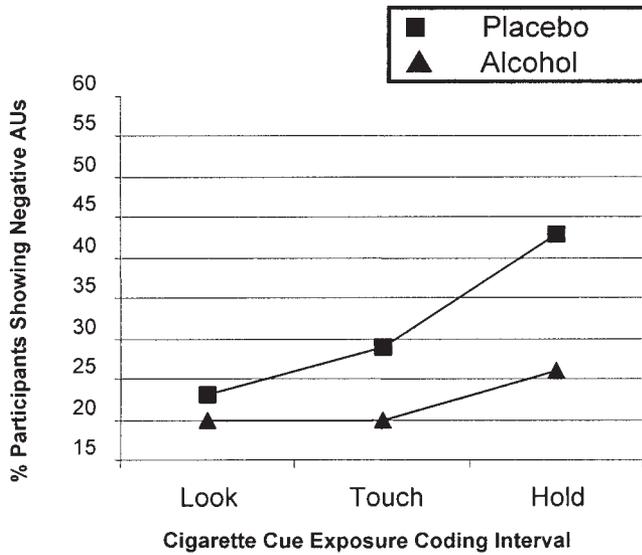


Figure 2. Percentage of alcohol and placebo participants expressing negative-affect-related action units (AUs) during smoking cue exposure.

exposure—and this effect remained when controlling for urges during control cues—and, qualitatively, these participants evinced facial expressions indicative of more positive affect and (marginally) less negative affect than did those who consumed a placebo.

#### Initial Postdrink Effects

Ten minutes into the drink, both the alcohol and placebo groups showed increased urge ratings, which did not differ from each other. These data, paralleling those reported by Burton and Tiffany (1997), suggest that cues associated with drinking, such as the smell of alcohol and the belief that one has consumed it, may trigger an urge to smoke. During this initial third of the drink period, placebo and alcohol consumption conditions were expected to be most similar, as alcohol participants have yet to experience much intoxication (Sayette, Martin, & Perrott, 2001). The finding that both alcohol and placebo consumption initially increased smoking urge is consistent with conditioning models of craving, which suggest that cues associated with smoking (e.g., alcohol) should elicit smoking craving.

#### Subsequent Postdrink Effects

After finishing drinking, when the alcohol and placebo effects began to diverge, participants drinking alcohol began to show a steeper rise in urge ratings than did those drinking the placebo. This suggests that, in addition to conditioning effects elicited by drinking cues, actual alcohol consumption also augments cigarette craving. This may be due to neurobiological priming mechanisms. For instance, both drinking and smoking activate dopaminergic neurotransmission in the ventral tegmental area (Pomerleau, 1995). Drinking alcohol may prime a smoker to crave nicotine (e.g., Baker et al., 1987; Robinson & Berridge, 1993). Of course, a conditioning analysis still could accommodate these data if one assumes that certain interoceptive cues related to intoxication (which are absent with a placebo) cue smoking craving.

#### Smoking Cue Exposure Effects

Alcohol consumption enhanced smoking-cue-elicited urges; that is, after adjusting for urges during control cues, reported urge to smoke during smoking cue exposure was greater for participants who consumed alcohol than for those who consumed a placebo. This differs from the results of Burton and Tiffany (1997), who found that alcohol increased overall urge ratings but not smoking-cue-specific urges. It is worth noting, though, that craving ratings were 20 points higher during *in vivo* smoking cue exposure ( $M = 75$ , on a 100-point scale) than during neutral cue exposure ( $M = 55$ ) in their alcohol condition (see Table 2 in Burton & Tiffany, 1997), which actually is a larger increase in cue-elicited urge than what we found in our alcohol groups. Burton and Tiffany did not find a significant drink effect because the placebo group also rose by 20 points (from 38 to 58). In contrast, our placebo groups showed increases of about 12 points. Thus, the large smoking-cue-specific urge in their placebo group made it very difficult for a significant drink effect to emerge. Consider that, to reveal a drink effect, the mean urge value for intoxicated smokers during smoking cue exposure likely would have needed to exceed 80, which is most unlikely for a group of nonabstinent smokers who are not in withdrawal. Other methodological differences between the two studies (e.g., number of cue exposures) also may have contributed to the discrepant findings.

Our FACS data suggest that alcohol consumption affects the emotional tone of cravings. During smoking cue exposure, smokers were more likely to display positive AUs and marginally less likely to display negative AUs when intoxicated than when sober. In prior research, we and our colleagues have observed that cravings can be associated with either positive or negative AUs, depending on a smoker's perceived opportunity to smoke (e.g., Sayette, Wertz, et al., 2003). The notion that cravings sometimes can be linked to positive affect suggests that drug craving, separate from actual drug effects, can be rewarding, a process described by Loewenstein (1987) as "savoring." If ingesting alcohol causes cravings to become more rewarding—perhaps because alcohol consumption increases the perception that drug use will ensue—then it follows that alcohol consumption may facilitate individuals placing themselves in high-risk craving situations.

Although the FACS data revealed effects of alcohol consumption during smoking cue exposure, it remains to be determined whether alcohol consumption would have a similar effect in response to any stimulus eliciting an emotional response. Regardless of the specificity of this effect, the present study supports the conclusion that smoking cues are an especially important set of stimuli that happen to be sensitive to this effect of alcohol consumption.

#### TCs

During smoking cue exposure, HSs reported stronger urges than did TCs. This effect replicates prior findings with these measures (Sayette, Martin, Wertz, et al., 2001). TCs and HSs evinced similar patterns of AUs. The lack of group differences on the FACS measures is consistent with prior data (Sayette, Wertz, et al., 2003).

Most pertinent to the present study was the lack of Drink  $\times$  Group interactions; that is, alcohol produced similar effects across

measures for both HSs and TCs. It appears that, consistent with data showing alcohol to be an especially potent cue for smoking among TCs (Shiffman et al., 1994), the potentiating effects of alcohol consumption on cigarette cravings are not confined to HSs. In addition, it is interesting to see that our TCs, despite drinking in a similar fashion as the HSs, reported greater intoxication than did HSs, irrespective of drink condition. Future research is indicated to examine more fully potential differences between TCs and HSs in the development of tolerance to drugs other than nicotine.

### Conceptual Implications

Alcohol's potentiation of cue-elicited urge is consistent with several theories. Steele and Josephs's (1990) alcohol myopia model proposes that intoxication restricts processing resources to the most salient cues and that these immediate aspects of experience can disproportionately influence emotion. Consequently, alcohol consumption, through its myopic effects, may enhance the intensity of cravings. This finding also is consistent with predictions derived from an appetitive-motivational model of craving (e.g., Stewart, de Wit, & Eikleboom, 1984), which proposes that over time (through conditioning), drug cues come to provide an incentive, experienced as craving, to receive more of the drug. As Burton and Tiffany (1997) noted, consumption of other drugs of abuse, such as alcohol, should therefore promote activation of this appetitive motivational state. Future studies are needed to determine conditions under which these theories may provide different predictions.

Certain study limitations should be noted. The CO measure lacked the necessary precision to state definitively that all smokers abstained for at least 12 hr. Moreover, because CO levels were not also recorded during nonabstinent states, we were unable to compare participants' abstinent levels to their own baselines. Nevertheless, mean CO levels in this study are consistent with what would be expected on the basis of other studies (e.g., Drobos & Tiffany, 1997). The present study included a moderate dose of alcohol. It is possible that different doses may produce a range of effects on smoking craving. For instance, smokers who smoke in part to counter the sedative effects of alcohol intoxication might best be tested when these sedative effects are most apparent, by using higher doses or assessing urge on the descending limb of the BAC curve. In addition, this study did not include a no-alcohol control group. Inclusion of a no-alcohol control condition would further isolate the effects on smoking craving of believing one has consumed alcohol (see Martin & Sayette, 1993). Comparisons between a placebo and a no-alcohol control permits a test of the effects of beverage expectancy on smoking craving in the absence of ethanol.

We cannot rule out the possibility that enhanced effects of alcohol on smoking-cue-elicited urges were due to the study design, in which smoking cues always followed control cues. If the effects of alcohol were more pronounced during the second than the first cue, then greater urges would be expected during the second (in this case, the smoking) cue exposure. It is unlikely, though, that differences are simply due to this time effect, as the two exposures occurred within 4 min of each other. Consistent with the majority of cue exposure studies (see Carter & Tiffany, 1999) we decided against counterbalancing the order of cues because of our concerns that nicotine-deprived smokers exposed to

smoking cues first would still be experiencing elevated urges during the subsequent control cue exposure conditions (Hutchinson et al., 1999; Rickard-Figueroa & Zeichner, 1985). Future studies might consider using cue as a between-subjects variable.

It is worth noting that drinking alcohol seemed to potentiate increases in urge ratings not only during smoking cues but also during control cues. Although conventional analysis indicates that alcohol consumption enhanced smoking-cue-specific urge (by examining urge during smoking cue exposure after adjusting for urge during control cue exposure), our interpretation is tentative. Given the ubiquity of smoking cues throughout the study (including the consumption of alcohol and urge assessment itself), urge ratings obtained before and during our control cue exposure likely were measuring urge following some degree of smoking cue exposure, especially for nicotine-deprived smokers. As has been found elsewhere, merely informing smokers of the opportunity to smoke affected responding to smoking-related words on a color-naming emotional Stroop task (Wertz & Sayette, 2001).<sup>1</sup> That is, under the right "high-craving" conditions, it may not take a particularly strong or explicit smoking cue to "cue" cravings.

This study provides evidence that drinking alcohol affects both the intensity and emotional valence of smoking craving. It also suggests that alcohol consumption can augment urges when explicit smoking cues are present or absent. To better understand the effects of alcohol on smoking craving, research is needed that includes a range of stimuli (see Burton & Tiffany, 1997) and alternative measures of craving-related processes. In addition to replicating these findings, studies are needed to test competing theoretical explanations and to identify conditions under which different models of drinking and smoking operate.

<sup>1</sup> We thank Timothy Baker for this observation.

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