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The Incidence and Severity of Hangover the Morning after Moderate Alcohol Intoxication

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Abstract

Background—Differential propensity for hangover may play a role in determining individuals' drinking practices so predictors of incidence and severity are needed.

Methods—Data were combined from three randomized crossover trials investigating the residual effects of heavy drinking on next-day performance. All 172 participants received either an alcoholic beverage (M=.115 g% breath alcohol concentration [BrAC]) or placebo matched on type and amount one night and a week later received the other beverage. Alcoholic beverages were vodka, bourbon or high alcohol beer. After each drinking session, following a 9-hour period for sleep and breakfast, participants completed questionnaire a hangover measure.

Results—No hangover was reported by 24% of participants, mild hangover by 44% and moderate hangover by 32%. Neither alcoholic beverage type nor participant characteristics (sex, age, drinking practices, tobacco use, or family history of alcohol problems) were associated with incidence of hangover.

Conclusion—The majority of people experienced mild to moderate hangover the morning after this level of intoxication. Further studies are required to investigate other hypothesized causes of variation in the propensity for hangover.

Keywords

hangover; heavy drinking; family history of alcohol problems

Introduction

Hangover refers to the cluster of symptoms, including headache, nausea, thirst, and fatigue, occurring immediately after much or all of the alcohol has left the body following heavy drinking. The causes of hangover are unknown although hypotheses have been reviewed elsewhere (Wiese, Shlipak and Browner, 2000; Swift and Davidson 1998; Chapman 1970). Regardless of the cause, it is important to understand the incidence of hangover, and factors associated with variation in susceptibility, because hangover may play a role in the progression of social drinking to problematic drinking.

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Hangover is experienced at least occasionally by many drinkers. Although we found limited information on the incidence of hangover in the general population, some survey data is available for specific populations. For example, Frone (2006) recently reported that 7% (equal to about 11,595,377 workers) of a probability sample of U.S. workers experienced hangover at work during the prior year. One survey of college students found that 25% reported hangover during the previous week (Meilman et al., 1990). In a survey of Dutch university students, Vester (2006) found that 92% were drinkers, 61% reported a monthly average of 2.7 hangovers, and 32% reported no hangover during the previous year. Other surveys report hangover incidence for drinkers only. In a general survey of 1041 adults, Smith and Barnes (1983) found that 35% of drinkers (42% of males and 27% of females), and 50% of those who drank at least 2 drinks per day, experienced at least one hangover during the prior year. In a survey of drinkers by Harburg, et al. (1993), 23% reported never experiencing hangover symptoms, regardless of the amount that they drank. Using the 2001–2002 National Epidemiologic Study of Alcohol Related Conditions (NESARC) of a sample (N=43,093) of U.S. adults, we calculated that overall 21% of drinkers (22% of males and 21% of females) experienced at least one hangover symptom within the prior year. One study of patients admitted for treatment for alcoholism found that 23% reported never having had a hangover, despite a history of heavy drinking (Pristach, Smith, and Whitney, 1983). However, in surveys the amount of alcohol respondents consumed may vary widely, with some people possibly not drinking enough to experience hangover, so the prevalence of hangover at specific breath alcohol concentrations (BrAC) cannot be determined. Furthermore, surveys are subject to issues of recall and attribution bias, even when the word “hangover” is omitted from the measure.

In contrast, experimental studies could allow determination of the incidence and severity of hangover following specific ranges of intoxication, after all measurable alcohol has been eliminated. By tightly controlling the breath alcohol level attained and other controllable factors that could affect symptom reports (e.g., sleep derivation, food, medications) and assessing empirically supported symptoms when they are predicted to maximally occur, the best evidence can be obtained about the probability of experiencing hangover after a specific narrow range of alcohol ingestion. Although a number of experimental studies report on the next-day effects of alcohol consumption on neurocognitive performance (Finnigan et al., 1998; Chait and Perry, 1994; Lemon et al., 1993; Takala et al., 1957) or occupational performance (Rohsenow et al., 2006; Streufert et al.,1995; Yesavage et al. 1994; Morrow et al., 1990, 1991, 1993; Taylor et al., 1994, 1996; Tornos and Laurell, 1991; Yesavage and Leirer et al., 1986; Collins, 1980; Collins and Chiles, 1980; Wolkenberg et al.,1975), few studies provide details on the incidence of hangover the day after controlled experimental alcohol administration.

The one exception is the study by Chapman (1970). He administered beverage alcohol to 91 (50 males, 41 females) healthy young adult occasional or moderate drinkers. Ten received 1.0 ml/kg of alcohol (mean peak BrAC: .066 g%); 10 received 1.25 ml/kg (mean peak BrAC: .11 g%); 60 received 1.5 ml/kg (mean peak BrAC: .13 g%); and another 11 received 1.75 ml/kg (mean peak BrAC: .14 g%). Overall, 68% of participants reported any hangover the morning after alcohol administration. The percent of participants with hangover increased roughly, but not linearly, with the amount of alcohol received: 40% reported any hangover at 1.0 ml/kg of ethanol; 20% at 1.25 ml/kg; 80% at 1.5 ml/kg; and, 73% at 1.75 ml/kg. However, since three of these dose levels had only 10 or 11 participants, the figures are unstable. Also, since individual differences in absorption of alcohol cause variability in attained peak BrAC, and Chapman’s data support the idea that peak BrAC is more important than g/kg dose of alcohol administered in determining hangover severity level, studies targeting specific BrACs are needed. No other studies with a large number of participants have reported incidence of hangover at specific alcohol doses or BrAC levels. By

investigating the incidence of hangover at various levels of severity at a targeted BrAC (resulting in a narrow range of attained peak BrAC levels) the present study addresses a gap in the literature.

Susceptibility to hangover may vary across individuals who have consumed equivalent amounts of alcohol. Because this propensity may affect drinking patterns it would be useful to know what variables covary with hangover incidence and intensity.

One variable that may affect hangover incidence or intensity is the congener content (e.g., acetone, acetaldehyde, methanol) of the beverages. Bourbon has 37 times the amount of congeners as vodka (Nathan, et al., 1970), and in some studies (Katkin et al., 1970), but not others (Nathan et al., 1970), intoxication with bourbon is more performance impairing than intoxication with vodka. Smith and Barnes (1983) found no differences in the incidence of hangover attributable toialcoholic beverage preferences (wine, beer, liquor) when controlling for levels of consumption, but each of these classes of beverage varies widely in congener content (Greizerstein, 1970) so this comparison does not address congener effects. In Chapman (1970), 78% of those receiving bourbon, vs. 59% of those receiving vodka, reported any hangover, although this difference was not significant. One other study reported hangover effects increasing with increasing congener content, but without details on incidence (Pawan, 1973). We could investigate congener effects by comparing bourbon to vodka since these have been shown to have 422 vs. 11 mg/100ml total congeners (Nathan et al., 1970). However, the congener content of high-alcohol beer is not available, although regular strength beers average 6.8 times as many congeners as 80 proof vodka (Greizerstein, 1970), and thus should have intermediate congener content.

A second possible variable that could affect hangover incidence or intensity is family history of drinking as this may reflect biological differences in response to alcohol. Generally survey studies have found that family history of drinking problems is associated with greater frequency of hangover, controlling for reported drinking practices (Newlin and Pretorius, 1990; McCaul et al., 1991; Span and Earleywine, 1999; Slutske, Piasecki, Hunt-Carter, 2003; Piasecki, et al., 2005), with the exception of Earleywine (1993) and Richardson, Piasecki and Sher (2004), who found no association in samples of college students.

Recent quantity of drinking could also affect hangover incidence and intensity because of tolerance or other forms of adaptation to ingestion. Several survey studies have examined the relationship between drinking practices and the propensity for hangover, with mixed results (Smith and Barnes, 1983; Pristach et al., 1983; Kauhanen et al., 1993; Verster, 2006). Smith and Barnes (1983), Kauhanen et al. (1997), and Verster (2006) found that heavier drinkers reported more frequent hangovers than lighter drinkers probably due to more frequent drinkers more often attaining BAC levels that lead to hangover. In contrast, Pristach et al. (1983) found that among a group of alcoholics admitted for treatment, 50% had not had a hangover in the previous year. No studies investigate effects of heaviness of recent drinking while controlling for acute BAC the night before the hangover so these studies are generally confounded.

Use of tobacco might be another factor affecting propensity for hangover. Studies with animal models have shown that mesolimbic dopamine release is affected by the nicotinic receptor (Nadal & Samson, 1999) indicating that nicotinic receptors might modulate reinforcing effects of drugs on these neurons in this region, and inducing ethanol dependence in animals alters nicotinic receptor binding in specific brain regions (Booker & Collins, 1995). Thus, nicotine's pharmacologic actions in these neural systems could alter response to ethanol, as supported by its ability to increase alcohol consumption in naïve and ethanol-experienced rats (e.g., Nadal & Samson, 1999; Blomqvist et al., 1996). While

nicotine administration did not affect alcohol withdrawal in rats (Penland et al., 2001), withdrawal is not the same as hangover and acute administration is not the same as studying effects of chronic nicotine administration which could result in chronic receptor changes. Indeed, preliminary results from a recent survey study (Richardson et al., 2004) found hangover reports to be more frequent in smokers than in nonsmokers while controlling for quantity and frequency of drinking, family history of alcohol problems, and gender. While we could not experimentally administer nicotine at our clinical research facility, we could compare smokers to nonsmokers for severity and incidence of hangover.

Gender could be another predictor of differences in hangover. Neither Chapman's (1970) experimental study nor the survey study by Harburg et al. (1993) found differences in hangover incidence by sex. The survey study by Smith and Barnes (1983) found that more men than women drinkers experienced hangover but this could be due to differences in amounts consumed.

For the present study, we combined data from three randomized crossover studies we conducted on the effects of moderate/heavy alcohol administration (mean = .11g% BrAC) on next-day neurocognitive and/or simulated occupational performance. In two studies (Studies 1 and 2), participants were randomized to receive high-alcohol beer (7.3% alcohol); in the other study (Study 3) participants received (randomly) either bourbon or vodka. In our studies, participants rated their hangover on a scale ranging from "no hangover" to "incapacitating hangover". As Slutske et al. (2003) point out, this involves attribution to residual alcohol effects. However, in our studies, this item had the highest item-total correlation on a hangover scale and the highest validity (Rohsenow et al., in press). We also asked participants to rate, without attribution, several symptoms associated with hangover (Rohsenow, et al., in press) although these symptoms could occur in the absence of drinking. While participant populations, alcoholic beverages, and next-day performance measures differed across these studies, the same administration procedures, targeted BrAC levels, and participant eligibility criteria were used. Thus, these data allow us to compare three alcoholic beverages, at comparable mean BrACs, with respect to: (1) the percentage of drinkers who reported any hangover; (2) the frequency of hangover by severity; (3) frequency of specific hangover symptoms. We also could investigate the relationship of incidence of hangover reports to participant characteristics (age, gender, drinking practices, family history of alcohol problems). Our aims were: (1) to investigate the incidence of any hangover and of severity of hangover after a specific narrow BrAC range among nonalcoholic drinkers; and (2) to investigate congener content (bourbon vs. vodka) and participant differences as determinants of variability in hangover incidence.

Methods

Participants

In Study 1, participants were 54 professional merchant marine deck officers attending a recertification course at Kalmar Maritime Academy (Kalmar, Sweden) and in the other two studies participants were 118 university students or recent graduates recruited from greater Boston. All participants had to be at least 21 years of age and meet the following criteria: (1) no serious drinking problems (score < 5 on the Short Michigan Alcohol Screening Test [SMAST]) (Selzer et al., 1975) and no history of treatment or counseling for alcohol problems; (2) 5 or more drinks on a single occasion (4 if female) at least once in the 30 days prior to screening; (3) no health problems or current medication use contraindicated for alcohol; (4) fluent English; (5) recently graduated from, or currently attending, an institution of higher learning (Studies 2 and 3); and (6) negative pregnancy test and not nursing, if female. Each day, prior to beverage administration, participants who reported consuming alcohol, caffeine, prescription or over-the-counter drugs within the prior 24 hours, or food or

beverage within the prior 3 hours, were rescheduled. Participants presenting with a positive BrAC were excluded from further participation.

Study Design

The three studies included in this report used a double-blinded, within-subjects, crossover design (i.e., each subject acts as his/her own control). Thus, all the subjects included in these analyses received two beverage conditions (alcohol and placebo) in counter-balanced order. Participants took part in the studies over four days (two consecutive days followed a week later by two consecutive days).

Procedures

On the first evening, participants were randomized within gender to receive either the alcoholic beverage or an equivalent amount of placebo; on the second evening they received the other beverage. Participants were told that they had a 50% chance of receiving alcohol or no alcohol the first night and would receive the other beverage the other night; they were not debriefed about what they received until completion of the study.

Beverage administration procedures

Alcoholic beverage administration was designed to yield .10g% BrAC (1.2 g/kg body weight for men and 1.1 g/kg for women [Friel, 1999]). The target BrAC of .10 g% was based on early dose-response studies (e.g., Chapman, 1970) where the results indicated that achieving a BrAC of at least .10 g% was a more important determinant of hangover than fixing a g/kg dosage of alcohol and permitting BrAC to vary more widely. If participants randomized to alcohol did not reach .10 g% BrAC after their final scheduled drink, the ratio of actual to target BrAC was used to estimate an additional amount of beverage to administer. Consistent with blinding the beverage condition, the same number of placebo participants was given an extra drink in a matched quantity.

In Studies 1 and 2 the beverage alcohol was beer containing 7.3% alcohol (Elephant Beer®, Carlsberg A/S, 100 Ny Carlsberg, DK-1760, Copenhagen) and the placebo was Clausthaler® non-alcoholic beer (Radeberger Gruppe, Darmastaedter Landstrasse 185, Frankfurt, DEU). In study 3, the alcoholic beverages were bourbon (101 proof Wild Turkey ®) or 100 proof vodka (Absolut ®), mixed with chilled caffeine-free cola (Coke ®). The placebo for both of these beverages was caffeine-free cola beverage plus decarbonated tonic, in amount equivalent to the alcoholic beverage, chilled, with a few drops of vodka or bourbon floated on top.

Beverages were administered between the hours of 8:45 PM and 10:00 PM in Studies 2 and 3, between 7:30–9:00 PM in Study 1, in small groups of 3–5 participants. Double-blinding of the beverage was maintained; the research staff who prepared the beverages and conducted the breath tests were different from those who interacted with the participants when collecting all other measures.

After drinking and a 30-minute absorption period, participants completed subjective measures, received snacks and were escorted to rooms where they had 8 hours of bed rest, observed by nursing students (Study 1) or a licensed emergency medical technician (Studies 2 and 3).

In the morning, participants were awakened at 7:00 a.m., ate breakfast, were breath-tested. At 7:10–7:20 they completed the hangover ratings and at 8:00 am were administered other questionnaires followed by performance measures (these times were 1 hour earlier for Study 1). The time of day was chosen based on literature indicating that subjective effects of

hangover are most detectable before 10:00 a.m.. (Ylikahri et al., 1974) and that decrements in automobile driving performance the day after heavy drinking were seen only in the morning (Tornos and Laurell, 1991). Performance during the first 30 min or so after waking is likely to be impaired by sleep inertia (Tassi and Muzet, 2000) so allowing an hour before performance testing avoids confounding by this factor.

The studies included in these analyses were approved by the Institutional Review Boards at Boston University Medical Center and Brown University (Studies 1–3), and the University of Michigan (Studies 2 and 3).

Assessments

The morning following beverage administration, participants completed a nine-item Acute Hangover Scale (Rohsenow et al., in press) upon arising. They rated "hangover" on scales ranging from 0 (none) to 4 (moderate) to 7 (incapacitating), plus eight symptoms often associated with hangover: thirst, tiredness, headache, dizziness or faintness, nausea, stomach ache, heart racing, and loss of appetite, based on empirical work on hangover effects (Chapman, 1970; Ylikahri et al., 1974; Roehrs, Yoon and Roth, 1991). In this study, the data analyses for this study, the discrete symptoms other than "hangover" were coded as present or absent (positive rating vs. zero). Hangover severity was coded for severity as follows: 0 = none; 1–2 = mild; 3–5 = moderate; and 6–7 = severe.

To determine family history of alcohol problems, participants completed the interviewer-administered Family Tree Questionnaire developed by Mann, Sobell, Sobell and Pavan (1985). Anyone identifying a first- or second-degree relative with alcohol problems was coded as family-history positive.

Quantity and frequency of recent drinking was estimated using a two-item alcohol use questionnaire: 1) "Considering all your drinking times in the past 30 days, about how often did you have any beer, wine or liquor?", Likert-rated from 1 "once a day" to 7 "did not drink" with each point anchored; 2) "In the past 30 days, on a typical day that you drank, about how much did you have to drink in one day?", rated from 1 to 8, with choices of 1 to 7 drinks and "8 or more drinks". One drink was defined as 12 oz of beer or wine cooler, 4 oz of wine or 1 oz of liquor. Only quantity was used to test this hypothesized predictor of hangover.

Participants self-identified as smokers or nonsmokers. Smokers were enrolled only in Study 1. A few smokers were initially enrolled in Studies 2 and 3 but smoking became an exclusion criterion for Studies 2 and 3 because the site for these studies did not accommodate smoking and nicotine withdrawal symptoms could have been a confound to our study aims.

Results

Description of Participants

See Table 1 for participant characteristics. The peak mean BrAC was.11 g% (SD = .01; range = .09 -.15). There were significant differences across beverage types for sex (x^2 (2) = 31.04; $p < .0001$), but not for age, family history of drinking problems, drinking practices, smoking or peak BrAC.

Frequency and Severity of Hangover

In the placebo condition 3% (5) reported mild hangover. In the alcohol condition, 24.4% (42) of participants reported no hangover; 43.6% (75) reported mild hangover; and 31.9%

(55) reported moderate hangover. None reported severe hangover. Incidences of all symptoms were significantly greater for those reporting greater hangover severity levels. Frequency of each symptom by hangover severity level and chi square statistics is displayed in Table 2.

Hangover by Characteristics of Participants and Beverage Type

In univariate chi square analyses, there were no significant differences in the distribution of hangover severity by sex, age, family history of drinking problems, drinks per day, smoking status or beverage type (Table 3).

Discussion

The morning after a mean peak BrAC of .11 g%, after BrAC had been eliminated, none of these healthy drinkers reported severe hangover; 24% reported no hangover, and 76% reported mild to moderate hangover. Our results cannot be compared to most survey data because surveys do not control for the level of alcohol consumption and because they tend to use some unit of time (e.g., prior year) in the incidence measure. It is noteworthy, however, that in a sample of drinkers, Harburg et al. (1993) found that 23% reported never having had a hangover, regardless of how much they drank, consistent with our results. Similarly, in a sample of alcoholics admitted for detoxification, Pritach et al. (1983) also found that 23% reported never having had a hangover.

In several respects our findings were comparable to the one experimental study that reported the incidence of hangover after intoxication (Chapman, 1970). In his study, 29% of participants in groups with a comparable BrAC level (mean BrAC = 12.4 g%; range = .10-.15 g%), reported no hangover the morning after alcohol administration, none reported severe hangover, 41% reported mild hangover and 30% reported moderate hangover, using the same scale metric as in our study. The consistency in the results across our study and Chapman's (1970) indicates that the incidence and severity levels we obtained are probably reliable for this level of intoxication.

Congener differences between beverages did not account for differences in hangover severity. This is not consistent with Chapman's (1970) results finding that bourbon resulted in more people with moderate hangover (33%) than vodka did (3%), since 285 of our vodka drinkers reported moderate hangover.

The other individual characteristics we investigated as predictors of hangover level severity were all non-significant. The lack of difference in hangover incidence by sex is consistent with some other studies (Chapman, 1970; NESARC data; Richardson, Piasecki and Sher, 2004; Harburg et al., 1993). The sex difference reported by Smith and Barnes (1983) did not include significance tests and could have been confounded by differences in proportion of heavy drinkers. Age was not significantly associated with hangover incidence in our study. Although the range of ages in our study was from 21–57, 80% were under 30 years of age. Other studies have not reported on the relationship between age and hangover propensity.

Our finding of no relationship between self-reported usual drinking quantity and hangover incidence was inconsistent with the most of the survey literature, but we might not have been able to test this hypothesis given that only 3.5% of our participants were heavy drinkers. Alternatively, survey studies reviewed might have found a relationship because people who drink heavily more frequently have more opportunities to experience hangover.

The lack of relationship between smoking status and hangover intensity is inconsistent with preliminary survey results reported by Richardson, et al. (2004). It is possible that the survey

results were confounded by differences in peak BrAC achieved, given that smokers are usually heavier drinkers than nonsmokers (Istvan and Matarazzo, 1984). Alternatively, it is possible that smoking status only predicts how often hangovers occur in general but not severity or probability at a specific fixed BrAC.

Our finding of no association between hangover and family history of alcohol problems contrasts with those of several other investigators (Newlin and Pretorius, 1990; McCaul et. al., 1991; Span and Earleywine, 1999: Piasecki et. al., 2005), but are consistent with the findings of Earleywine (1993) and Richardson, Piasecki, and Sher. (2004). However, family history could have been confounded with individual differences in peak BrAC achieved by participants in survey studies; peak BrAC levels are not possible to control for in such studies. Only one of these previous studies involved experimental administration of alcohol (Span and Earleywine, 1999) and interestingly that study found that family history positive participants reported higher incidence of “hangover” following both alcohol and placebo administration, indicating that the symptom reports might not really be due to hangover. Family history, however, is a complex construct that potentially includes both genetic and environmental components. While it is not possible to disentangle these components in studies comparing people only by family history, genetic factors per se might be an avenue for further exploration. Any genetic variation that impacts the effects of alcohol on sleep, diuresis, hypoglycemia, vasodilation, or withdrawal could potentially influence hangovers through differential sleep disruption, dehydration, impact on blood sugar, headache or withdrawal, respectively. Similarly, variation in genes responsible for the metabolism of congeners or stages of ethanol metabolism could moderate hangover propensity.

Our study also adds information about the prevalence of various symptoms commonly associated with hangover at different levels of hangover severity. All of the symptoms were ones validated in previous experimental investigations of hangover and the set of symptoms form a highly reliable scale across several data sets (Rohsenow et al., in press). While most of the symptoms were far less frequently reported by those reporting no hangover than by those with mild or severe hangover, thirst and tiredness were reported by 85–90% of those who reported no hangover. This could indicate that these individuals were experiencing residual effects of alcohol but not attributing them to the drinking the night before, or it could mean that people commonly wake up tired and thirsty. Since the severity of these two symptoms differed between alcohol and placebo days at $p < .0001$ (Rohsenow et al., in press), they are likely to be valid symptoms but it may be that most people experience these the morning after drinking to intoxication whether or not they attribute the cause to hangover.

Limitations of our study include having only this one specific targeted BrAC level, not having a broader range of different drinking patterns (for safety reasons), having no measure of acquired tolerance to alcohol effects, and not having more putative predictors of hangover that we could have investigated. However, this study is one of the only ones to report levels of hangover severity after a controlled level of intoxication has been induced so it adds to a limited literature.

Our findings are consistent with previous research on: (1) the incidence of hangover in response to a moderate/heavy dose of alcohol; and (2) the absence of evidence that sex predicts hangover. Our findings differ from some but not all previous studies on an association between family history of alcohol problems and the propensity for hangover and thus underscore the need for research exploring genetic predictors of hangover incidence. The lack of significant individual difference predictors despite observed variation in hangover intensity also demonstrates the need for further research to determine individual differences that account for differing levels of hangover severity after about the same peak

BrAC level. In summary, our findings on the propensity for hangover, the findings from the previous experimental study (Chapman, 1970), and those from surveys of drinkers (Harburg et al., 1993; Pristach et al. 1983), suggest that 25 to 30% of drinkers may be resistant to hangover.

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Table 1

Participant Characteristics

	Beverage Studied			
	7.2% beer (n=93)	**Bourbon (n=36)**	**Vodka (n=43)**	**Total (n=172)**
Male*	72 (77.4%)	15 (41.7%)	13 (30.2%)	100 (58.1%)
Age				
Mean ± SD	28.9 ± 8.9	24.2 ± 2.6	24.2 ± 2.6	26.7 ± 7.2
Range	21–57	21–31	21–31	21–57
Family history of drinking problems	26 (28.3%)	8 (22.2%)	11 (25.6%)	45 (26.2%)
Drinking practices				
Heavy drinker (≥ 4 drinks/day)	5 (5.4%)	0 (0.0%)	1 (2.3%)	6 (3.5%)
Light to moderate drinker	88 (94.6%)	36 (100.0%)	42 (97.7%)	166 (96.5%)
Smokers	17 (18.3%)	3 (8.3%)	3 (7.0%)	23 (13.4%)

P < .0001

Table 2

Frequency of Hangover Symptoms by Hangover Severity

Symptoms associated with hangover	Hangover Severity			
	No hangover (n=42)	Mild (n=75)	Moderate (n=55)	X^2 (2 df)
Thirst	38 (90.5%)	75 (100%)	55 (100%)	8.44***
Tiredness	36 (85.7%)	73 (44.5%)	55 (100%)	12.4*
Headache	6 (14.3%)	43 (57.3%)	43 (78.2%)	39.87***
Dizziness/faintness	3 (7.1%)	28 (37.3%)	37 (67.3%)	36.29***
Loss of appetite	10 (23.8%)	28 (37.3%)	30 (54.6%)	9.68*
Stomach ache	3 (7.1%)	12 (16.0%)	22 (40.0%)	17.62***
Nausea	1 (2.4%)	12 (16.0%)	21 (28.2%)	20.44***
Heart racing	1 (2.4%)	8 (10.7%)	15 (27.3%)	13.49**

* p<.01

** p<.001

*** p<.0001

Table 3

Hangover Severity under Alcohol Condition by Participant Characteristics

Predictor Variables	Hangover Severity		
	No hangover (n=42)	Mild (n=75)	Moderate (n=55)
Sex			
Male	22 (22.0%)	44 (44.0%)	34 (34.0%)
Female	20 (27.7%)	31 (43.1%)	21 (29.2%)
Age			
21–25	28 (27.5%)	48 (47.0%)	26 (25.5%)
26–40	10 (19.6%)	22 (43.1%)	19 (37.3%)
> 40	3 (23,1%)	3 (23.1%)	7 (53.8%)
Family history positive for drinking problems Positive	12 (26.7%)	23 (51.1%)	10 (22.2%)
Drinking practices Heavy drinkers (≥ 4 drinks/day)	3 (50.0%)	2 (33.3%)	1 (16.7%)
Light to moderate drinker	39 (23.5%)	73 (44.0%)	54 (32.5%)
Use tobacco			
Yes	6 (26.0%)	8 (34.8%)	9 (39.1%)
Beverage type			
Beer	24 (25.8%)	43 (46.2%)	26 (28.0%)
Bourbon	7 (19.4%)	12 (33.3%)	17 (46.5%)
Vodka	11 (25.63%)	20 (46.5%)	12 (27.9%)

Note: All differences were non-significant.