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Risk Factors for Treatment Failure in Smokers: Relationship to Alcohol Use and to Lifetime History of an Alcohol Use Disorder

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Abstract

Little is known about the impact of alcohol involvement on smoking cessation relapse or possible mechanisms for these associations. We addressed these issues using data from a randomized clinical trial of 2 types of framed messages (gain vs. loss) in conjunction with open label sustained-release (SR) bupropion (Toll et al., 2007) ($N = 249$). Participants were categorized according to whether or not they were diagnosed with a lifetime alcohol use disorder (AUD; i.e., current or past alcohol abuse or past alcohol dependence) and according to 3 levels of alcohol use: abstinence, moderate or hazardous use. Alcohol use categories were established for drinking at baseline, during the 6-week treatment period and through 12 weeks post-quit. There were few significant differences by baseline alcohol use level or AUD history for a series of predictors of smoking cessation failure (e.g., depressive symptoms). During treatment and follow-up, the probability of any smoking on heavy drinking days was significantly higher than the probability of smoking on moderate drinking or abstinent days. AUD history did not predict smoking cessation relapse in any analysis, nor were any alcohol use $\times$ AUD history interactions significant. Moderate alcohol users and to a lesser extent, abstainers from alcohol at baseline were less likely than hazardous drinkers to have relapsed at 12 weeks post-quit. Based on these findings, it appears that risk of any smoking and of relapse was associated primarily with heavy drinking days and a hazardous pattern of use respectively, rather than with moderate drinking.

Keywords

smoking cessation; alcohol use; hazardous alcohol use; alcohol use disorders; relapse; outcome predictors

Introduction

Smoking is a major public health concern. An estimated 440,000 Americans die yearly from smoking-related conditions, and the overall economic toll associated with smoking is over \$157 billion per year in the United States (Carmona, 2004). Unfortunately, most smokers fail during a given quit attempt (Piasecki, Fiore, McCarthy, & Baker, 2002), and those who eventually succeed typically make several unsuccessful attempts first (Fiore et al., 2000). Thus,

it is important to learn which types of smokers may be at particular risk of treatment failure and to understand the factors underlying their elevated risk so that interventions can be designed to aid their efforts.

Recently, attention has been paid to the impact of a smoker's history of alcohol problems on likelihood of smoking cessation (e.g., Hughes & Kalman, 2006). Some evidence has linked a history of alcohol problems to established predictors of smoking cessation failure such as severe nicotine dependence (e.g., Novy, Hughes, & Callas, 2001) and depressive symptoms (Cargill, Emmons, Kahler, & Brown, 2001). Less attention has been paid to the impact of current alcohol use on smoking cessation and to associations between current alcohol use and traditional predictors of smoking cessation relapse.

Smoking and a history of alcohol problems

Research on the relationship between alcohol problems and smoking cessation relapse has been motivated by strong evidence of co-morbidity between smoking and Alcohol Use Disorders (AUDs; i.e., meeting diagnostic criteria for either alcohol abuse or dependence). Among those with nicotine dependence, the 12-month co-morbidity of an AUD is 22.8%, compared with 8.5% in the general population, and among those with an AUD, the 12-month co-morbidity of nicotine dependence is 34.5%, compared with 12.8% in the general population (Grant, Hasin, Chou, Stinson, & Dawson, 2004).

In a large, nationally representative sample in the U.S., the odds of lifetime smoking cessation were lower among smokers with a lifetime history of an AUD, although those with an AUD history who were currently abstinent had cessation rates similar to those in a comparison group with no history of any mental illness (Lasser, Boyd, Woolhandler, Himmelstein, McCormick, & Bor, 2000). Among those participating in smoking cessation trials, smokers with a *past* history of alcohol problems do not appear to be at a considerable disadvantage (Aubin, Lebagry, Berlin, Bidaut-Mazel, Chemali-Hudry, Lagrue, 2004; Croghan et al., 2003; Hayford et al., 1999; Hughes et al., 2003; Humfleet, Munoz, Sees, Reus, & Hall, 1999; Hurt et al., 1995; Murray, Istvan, Voelker, Rigdon, & Wallace, 1995; Simon, Duncan, Carmody, & Hudes, 2004), with some exceptions (Hughes, 1993; Hays et al., 1999). It has been proposed that smokers with a past history of alcohol problems may benefit by applying skills they used in stopping or reducing their drinking to quitting smoking (Hughes & Kalman, 2006).

Smoking and alcohol use

Epidemiological and laboratory findings support the notion that alcohol use and cigarette smoking are closely associated. Findings from the National Epidemiologic Study of Alcohol and Related Conditions (NESARC) have shown that alcohol use, even at light-to-moderate levels, is associated with an increased likelihood of daily tobacco use and nicotine dependence when compared with abstinence from alcohol (Falk, Yi, & Hiller-Sturmhofel, 2006). In addition, associations have been found specifically between current smoking and heavier alcohol consumption, in the NESARC data (McKee, Falba, O'Malley, Sindelar, & O'Connor, 2007) and in a survey of medical and dental patients (Kranzler et al., 2002). These findings are paralleled by human laboratory research suggesting that alcohol consumption leads to increased smoking behavior (e.g., Glautier, Clements, White, Taylor, & Stolerman, 1996) and decreased ability to resist smoking (McKee, Krishnan-Sarin, Shi, Mase, & O'Malley, 2006) in smokers who drink regularly but are not alcohol dependent. While epidemiologic and laboratory findings have offered consistent findings linking alcohol use with smoking, findings in clinical samples have been less consistent. In both a sample of hospitalized smokers (Cargill et al., 2001) and a sample of clinical trial participants assessed at baseline (Humfleet et al., 1999), null findings were reported for associations of alcohol use with cigarettes smoked per day and nicotine dependence.

While the impact of current drinking on likelihood of smoking cessation has received scant attention, particularly in the clinical trials literature, most findings have supported the conclusion that alcohol use decreases the likelihood of smoking cessation. This has been the case in both epidemiologic/survey (Agudo et al., 2004; Dawson, 2000; Zimmerman, Warheit, Ulbrich, & Auth, 1990) and in clinical trial research (Humfleet et al., 1999; McClure et al., 2002; Murray et al., 1995; but see Hughes & Oliveto, 1993 for null results). Alcohol use has also been cited frequently as a precipitant of smoking relapse (Baer & Lichtenstein, 1988; Shiffman, 1986). Of note, an increased risk of relapse to smoking with alcohol use applies even after a long period of abstinence from cigarettes (Krall, Garvey, & Garcia, 2002).

An unanswered question is whether any alcohol use increases likelihood of smoking cessation relapse, or whether risk is associated primarily with heavy or hazardous alcohol use. Humfleet and colleagues (1999) reported that alcohol use at baseline and during treatment predicted smoking cessation failure at all assessment points from Week 24 through Week 64. This study made use of dichotomous use/non-use variables, thus heavier alcohol users were not distinguished from moderate users. Studies making this distinction have found that heavier use is most strongly associated with a decreased likelihood of smoking cessation. This has been true of both epidemiologic/survey (Agudo et al., 2004; Dawson, 2000; Zimmerman et al., 1990) and clinical trial findings (McClure et al., 2002; Murray et al., 1995), although Hughes and Oliveto (1993) reported that heavier drinkers in a small clinical trial sample were at no disadvantage in smoking cessation (though mean alcohol use was low in their sample).

Theoretical models regarding associations between alcohol and smoking

A number of theoretical models have been put forth to explain the strong associations between smoking and drinking behavior and for co-morbidity between nicotine and alcohol dependence. Theories regarding concurrent smoking and drinking typically relate to the effect of one substance priming use of the other substance. Rohsenow and colleagues (1997) outlined mechanisms for cross-substance priming including neurological processes, associative learning and cognitive or semantic associations between the substances. After alcohol and tobacco have been used together a number of times, cues for one substance can come to prime use of the other substance through one or more of these mechanisms. Thus, the experience of alcohol-related cues could lead to smoking relapse for co-users of alcohol and cigarettes (Tiffany, 1990).

Most models designed to explain co-morbidity between alcohol and nicotine dependence emphasize shared vulnerability factors common to both addictions and/or reciprocal influence between the two substances (Little, 2000; Rohsenow et al., 1997; Sher, Gotham, Erickson, & Wood, 1996). Examples of shared risk factors include a positive family history/genetic background (Grucza & Bierut, 2006) and psychological factors such as depressive symptoms (Cargill et al., 2001), which have also been found to predict smoking cessation failure (e.g., Carey, Kaltra, Carey, Halperin, & Richards, 1993; Ockene et al., 2000; Rohde, Kalher, Lewinshohn, & Brown, 2004). The extent to which shared vulnerability applies to other predictors of smoking cessation failure such as an early age of initiation (e.g., Khuder, Dayal, & Mutgi, 1999; Ockene et al.), a sparse prior history of quitting behavior (e.g., Garvey, Kinnunen, & Nordstrom, 2000; Ockene et al.) or low motivation and/or self-efficacy to quit (e.g., Hedeker & Mermelstein, 1996; Ockene et al.) is unclear.

There are several biological mechanisms thought to underlie reciprocal influence, including the stimulant properties of nicotine offsetting the sedative properties of alcohol, cross-tolerance in which one substance increases tolerance to the other and both substances operating on some of the same systems in the brain including the mesolimbic dopamine system and the endogenous opiate system (see Funk, Marinelli, & Le, 2006 and Li, Volkow, Baler, & Elgi, 2007 for recent reviews of biological mechanisms). Reciprocal influences could lead smokers

with histories of alcohol problems or who currently drink heavily to smoke more and be more nicotine dependent. Several studies involving smokers with a history of alcohol dependence have found that these individuals smoke more cigarettes per day (Cargill et al., 2001; Novy et al., 2001) and report more severe nicotine dependence than those without this history (Cargill et al., 2001; Hughes, 1993; Hurt, Dale, Offord, Croghan, Hays, & Gomez-Dahl, 1995; Novy et al., 2001).

The present study

The link between alcohol and smoking has wide-reaching public health implications, yet little is known about the impact of alcohol involvement on likelihood of smoking cessation, nor about potential mechanisms for such an association. We addressed these issues using data from a randomized clinical trial of counseling involving two types of framed messages (gain vs. loss) in conjunction with open label sustained-release (SR) bupropion (Toll et al., 2007). Most clinical trials of bupropion SR exclude participants with recent alcohol problems (e.g., alcohol dependence or excessive alcohol use) (Ahluwalia, Harris, Catley, Okuyemi, & Mayo, 2002; Hurt et al., 1997; Jorenby et al., 1999) due to the increased seizure risk associated with bupropion SR. While smokers were excluded from the present study if they met criteria for current alcohol dependence, a number of smokers with different levels of alcohol involvement *were* included in this study, including those meeting criteria for current alcohol abuse and those reporting heavy drinking at baseline. Given that those with current diagnoses of alcohol dependence were excluded from the present clinical trial, it was possible to examine the effect of lifetime AUD history in the same statistical models as level of alcohol use given that our lifetime AUD history and alcohol use variables were fully factorial (i.e., one could be at any level of alcohol use regardless of one's lifetime AUD history and vice-versa). To our knowledge, no other smoking cessation studies using bupropion SR have examined relationships between alcohol and smoking.

We categorized participants according to their lifetime AUD status and their level of alcohol consumption and conducted a series of analyses. Regarding baseline alcohol use, no predictions were made for associations with predictors of smoking relapse assessed at baseline, given that there has been little prior research on this issue. The priming hypothesis, however, would predict that alcohol consumption would increase the likelihood of smoking and, in turn, increase risk of relapse. To test these predictions, we first examined whether alcohol consumption (both any consumption and specifically, heavy use) increased the probability of any smoking. We also analyzed level of alcohol consumption both at baseline and post-quit along with AUD history as predictors of outcome. While we predicted that alcohol use would increase the likelihood of any smoking and the likelihood of smoking relapse in this trial, we made no specific prediction as to whether any drinking or specifically heavy drinking would increase risk of smoking relapse, given the scant evidence in the literature to date.

We predicted that smokers with a lifetime AUD would not be more likely to relapse than those without a history of alcohol problems. We also predicted that a lifetime AUD history would not be associated with the traditional predictors of smoking cessation relapse assessed in this study, nor did we predict significant alcohol use $\times$ AUD history interactions. There were theoretical and empirical justifications for this prediction. The priming hypothesis would predict that exposure to alcohol increases likelihood of smoking and accordingly, likelihood of relapse. Following the priming hypothesis, the main potential relapse risk associated with a lifetime history of an AUD would be through increased exposure to alcohol. However, lifetime AUD history would not necessarily be associated with repeated exposure to alcohol given that those with current alcohol dependence were excluded. Further, such an effect would be partialled out in statistical models including both level of alcohol use and lifetime AUD history. Smokers with a lifetime history of an AUD could be at increased risk of relapse due

to other vulnerabilities (e.g., genetic, personality traits), however most findings in the clinical trial literature suggest no added risk for those with a *past* history of alcohol problems. Thus, we expected similar null findings.

Methods

Participants and Procedures

Participants were screened for inclusion between February 2003 and July 2004. Smokers were screened initially on the phone and callers who appeared to meet current alcohol dependence criteria were excluded. All participants gave written informed consent at intake and later completed a core battery of measures. To be eligible, smokers had to be between the ages of 18 and 70, English-speaking, smoking at least 10 cigarettes per day, with an expired carbon monoxide (CO) level greater than 10 parts per million (ppm) and at least one prior cessation attempt. No more than one person per household could enroll. Women were excluded if they were pregnant, nursing, or not using reliable birth control. Smokers were excluded for current serious neurologic, psychiatric, or medical illness (as assessed by the study physician), sharing an immediate work environment with a current or past participant, current alcohol dependence, current major depressive episode, history of anorexia nervosa or bulimia, current use of psychoactive medications, history of seizures, previous hypersensitivity to bupropion, or current use of smokeless tobacco, pipes, cigars, nicotine replacement products or marijuana. This study was approved by the Institutional Review Board of the Yale University School of Medicine.

All participants were provided with a psychosocial intervention that emphasized either the benefits of quitting smoking (gain-framed) or the losses related to continued smoking (loss-framed) and open label bupropion SR (150 mg per day for 3 days, then 300 mg daily). The psychosocial intervention was not designed to provide participants with any skills directly related to limiting their alcohol use. Brief mention was made of the increased seizure risk associated with heavy alcohol use while taking bupropion SR, but no further alcohol-related advice was given. There was a 1-week medication lead-in before the quit date. From the quit date, participants were provided bupropion SR for a 6-week period. Participants were seen twice before their quit date (Weeks -1 & 0), then bi-weekly afterward (Week 2, 4, & 6). Questionnaires were administered, and framed video and print messages encouraging smoking abstinence (i.e., 2 short videos, print matter, and a water bottle and air freshener with printed slogans on them) were provided at these appointments (Toll et al., 2007). Relapse was defined on a point-prevalence basis as self-reported cigarette use in the week prior to assessment, paired with a carbon monoxide reading greater than 10 ppm, or being lost to follow-up. Assessments to determine abstinence/relapse took place at the end of the 6-week treatment period and then approximately 12 weeks and 24 weeks following the quit date.

Measures

Demographics—Participants self-reported their age, racial/ethnic identity, highest educational attainment, and marital/cohabitation status. Participants reported on their family history of alcohol problems using a single item measure in which they were asked whether any of their blood relatives (parents or siblings) had experienced at least 1 of the following due to their drinking behavior: legal, health, marital/family or work problems/difficulties with responsibilities around the house; treatment for alcoholism or social problems/fights/loss of friends.

Alcohol involvement—Participants reported on their alcohol consumption at baseline and during smoking cessation treatment using a Timeline Followback interview (TLFB; Sobell & Sobell, 2003). In the TLFB, participants are presented with a calendar including memory

prompts (e.g., holidays) and are asked to estimate the number of standard drinks of alcohol they consumed on each day. Responses to the TLFB were used to define drinking levels at baseline and post-quit (see data analysis section below).

The alcohol disorders section of the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID; First, Spitzer, Gibbon, & Williams, 1996) was used to diagnose lifetime alcohol use disorders (AUDs). The SCID is designed to determine symptoms of AUDs according to DSM-IV criteria and has been shown to yield valid diagnoses of alcohol dependence (Kranzler, Kadden, Babor, Tennen, & Rounsaville, 1996). Because individuals meeting criteria for current alcohol dependence were excluded, the lifetime AUD group included participants with a past or current history of alcohol abuse or a past history of dependence.

Smoking—A questionnaire assessed basic smoking status and history, including number of cigarettes currently smoked per day; age of first cigarette use and age of initiation of daily smoking; whether a quit attempt was made in the past year and whether participants had quit for at least a year in the past. The Fagerström Test for Nicotine Dependence (FTND) was used to measure severity of dependence on nicotine. The FTND is a six-item scale by Heatherton et al. (1991) with an internal consistency reliability of .61. The total score of this scale is closely related to biochemical measures of intensity of smoking. Self-reports of smoking post-quit were obtained with the same TLFB that was used for drinking data. Blood samples for cotinine analysis were obtained at baseline and analyzed using high-performance liquid chromatographic techniques at the Yale TTURC laboratory core facilities. Baseline CO levels were measured using a Vitalograph Breath CO monitor (Vitalograph, Inc., Lenexa, KS). Both cotinine and CO have been shown to be accurate indicators of tobacco use (Benowitz, 1996; SRNT Subcommittee on Biochemical Verification, 2002).

Motivation to quit/self-efficacy—Temptation to smoke was measured using 9 self-report items designed to assess temptation in various situations involving positive affect, negative affect, and habitual/craving situations (Velicer, DiClemente, Rossi, & Prochaska, 1990). High scores are indicative of a high degree of temptation and low self-efficacy. Benefits and costs of smoking were assessed using a 6-item short form of the Pros and Cons subscales from the 24-item Decisional Balance scale (Velicer, DiClemente, Prochaska, & Brandenburg, 1985). Participants' motivation to quit was assessed using a single item on a 1–10 scale. Commitment to abstinence from cigarettes was assessed using another single item, with responses dichotomized to a goal of strict abstinence or not (Hall, Havassy, & Wasserman, 1990).

Psychological factors—Depression was assessed using the Center for Epidemiologic Studies – Depression Scale (CES-D; Radloff, 1977), a 20-item self-report instrument that has been extensively used in both clinical and non-clinical populations to estimate severity of depressive symptoms in adults (Weissman, Sholomskas, Pottenger, Prushoff, & Locke, 1977). Perceived stress was measured using a 4-item version of the Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983).

Data analysis

Variable definitions—The main analyses in this report involved 2 types of drinking variables: 1. categorical variables where participants were designated as abstinent, moderate or hazardous drinkers and 2. continuous variables depicting the likelihood of smoking on drinking and on non-drinking days. In addition, traditional alcohol variables (i.e., drinks per occasion, percent of days on which any drinking occurred and percent of days on which heavy drinking occurred) were examined in order to characterize the drinking habits of the sample. Categorical drinking class variables were defined for 3 time periods: the baseline period (i.e., the 28 days before participants began taking bupropion), the 6-week treatment period following

the quit date, and the 12 week post-quit period (i.e., the entire period beginning with the quit date through the follow-up appointment 6 weeks after the end of treatment). In defining these drinking variables, all available data were used leading up to the assessment date or the last date when participants provided data. Alcohol use during the 1-week medication lead-in period prior to the quit date was not considered.

Hazardous drinkers self-reported violating the daily and/or weekly maximum recommended quantity of alcohol consumption according to NIAAA guidelines (2005; i.e., no more than 4 drinks on any day or 14 in any week for males; no more than 3 drinks on any day or 7 in any week for females) at least once. Moderate drinkers reported consuming alcohol at least once but did not violate the daily or weekly drinking guidelines. Abstainers reported consuming no alcohol. To determine whether participants violated the weekly drinking guideline, each 7-day period was examined, beginning with the first day of the quit attempt (i.e., day 1–7, day 8–14, day 15–21, etc.). For participants who either dropped out or who had appointments that did not fall exactly 6 or 12 weeks after the beginning of their quit attempt, each complete 7-day period beginning with the quit date was examined, along with the 7-day point-prevalence period. If the participant violated weekly drinking guidelines during any of these 7 day periods, they were classified as a hazardous drinker. This procedure allowed use of all days on which participants self-reported drinking behavior in defining their drinking class.

Predictor analyses—Two types of analyses were conducted to assess relationships between baseline alcohol involvement and traditional predictors of smoking cessation failure. For continuous predictors, *MANOVAs* were used to assess the effects of baseline alcohol use, lifetime AUD history, and the interaction between the alcohol use and AUD history on 4 clusters of variables: 1) smoking severity (i.e., number of cigarettes per day, FTND score, and baseline cotinine and CO levels); 2) age of smoking initiation (i.e., age of first cigarette use and age of initiation of regular smoking); 3) motivation to quit and self-efficacy (i.e., self-efficacy to resist temptation in social/positive affect situations, negative affect situations, instances of craving, and instances when cigarettes are habitually smoked; pros and cons of smoking; and motivation to quit smoking); and 4) psychological factors (i.e., depression and perceived stress). Chi-square analyses were used to assess associations between baseline alcohol involvement and several dichotomous predictors of smoking cessation failure: gender, married/cohabitating or not, race (i.e., white vs. non-white), education level (i.e., high school or less versus some post high-school), presence or absence of other smokers in the household, family history of alcohol problems, at least 1 quit attempt in the past year or none, length of longest prior quit attempt (i.e., greater than or equal to 1 year or not) and treatment goal (i.e., strict abstinence or not).

Analyses to assess associations between smoking and drinking behavior—

Variables were created to assess associations between smoking and drinking behavior during the 6-week treatment and 12-week post-quit periods. Percentage of days on which both any smoking and any drinking occurred was divided by the overall percentage of drinking days to obtain the probability of any smoking on a drinking day. A similar approach was used to obtain the probability of any smoking on abstinent, moderate (i.e., 1–4 drinks for males, 1–3 for females) and heavy drinking days. Paired-samples *t*-tests were used to compare the probability of any smoking on drinking days versus abstinent days. To ascertain whether any drinking and/or heavy drinking specifically led to a greater probability of any smoking, these analyses were conducted once including participants reporting 1 or more heavy drinking days and then were repeated excluding heavy drinkers. Paired-samples *t*-tests were also used to compare the probability of any smoking occurring on heavy versus non-heavy drinking days (i.e., abstinent or moderate drinking). Repeated measures *ANOVAs* were used to compare probability of any smoking occurring on abstinent, moderate and heavy drinking days. To be included in a given analysis, participants had to have reported at least 1 of each type of drinking day assessed in

that analysis. For instance, to be included in the *ANOVA*, participants had to have reported at least 1 abstinent day, at least 1 moderate day and at least 1 heavy drinking day.

Outcome analyses—To assess relationships between alcohol involvement and smoking relapse, univariate logistic regressions were first performed to determine whether either lifetime AUD history or alcohol use level were associated with relapse at the end of the 6-week treatment period and/or at the end of the 12-week follow up. These outcomes were selected because participant retention was highest at end of treatment (70.7%) and at Week 12 (47.8%). We did not examine Week 24 outcomes because only 34.1% of the sample was retained at Week 24, meaning that two-thirds of the sample provided no additional alcohol or smoking data for the 3 months since the prior assessment. Following the univariate analyses, hierarchical logistic regression models were used in which counseling condition (i.e., gain or loss-framed) was entered at step 1; other predictors of smoking cessation failure (e.g., nicotine dependence) were entered at step 2; baseline level of alcohol use and lifetime AUD history were entered at step 3; level of alcohol use during treatment or during the 12-week post-period, depending on the outcome measure, was entered at step 4 and interactions of alcohol use level by AUD history were entered at step 5. Bivariate correlations between pairs of variables included in the same regression were checked to examine multicollinearity. Correlations of .70 or greater were considered to be indicative of multicollinearity, which is stricter than the .80 guideline recommended by Field (2000).

To select the predictor variables to be entered at step 2, a series of univariate analyses were conducted to assess each variable's association with end of treatment and 12-week smoking outcome. Those variables showing a statistical trend or greater in an association with an outcome measure (i.e., $p < .10$) were entered into regression analyses. Backward elimination based on the Wald statistic was used with the predictor variables entered at step 2. When the p value of a particular variable's Wald statistic was greater than or equal to .10, it was eliminated from the final model. Outcome analyses were performed in 2 ways: 1) including only those participants who reported post-quit drinking data and 2) with an intent-to-treat approach, in which baseline drinking class was carried forward for those who did not report post-quit drinking data.

All p values are two-tailed. Data from outliers were winsorized to 3 standard deviations above the mean. For all analyses, participants with missing data for any variable included in that analysis were omitted. For missing values within scales and subscales, mean imputation was used in the case of 1 missing item, but where 2 or more items were missing, the entire scale or subscale was entered as missing. All analyses were conducted using SPSS version 15.0 for Windows (SPSS Inc., 2006).

Results

Description of sample

The intent-to-treat sample for this study was comprised of 249 smokers who were declared eligible, enrolled in the study and started the study treatment (i.e., attended the first treatment appointment when they received medication and framed messages). The sample was primarily White. The most common minority group was African-Americans (10.1%), followed by Asians (2.8%). The gender distribution was almost equal and the sample had almost equal numbers of married/cohabitating and unmarried/non-cohabitating individuals. Most participants had at least some post-high school education (Table 1).

At baseline, the sample reported consuming just under 2 drinks per occasion ($M = 1.89$, $SD = 1.82$) and drinking heavily on just over 3% of reported days ($M = 3.3\%$, $SD = 7.5\%$). Both of these indices remained stable during treatment, as shown by highly non-significant paired

samples *t*-tests [drinks per occasion: $t(207) = 0.42, p = .68$; percent heavy drinking days: $t(207) = 0.52, p = .60$]. Mean percent of days on which any drinking occurred was 21.9% ($SD = 29.1\%$), which declined slightly to 19.8 ($SD = 28.2\%$) during treatment [$t(207) = 2.36, p = .019$].

Moderate drinkers made up the largest proportion of the sample at baseline and approximately two-fifths had a lifetime AUD history (Table 1). According to chi-square analysis, baseline drinking level was associated with lifetime AUD history in that abstainers and hazardous drinkers at baseline were more likely than moderate drinkers to have a lifetime AUD [$\chi^2(2, N = 249) = 6.06, p = .048$]. “Moderate drinker” remained the most common classification (38%) during the treatment period, followed by “hazardous drinker” (33.7%) and “abstainer” (28.4%). Taking the 12-week post-quit period into consideration, things changed slightly with “hazardous drinker” (38.9%) being the most common, followed by “moderate drinker” (36.5%) and “abstainer” (24.5%). Correlations between drinking classifications at baseline and during treatment (*Spearman rho* = 0.68, $p < .001$) and between baseline and the 12-week post-quit period (*Spearman rho* = 0.64, $p < .001$) were both highly significant, suggesting stability in drinking classifications from baseline to post-quit.

Differences in predictors of smoking relapse by baseline drinking level and AUD history

There were a few differences in demographic predictors of relapse across alcohol classifications. A lifetime AUD was associated with male gender [$\chi^2(1, N = 249) = 6.45, p = .011$] and not being in a marital or cohabiting relationship [$\chi^2(1, N = 249) = 4.21, p = .04$]. Regarding alcohol use patterns at baseline, hazardous drinkers were less likely to be married/cohabitating than were moderate drinkers [$\chi^2(2, N = 249) = 6.62, p = .037$] and were more likely than moderate drinkers and abstainers to have no other smokers in the household [$\chi^2(2, N = 249) = 6.49, p = .039$] (Table 1).

A series of chi-squares was conducted to assess differences in non-demographic, dichotomous predictors of treatment failure by alcohol involvement (Table 2). There were no significant differences by AUD history. Moderate and hazardous drinkers were more likely than abstainers to have made an attempt to quit smoking in the past year [$\chi^2(2, N = 218) = 7.42, p = .024$]. There were no significant differences with respect to commitment to a goal of strict abstinence from cigarettes or whether their longest time off cigarettes lasted at least 1 year.

Descriptives for predictors of smoking cessation failure that were included in the *MANOVAs* are reported in Table 3. The *MANOVAs* yielded few significant differences by baseline drinking level and no significant differences by lifetime AUD history. No interactions between baseline drinking level and AUD history were significant. In a *MANOVA* including number of cigarettes per day, FTND score, baseline cotinine and CO levels, there was a significant omnibus effect for baseline drinking level [Wilks' $\lambda = .91, F(8, 472) = 2.74, p = .006$], but not for AUD history [Wilks' $\lambda = .98, F(4, 236) = 1.08, p = .37$]. Based on tests of between-subjects effects for individual measures, the largest difference was in FTND scores, where abstainers were more nicotine dependent than hazardous drinkers [$F(2, 239) = 8.50, p < .001$]. Abstainers also had higher baseline cotinine than hazardous drinkers [$F(2, 239) = 3.90, p = .022$]. The results of a *MANOVA* on continuous measures of motivation to quit/self-efficacy revealed a significant omnibus effect of baseline drinking level [Wilks' $\lambda = .83, F(12, 460) = 3.86, p < .001$], but not of AUD history [Wilks' $\lambda = .99, F(6, 230) = 0.56, p = .76$]. Based on tests of between-subjects effects for individual measures, baseline abstainers had higher scores than hazardous drinkers for decisional balance in favor of continued smoking [$F(2, 235) = 3.59, p = .029$]. Neither of the other two *MANOVAs*—assessing differences in variables related to age of smoking initiation and psychological risk factors yielded—significant omnibus effects by either baseline drinking level or AUD history.

Analyses comparing probability of smoking on drinking versus abstinent days

During the 6-week treatment period, the probability of any smoking on a drinking day (24.3%, $SD = 37.1\%$) was significantly higher than the probability of any smoking on an abstinent day (15.9%, $SD = 28.7\%$) when participants reporting at least 1 heavy drinking day were included in the analysis [$t(137) = 3.71, p < .001$], but this difference was not significant when heavy drinkers were omitted [drinking day: 23.7%, $SD = 38.1\%$; abstinent day: 18.6%, $SD = 30.4\%$], $t(82) = 1.84, p = .07$]. As shown in Figure 1, this pattern held across the 12-week post-quit period [with heavy drinkers included: $t(147) = 3.39, p = .001$, heavy drinkers omitted: $t(79) = 1.12, p = .27$]. In addition, the probability of any smoking was significantly higher on a heavy drinking day (treatment: 34%, $SD = 42.9\%$, 12-week post-quit: 38.5%, $SD = 42.6\%$) than on a non-heavy drinking day (treatment: 11.9%, $SD = 25.1\%$, 12-week post-quit: 19.1%, $SD = 27.7\%$) both during the 6-week treatment [$t(58) = 4.67, p < .001$] and 12-week post-quit periods [$t(69) = 4.57, p < .001$].

In a three-way comparison, the probability of any smoking on a heavy drinking day (36.4%, $SD = 43.4\%$) was significantly greater than the probability of any smoking on either an abstinent (11.7%, $SD = 25.5\%$) or moderate drinking day (13%, $SD = 28.7\%$) during the treatment period. The sphericity assumption was violated in this analysis (Mauchly's $W = 0.35, X^2(2, N = 55) = 56.46, p < .001$), thus the Huynh-Feldt correction was used [$F(1.22, 65.93) = 22.40, p < .001$]. As shown in Figure 2, a similar pattern held for the 12-week post-quit period [Mauchly's $W = 0.69, X^2(2, N = 60) = 21.83, p < .001$; $F(1.55, 91.72) = 11.79, p < .001$]. [Insert Figures 1 and 2 about here]

Analyses predicting relapse using drinking levels, AUD history and other predictor variables

While the intent-to-treat sample contained 249 smokers, 41 of these individuals did not return post-quit to provide further information about their smoking and alcohol use behavior and were considered to have returned to smoking. Of these 41 participants, 33 had no lifetime history of an AUD. With respect to baseline drinking level, 13 were abstinent, 18 were moderate and 10 were hazardous drinkers. All analyses reported in this section were run using both the intent-to-treat sample and the portion of the sample who returned post-quit ($N = 208$). The percent of participants who relapsed in the intent-to-treat and post-quit samples at the end of the 6-week treatment period and at the end of the 12-week post-quit period are presented in Tables 4 and 5, respectively, for the sample as a whole and by AUD history and baseline drinking level.

By the end of the treatment period, 57.4% of the sample had relapsed, while 76.7% had relapsed by the end of the 12-week post-quit period. Based on the univariate logistic regression analyses, there were no significant differences in likelihood of relapse based on either AUD history or baseline drinking level (see Tables 4 and 5, results of the univariate regressions are available from the authors). Participants classified as moderate drinkers during treatment appeared slightly more likely (61.9% smoking in intent-to-treat, 53.2% post-quit sample) than either abstainers (56.9% in intent-to-treat, 47.5% in post-quit sample) or hazardous drinkers (52.5% in intent-to-treat, 45.7% in post-quit sample) to have returned to smoking by the end of the treatment period. However, those classified as moderate drinkers during the 12-week post-quit period appeared less likely (73.4% smoking in intent-to-treat, 67.1% post-quit sample) than abstainers (84.4% in intent-to-treat, 80.4% in post-quit sample) to have returned to smoking by the end of the 12 weeks, with hazardous drinkers having similar rates of smoking (74.7% in intent-to-treat, 71.6% in post-quit sample). However, based on univariate logistic regressions, none of these differences were statistically significant (results available from the authors).

Results of hierarchical logistic regressions predicting relapse by the end of treatment and by the end of the 12-week post-quit period are provided in Table 6. In these analyses, relapse was entered as “1” and abstinence was entered as “0,” therefore a variable with an odds ratio greater than 1 was associated with an increased likelihood of relapse. As the intent-to-treat and post-quit samples yielded similar results, we report results only from the post-quit sample below and in Table 6. The baseline drinking level $\times$ lifetime AUD diagnosis interaction and the during treatment/during post-quit drinking level $\times$ AUD diagnosis interactions were not significant predictors of relapse in any model at either time point, thus all findings reported below and in Table 6 are from step 4 in the model, when post-quit drinking level was first entered, prior to the step when the interactions were entered.

Two predictor variables met criteria for inclusion in the model predicting relapse by the end of treatment: perceived stress and decisional balance against continued smoking. The model predicting relapse at end of treatment was not significant, $X^2(8, N = 203), p = .07$, and none of the individual predictor variables were significant predictors of relapse (Table 6).

Four predictor variables met criteria for inclusion in the model predicting relapse by the end of the 12-week post-quit period: age, marital/cohabitation status, education level and a treatment goal of strict abstinence. This model was statistically significant, $X^2(10, N = 194), p = .001$. In comparison with hazardous drinking at baseline, abstinent and moderate drinking at baseline were both associated with a significantly decreased likelihood of relapse, however drinking level during the post-quit period was not predictive of relapse. Of note, abstinence from alcohol at baseline was not a significant predictor of relapse at step 3 [$\beta = -0.59$, standard error = 0.48, odds ratio = 0.55 (95% confidence interval = 0.22–1.41), $p = 0.22$], but became a significant predictor at step 4 when drinking level during the post-quit period was entered into the model. Age had a significant negative association with relapse, meaning that the older the participants were, the less likely they were to relapse. Being married/cohabitating, having some post-high school education and reporting a treatment goal of strict abstinence from cigarettes were all significantly associated with a decreased likelihood of relapse as well. AUD history and counseling condition were not significant predictors of relapse (Table 6).

Correlations among predictor variables included in the same regression were checked to assess multicollinearity. All correlations were under .70, thus we conclude that multicollinearity was not a threat to the validity of any of the multiple regression analyses reported here.

Discussion

The present study evaluated whether level of alcohol use and/or a lifetime history of an AUD were related to known predictors of smoking cessation failure and to smoking cessation relapse in a clinical trial of bupropion SR. This study is novel for a number of reasons. In general, there has been a lack of attention to alcohol involvement in the smoking cessation literature, particularly with respect to current alcohol use. To our knowledge, this is the first report concerning the effects of alcohol involvement from a clinical trial of bupropion SR. Findings from this study suggest that smoking is more likely to occur on heavy drinking days than on other days and that hazardous drinking predicts greater risk of relapse than either moderate drinking or abstinence from alcohol.

There were few differences in predictors of smoking cessation failure by either baseline drinking level or AUD history. There was some evidence that abstainers had more risk factors than either moderate or hazardous drinkers. Abstainers reported more severe nicotine dependence and had higher cotinine levels than hazardous drinkers. Abstainers also reported decisional balance in favor of continued smoking to a greater extent than hazardous drinkers did. Abstainers were also less likely than moderate and hazardous drinkers to have made a quit

attempt in the past year and were more likely than hazardous drinkers to reside with at least one other smoker. It is possible that these differences may have contributed to moderate drinking offering a slightly greater advantage in smoking cessation compared with abstainers. However, these risk factors did not appear to place abstainers in any real jeopardy with regard to smoking cessation as baseline abstinence offered a significant advantage in avoiding relapse at the end of 12 weeks in comparison with baseline heavy drinking when post-quit drinking was added to the regression model. Other findings from the predictor analyses were that moderate drinkers were more likely than hazardous drinkers to be married/cohabitating and those with a lifetime AUD were more likely to be male and unmarried/non-cohabitating. These were the only significant differences by AUD history.

The probability of any smoking was greater on drinking days than on abstinent days during both the 6-week treatment period and the 12-week post-quit period. This pattern was driven by heavy drinking in that the probability of any smoking was greater on drinking days only when those with at least 1 heavy drinking day were included in the analyses. Also, the probability of any smoking was higher on heavy drinking than on non-heavy drinking days and in 3-way analyses, smoking probability was higher on heavy than on moderate or abstinent days.

AUD history did not predict smoking cessation relapse either by the end of the 6-week treatment period or by the end of the 12-week post-quit period in either the univariate or the hierarchical regression analyses. Baseline drinking level did not predict 6-week treatment or 12-week post-quit smoking relapse in univariate analyses. In the hierarchical regressions, baseline drinking level was not predictive of 6-week treatment relapse, but was predictive of relapse at the end of the 12-week post-quit period. Baseline moderate drinking and, to a lesser extent, abstinence were predictive of a lower likelihood of relapse than baseline hazardous drinking.

Post-quit alcohol use was not predictive of smoking cessation relapse in any analyses. It is possible that drinking patterns at baseline were more indicative of the “true” drinking habits of smokers in this study. It is difficult to determine what effect, if any, smoking cessation treatment may have had on participants’ spontaneous drinking in this population of individuals who were seeking treatment for smoking but not for drinking. There was a high degree of continuity between drinking at baseline and post-quit, with only a significant decrease in percent drinking days being observed. These findings suggest that notable changes in drinking behavior did not occur for the most part, but it is possible that more subtle changes were made. The lack of a placebo control makes it impossible to ascertain whether bupropion had an impact on drinking behavior in this trial. Standard deviations for continuous alcohol variables and odds ratios for categorical alcohol variables in the logistic regressions were high. This means that there was a great deal of variability in associations between alcohol use and smoking among participants in this study. For example, any smoking occurred on only about 1/3 of heavy drinking days during the 12 week post-quit period, meaning that on most days, participants did not smoke even when consuming a high quantity of alcohol. Reasons why alcohol use leads to smoking and eventual relapse for some individuals, but not for others, is an important but as yet unanswered question in the smoking epidemiologic, laboratory and clinical trial literature. As there were few differences in the predictor variables assessed in this study by drinking level, findings from the present study will unfortunately not advance this issue.

We examined AUD history as a predictor of relapse and as a potential moderator of associations between current alcohol use and relapse. As predicted, based on the priming hypothesis and prior findings (Aubin et al., 2004; Croghan et al., 2003; Hayford et al., 1999; Hughes et al., 2003; Humfleet et al., 1999; Hurt et al., 1995; Murray et al., 1995; Simon et al., 2004), smokers with a lifetime AUD were not more likely to relapse and AUD history did not moderate associations between drinking level and relapse. Unlike many prior studies, the AUD history

group in the present study included those with current alcohol abuse. Nonetheless, the present findings were similar to those in the literature.

Our findings that moderate drinking offered an advantage in smoking cessation compared with hazardous drinking appear to contradict the most recent clinical practice guidelines for smoking cessation, in which abstinence from alcohol is recommended during smoking cessation attempts (Fiore et al., 2000). While, both the present findings and the clinical practice guidelines point to the importance of limiting drinking while attempting to quit smoking, the present findings suggest that increased risk of smoking cessation relapse may be associated with hazardous use to a greater extent than it is with moderate use.

Findings from the present study support the priming hypothesis (see Rohsenow et al., 1997; Tiffany, 1990). This conclusion is speculative given that we did not directly assess priming of smoking by alcohol in this study. Further, this conclusion must be qualified with the observation that the increased probability of any smoking was associated with heavy drinking days to a greater extent than with moderate drinking days and that hazardous rather than moderate use reported at baseline predicted relapse. The present findings follow prior findings from epidemiologic/survey studies (Agudo et al., 2004; Dawson, 2000; Zimmerman et al., 1990) and clinical trials (McClure et al., 2002; Murray et al., 1995), in which heavier drinkers, rather than drinkers in general, have been found to be at particular risk of smoking cessation failure.

The extent to which these findings would translate to smoking cessation treatments other than bupropion is uncertain and requires further research. The sample in this study was composed of treatment-seeking smokers and accordingly, the present findings may not apply to non-treatment seekers. The exclusion of smokers currently consuming alcohol at exceptionally high quantities on a regular basis and those with current alcohol dependence were important limitations to this study. While the alcohol-related exclusion criteria in the present trial were less restrictive than in many other smoking cessation trials (Leeman, Huffman, & O'Malley, 2007), the subgroup of smokers with the highest levels of current alcohol use and current alcohol dependence were excluded, and thus our findings do not necessarily apply to these smokers. A review of studies examining smoking cessation in current alcoholic and alcohol dependent smokers in alcohol treatment suggest that this population may have particular difficulties in quitting smoking (Cooney, Cooney, Patten, & George, 2004). A review of retrospective smoking cessation studies was conducted by Hughes (1995), who found that a median of 17% of current alcoholics who were lifetime smokers were successful in quitting smoking, which was about half the quit rate that was observed over the general population.

In conclusion, smoking was more likely to occur on heavy drinking days compared to moderate drinking or abstinent days and a hazardous pattern of alcohol use at baseline was associated with an increased likelihood of smoking cessation relapse defined on a point-prevalence basis 12 weeks after the quit date. Thus, risk of any smoking and of smoking cessation relapse appeared to be associated with heavy and hazardous use respectively, rather than with moderate alcohol use. Further research, particularly analyses of clinical trial data, is needed to elucidate reasons why the alcohol/smoking link is stronger for some smokers than others. Lifetime AUD history did not predict relapse in the present clinical trial. This finding further supports the conclusion that smokers with a history of alcohol problems are not at a significant disadvantage in a given quit attempt (see Hughes & Kalman, 2006, & Leeman et al., 2007, for an overview), although likelihood of cessation among those with current alcohol dependence may be lower. Based on these findings, the exclusion of individuals reporting a past history of alcohol abuse or dependence from smoking cessation studies does not appear to be warranted (Leeman et al., 2007), and doing so would exclude a considerable population of smokers. To better understand

the role of alcohol involvement as a predictor of treatment response, smoking researchers should assess alcohol use and history of alcohol problems in all nicotine and tobacco studies.

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** significantly higher probability of smoking at $p < .01$

Analyses include only participants who reported at least 1 drinking day *and* at least 1 abstinent day during treatment

Figure 1.

Comparisons between the probability of any smoking on drinking versus abstinent days during the 12-week post-quit period in analyses including and omitting participants reporting at least 1 heavy drinking day

"a" > "b" at $p < .001$

Analyses include only participants who reported at least 1 heavy drinking day, at least 1 moderate drinking day *and* at least 1 abstinent day

Figure 2.

Comparisons between the probabilities of any smoking on abstinent, moderate and heavy drinking days during the 12-week post-quit period

Table 1
Demographics by baseline alcohol use level and alcohol use disorder history

Variables	Entire sample (N = 249)	Abstainer	Moderate Drinker	Hazardous Drinker	p value/ Significant main effects	No lifetime history	Alcohol use disorder history Lifetime history	p value/ Significant main effects
Alcohol abstainer at baseline	29.3%					26.8%	33.7%	History: Haz. > Mod. *
Moderate drinker at baseline	38.2%					43.9%	28.3%	No history: Mod. > Abs. & Haz. *
Hazardous drinker at baseline	32.5%					29.3%	38%	
No lifetime AUD history	63.1%	57.5%	72.6%	56.8%	Mod: No history > history *			
Lifetime AUD history	36.9%	42.5%	27.4%	43.2%				
Male	48.2%	39.7%	48.4%	55.6%	0.15			
Female	51.8%	60.3%	51.6%	44.4%		42%	58.7%	History: M > F *
Married/cohabitating	49.8%	49.3%	58.9%	39.5%	Mod: Married > Unmarried *	58%	41.3%	No history: F > M *
Unmarried/not cohabitating	50.2%	50.7%	41.1%	60.5%	Haz: Unmarried > Married *	54.8%	41.3%	History: Un > Married *
White	79.8%	75.3%	77.7%	86.4%	0.19	45.2%	58.7%	No history: Married > Un *
Non-White	20.2%	24.7%	22.3%	13.6%		79%	81.3%	0.66
High school or less education	36.5%	45.1%	35.8%	29.5%	0.14	21%	18.7%	
Some post high school	63.5%	54.9%	64.2%	70.5%		35.1%	38.9%	0.55
No other smokers in household	64.3%	57.5%	60%	75.3%	Haz: No one > at least 1 *	64.9%	61.1%	0.98
At least 1 other smoker	35.7%	42.5%	40%	24.7%		35.7%	35.9%	
Positive family history of alc.	37%	41%	34.1%	37.1%	0.69	32.8%	43.5%	0.11
Negative family history of alc.	63%	59%	65.9%	62.9%		67.2%	56.5%	
Mean age in years (SD)	42.66 (11.55)	44.93 (10.94)	42.42 (11.63)	40.90 (11.78)	0.09	43.03 (12.54)	42.03 (9.66)	0.51

* $p < .05$

mod = moderate drinker, haz = hazardous drinker

Baseline level of alcohol use was based on a 28-day Timeline Followback: abstainers reported no alcohol use; moderate drinkers reported drinking but never violated NIAAA daily or weekly drinking guidelines, heavy drinkers reported violating NIAAA daily or weekly guidelines at least once

Lifetime history of an alcohol use disorder is a past or current DSM-IV diagnosis of alcohol abuse or a past diagnosis of alcohol dependence according to the SCID

Non-demographic, dichotomous predictor variables by baseline level of alcohol use and alcohol use disorder history

Table 2

Predictor variables <i>N/n</i> (percent of sample)	Entire sample 249	Abstainer 73 (29.3%)	Moderate Drinker 95 (38.2%)	Baseline level of alcohol use		Significant main effects	No lifetime history 157 (63.1%)	Alcohol use disorder history Lifetime history 92 (36.9%)	Significant main effects
				Hazardous Drinker 81 (32.5%)					
No quit attempts in the past yr.	46.3%	60.3%	43.2%	37.8%	Abs: None > at least 1 Mod & Haz: at least 1 > none*	0.64	48.1%	43.4%	0.49
At least 1 quit attempt	53.7%	39.7%	56.8%	62.2%			51.9%	56.6%	
Longest prior quit attempt < 1 yr.	74.8%	79.3%	72.6%	73.7%			73.5%	76.8%	
Longest prior quit attempt ≥ 1 yr.	25.2%	20.7%	27.4%	26.3%			26.5%	23.2%	
Goal of strict abstinence	63.5%	63.8%	58.7%	68.8%	0.39		63.2%	64%	0.89
Alternate treatment goal	36.5%	36.2%	41.3%	31.3%			36.8%	36%	

*
p < .05

abs= abstinent, mod = moderate drinker, haz = hazardous drinker

Baseline level of alcohol use was based on a 28-day Timeline Followback: abstainers reported no alcohol use; moderate drinkers reported drinking but never violated NIAAA daily or weekly drinking guidelines, heavy drinkers reported violating NIAAA daily or weekly guidelines at least once

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Table 3
Adjusted means (SE) and analyses for continuous predictors of treatment failure by baseline level of alcohol use and alcohol use disorder history

Predictor variables N/n (percent of sample)	Entire sample 249	Baseline level of alcohol use			p value/ Significant main effects	Alcohol use disorder history			p value/ Significant main effects
		Abstainer 73 (29.3%)	Moderate drinker 95 (38.2%)	Hazardous drinker 81 (32.5%)		No lifetime history 157 (63.1%)	Lifetime history 92 (36.9%)		
1. Cigarette consumption/dependence									
Cigarettes per day (CPD)	22.52 (SD = 9.18)	24.47 (1.10)	22.51 (1.05)	21.48 (1.03)	0.14	22.51 (0.76)	23.13 (0.96)	0.62	
FTND score	5.38 (SD = 2.06)	6.01 (0.24)	5.59 (0.23)	4.69 (0.23)	abs > haz ***	5.42 (0.17)	5.44 (0.21)	0.92	
Cotinine level	260 (SD = 125)	318 (14.65)	288 (14.01)	262 (13.72)	abs > haz *	273 (10.13)	305 (12.79)	0.05	
Carbon monoxide level	22 (SD = 9.24)	23.23 (1.11)	21.11 (1.07)	21.88 (1.04)	0.38	21.54 (0.77)	22.61 (0.97)	0.39	
2. Age of smoking initiation									
Age of first cigarette	14.97 (SD = 3.35)	14.62 (0.41)	15.64 (0.39)	14.53 (0.38)	0.08	15.03 (0.28)	14.83 (0.36)	0.65	
Age regular smoking began	16.95 (SD = 3.46)	16.58 (0.42)	17.24 (0.41)	16.79 (0.39)	0.51	17.13 (0.29)	16.62 (0.37)	0.28	
3. Motivation to quit and self-efficacy									
Temp.: positive affect/social situations	3.58 (SD = 0.80)	3.44 (0.10)	3.60 (0.09)	3.68 (0.09)	0.17	3.55 (0.07)	3.59 (0.08)	0.68	
Temp.: negative affect situations	3.75 (SD = 0.92)	3.61 (0.11)	3.91 (0.11)	3.68 (0.10)	0.12	3.76 (0.08)	3.72 (0.10)	0.75	
Temp.: habitual/craving situations	3.20 (SD = 0.84)	3.28 (0.10)	3.29 (0.10)	3.02 (0.10)	0.09	3.22 (0.07)	3.17 (0.09)	0.63	
Decisional balance: pro	2.84 (SD = 0.96)	3.00 (0.11)	2.83 (0.11)	2.59 (0.11)	abs > haz *	2.89 (0.08)	2.72 (0.10)	0.18	
Decisional balance: con	3.12 (SD = 1.03)	2.93 (0.12)	3.31 (0.12)	3.04 (0.12)	0.07	3.11 (0.09)	3.08 (0.11)	0.81	
Motivation to quit	8.88 (SD = 1.24)	9.04 (0.15)	8.90 (0.15)	8.74 (0.14)	0.35	8.87 (0.10)	8.91 (0.13)	0.81	
4. Psychological risk factors									
CES-D score	8.79 (SD = 6.89)	8.40 (0.84)	9.15 (0.81)	8.94 (0.79)	0.80	8.61 (0.58)	9.05 (0.74)	0.65	
Perceived stress scale score	4.02 (SD = 2.60)	3.95 (0.32)	4.20 (0.31)	4.08 (0.30)	0.86	3.87 (0.22)	4.23 (0.28)	0.26	

* $p < .05$
** $p < .01$
*** $p < .001$

abs = abstainer, haz = hazardous drinker

FTND: Fagerstrom Test of Nicotine Dependence; Temp.: temptation to smoke in different situations (high scores indicate more temptation and less self-efficacy) and decisional balance (1 to 5 scale); motivation to quit was measured with a single item (1 to 10 scale); CES-D: Center for Epidemiologic Studies-Depression scale; perceived stress scale (0 to 16 scale)

Current alcohol use × alcohol use disorder diagnosis interaction was entered into each analysis but was not found to be significant in any

Percentage relapsed at the end of the 6-week treatment period by alcohol use disorder history and baseline drinking level in the intent-to-treat sample and among those providing post-quit information

		Intent-to-treat sample											
		AUD history main effect				Baseline drinking main effect				Baseline drinking × AUD effects			
Group: <i>N/n (percent of sample)</i>	Entire sample	No AUD history	AUD history	Abstinent	Moderate Drinker	Hazardous Drinker	Abstinent no AUD	Abstinent w/AUD	Moderate no AUD	Moderate w/AUD	Hazardous no AUD	Hazardous w/AUD	
	249 57.4%	157 (63.1%) 56.1%	92 (36.9%) 59.8%	73 (29.3%) 61.6%	95 (38.2%) 52.6%	81 (32.5%) 59.3%	42 (16.9%) 64.3%	31 (12.4%) 58.1%	69 (27.7%) 49.3%	26 (10.4%) 61.5%	46 (18.5%) 58.7%	35 (14.1%) 60.0%	
	Percent relapsed:												
		AUD history main effect				Baseline drinking main effect				Baseline drinking × AUD effects			
Group: <i>N/n (percent of sample)</i>	Entire sample	No AUD history	AUD history	Abstinent	Moderate Drinker	Hazardous Drinker	Abstinent no AUD	Abstinent w/AUD	Moderate no AUD	Moderate w/AUD	Hazardous no AUD	Hazardous w/AUD	
	208	124 (59.6%)	84 (40.4%)	60 (28.8%)	77 (37%)	71 (34.1%)	33 (15.9%)	27 (13%)	52 (25%)	25 (12%)	39 (18.8%)	32 (15.4%)	
	Percent relapsed:												
		44.4%	56.0%	53.3%	41.6%	53.5%	54.5%	51.9%	32.7%	60.0%	51.3%	56.3%	

Relapse was defined as having smoked during the week prior to assessment by self-report and/or carbon monoxide > 10 ppm at assessment appointment or being lost to follow-up

Level of current alcohol use was based on a 28-day Timeline Followback: abstainers reported no alcohol use; moderate drinkers reported drinking but did not violate

NIAAA daily or weekly drinking guidelines; hazardous drinkers reported violating NIAAA daily or weekend drinking guidelines at least once.

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Percentage relapsed at the end of the 12-week post-quit period by alcohol use disorder history and baseline drinking level in the intent-to-treat sample and among those providing post-quit information

		AUD history main effect			Baseline drinking main effect			Intent-to-treat sample				Baseline drinking × AUD effects						
Group: <i>N/n (percent of sample)</i>	Entire sample	AUD history main effect		AUD history	Baseline drinking main effect		Heavy Drinker	Intent-to-treat sample		Abstinent no AUD	Abstinent w/AUD	Baseline drinking × AUD effects		Moderate no AUD	Moderate w/AUD	Baseline drinking × AUD effects		
		No AUD history	No AUD history		Abstinent	Moderate Drinker		Heavy Drinker	Abstinent no AUD			Abstinent w/AUD	Moderate no AUD			Moderate w/AUD	Hazardous no AUD	Hazardous w/AUD
		157 (63.1%) 77.1%	92 (36.9%) 76.1%	73 (29.3%) 78.1%	95 (38.2%) 69.5%	81 (32.5%) 84%	42 (16.9%) 78.6%	31 (12.4%) 77.4%	69 (27.7%) 68.1%	26 (10.4%) 73.1%	46 (18.5%) 89.1%	35 (14.1%) 77.1%						
Percent relapsed:																		
		AUD history main effect			Baseline drinking main effect			Post-quit sample				Baseline drinking × AUD effects						
Group: <i>N/n (percent of sample)</i>	Entire sample	No AUD history	AUD history		Abstinent	Moderate Drinker	Heavy Drinker	Abstinent no AUD	Abstinent w/AUD	Moderate no AUD	Moderate w/AUD	Hazardous no AUD	Hazardous w/AUD					
	208	124 (59.6%)	84 (40.4%)		60 (28.8%)	77 (37%)	71 (34.1%)	33 (15.9%)	27 (13%)	52 (25%)	25 (12%)	39 (18.8%)	32 (15.4%)					
Percent relapsed:	72.1%	71.0%	73.8%		73.3%	62.3%	81.7%	72.7%	74.1%	57.7%	72.0%	87.2%	75.0%					

Relapse was defined as having smoked during the week prior to assessment by self-report and/or carbon monoxide > 10 ppm at assessment appointment or being lost to follow-up

Level of current alcohol use was based on a 28-day Timeline Followback: abstainers reported no alcohol use; moderate drinkers reported drinking but did not violate

NIAAA daily or weekly drinking guidelines; hazardous drinkers reported violating NIAAA daily or weekend drinking guidelines at least once.

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Summary of logistic regression analyses predicting relapse at the end of treatment and at the end of 12 weeks post-quit by level of alcohol use, lifetime AUD diagnosis, treatment group assignment and other predictor variables assessed at baseline

Relapse at end of treatment Variable	OR (95% CI)	Variable	Relapse at end of 12-week post-quit period OR (95% CI)
Assignment to gain framed group	0.68 (0.38–1.22)	Assignment to gain framed group	0.61 (0.30–1.24)
Perceived stress score	1.11 (0.99–1.26)	Age	0.97* (0.93–1.00)
Self-efficacy: decisional balance against smoking	0.78 (0.58–1.04)	Married/cohabitating status	0.44* (0.21–0.92)
		Post-high school education	0.41* (0.18–0.95)
		Treatment goal of strict abstinence	0.42* (0.19–0.94)
Baseline alcohol abstinence	0.87 (0.32–2.33)	Baseline alcohol abstinence	0.28* (0.09–0.92)
Baseline moderate alcohol use	0.50 (0.22–1.15)	Baseline moderate alcohol use	0.25* (0.10–0.66)
No lifetime history of an AUD	0.67 (0.37–1.20)	No lifetime history of an AUD	1.41 (0.68–2.93)
Post-quit alcohol abstinence	1.33 (0.49–3.63)	Post-quit alcohol abstinence	3.21 (0.91–11.41)
Post-quit moderate alcohol use	1.93 (0.85–4.39)	Post-quit moderate alcohol use	1.19 (0.49–2.88)

* $p < .05$

** $p < .01$; ChiSq: Chi-square, OR: odds ratio, CI: confidence interval

These analyses include only those participants who provided post-quit data

All findings are from step 4 of the regressions when post-quit drinking level was first entered, before the alcohol use $\times$ history interactions, which were not significant, were entered

Relapse was defined as having smoked the week prior to assessment by self-report and/or CO > 10 ppm, or being lost to follow-up. Relapse was entered as “1” and abstinence was entered as “0,” meaning that odds ratios > 1 indicate an increased likelihood of relapse

Level of baseline alcohol use was based on a 28-day Timeline Followback: abstainers reported no alcohol use; moderate drinkers reported drinking, but did not violate the NIAAA daily or weekly drinking guidelines; hazardous drinkers violated the guidelines at least once

Lifetime history of an alcohol use disorder is a past or current DSM-IV diagnosis of alcohol abuse or a past diagnosis of alcohol dependence according to the SCID