HUMAN PSYCHOPHARMACOLOGY
Hum. Psychopharmacol Clin Exp (2012)
Published online in Wiley Online Library
(wileyonlinelibrary.com) **DOI**: 10.1002/hup.2225

Direct comparison of the cognitive effects of acute alcohol with the morning after a normal night's drinking

Adele McKinney[1]*, Kieran Coyle[1] and Joris Verster[2]

[1]*University of Ulster, Derry, Northern Ireland, UK*
[2]*Utrecht Institute for Pharmaceutical Sciences, Division of Pharmacology, Utrecht University, Utrecht, The Netherlands*

Objective The aim of this study was to compare performance measures after acute alcohol consumption (intoxication) with the performance the day after a normal night's drinking (hangover).
Methods Eighty-four social drinkers took part in two studies that followed a counterbalanced repeated measure design. Fifteen men and 33 women were tested the morning (09:00, 11:00 or 13:00 h) following normal/usual alcohol consumption and the morning after no alcohol consumption; the order of testing was counterbalanced. In a second study, 36 participants (18 men and 18 women) were tested after receiving alcohol to attain a blood alcohol concentration of 0.08%, and after no alcohol administration, the order of testing was counterbalanced. In both studies, participants completed a task battery of memory, reaction time and attention tasks.
Results Alcohol had no effect on the free recall task and the spatial attention task. Alcohol consumption, either acute or the next day, significantly affected reaction time, divided attention, selective attention and Stroop interference. The impairments during intoxication and hangover were of comparable magnitude. Performance on tasks of delayed recognition and irregular interstimulus reaction time was worse during hangover when compared with intoxication.
Conclusion It is evident that awareness needs to be raised that performance the morning after alcohol consumption is at the same level if not worse than when participants are at the legal limit for driving (0.08% blood alcohol concentration). Copyright © 2012 John Wiley & Sons, Ltd.

KEY WORDS—acute alcohol; hangover; cognitive performance

INTRODUCTION

Alcohol consumption, both at acute levels and during post intoxication when blood alcohol concentration (BAC) has reached 0, is known to produce effects on cognitive performance. There is a long history of research examining the acute effects of alcohol on mood and performance (for reviews, see Finigan and Hammersley, 1992; Tzambazis and Stough, 2000; Sullivan and Pfefferbaum, 2005). In contrast, it is only recently that the next-day effects of alcohol have attracted attention (for reviews, see Prat *et al*., 2008; Stephens *et al*., 2008; Ling *et al*., 2010).

The cognitive and performance impairments due to acute alcohol consumption have been extensively investigated by using laboratory-based experimental designs (for reviews, see Moskowitz and Robinson, 1987; Ferrara *et al*., 1994). Laboratory studies have revealed that acute alcohol consumption results in poorer memory performance (for reviews, see Curran and Weingartner, 2002; White, 2003), impairment in tasks of divided attention (Maylor *et al*., 1990) and impaired executive functions such as planning and decision making (Weissenborn and Duka, 2003; Geroge *et al*., 2005). Most research on the residual alcohol effects on cognitive performance has followed an experimental design (for reviews, see Prat, *et al*., 2008; Stephens *et al*., 2008; Ling *et al*., 2010; Verster *et al*., 2010). Experimental studies induce hangover in a laboratory setting and measure cognitive performance the morning after, when BAC is zero. Laboratory studies have revealed decreased performance in attention (Myrstein *et al*., 1980; Roehrs and Roth, 2001; Howland *et al*., 2010; Rohsenow *et al*., 2010) and skills related to driving and flying (Seppala *et al*. 1976; Laurell and Törnos, 1983; Tornros and Laurell, 1991; Verster, 2007). However, many other laboratory-based studies have not observed any next-day effects of alcohol on performance (Collins and Chiles, 1980; Lemon *et al*., 1993; Chait and Perry, 1994; Finigan *et al*., 1998; Rohsenow *et al*.,

*Correspondence to: A. McKinney, Life and Health Sciences, Magee, Derry, Northern Ireland, UK. E-mail: a.mckinney@ulster.ac.uk

Received 5 September 2011
Accepted 1 March 2012

2006; Kruisselbrink *et al.*, 2006). The absence of impairment in these studies often can be explained by a combination of easy tests of short duration and various methodological shortcomings (discussed in Verster *et al.*, 2003; Verster, 2008).

More recently, attention has been directed towards investigating alcohol in a naturalistic setting. It is important to note that there are various differences between controlled and naturalistic study designs and that these differences may have an impact on study outcome (Verster *et al.*, 2010). Firstly, the amount and type of beverage usually differ between the designs. Within experimental investigations of the effects of alcohol, it is usual that a fixed dose of alcohol is administered, which may be adapted for gender and weight to realize the same BAC level for each participant. Also, the type of beverage and pace of drinking are usually controlled by the investigator. The participants are not aware of the specific quantity, and it could be argued that this loss of personal control over quantity and beverage type could obscure the subjective experience of alcohol consumption. However, Myrsten (1971) revealed that the self-reported ratings of subjective intoxication closely resembled the measured BAC curve. Thus, subjective awareness closely mirrors the changes resulting from increasing BAC. Numerous other studies, which assessed the subjective state of intoxication, have revealed that participants can differentiate between alcohol and placebo or between different doses of alcohol (e.g. Hamilton *et al.*, 1984; Miller, 1984). Successful deception is consistently reported with doses up to 0.5 mL/kg. Because of the known physiological changes after alcohol consumption, it is difficult to deceive participants that alcohol has been consumed when in fact they have consumed a placebo. This raises the issue of the expectancy effects of alcohol being as strong as the effect of actual alcohol consumption. Lyvers and Maltzman (1991) concluded that a balanced placebo design investigating the effects of alcohol cannot independently evaluate effects of both alcohol and beverage instructions, when behaviourally significant doses of alcohol are administered.

A second major difference is that in naturalistic studies, various factors that may have an impact on hangover severity are not controlled by the investigator. For example, participants are allowed to smoke, dance, engage in other activities during drinking (e.g. playing darts) and consume different types of alcoholic beverages (with different congener contents) at their own drinking rate. These factors have been shown to influence the presence and severity of alcohol hangover symptoms (Piasecki *et al.*, 2010; Rohsenow *et al.*, 2010; Verster *et al.*, 2010). Because these are all natural behaviours, they are allowed in a naturalistic design. As a result, these studies have a high ecological validity; that is, in contrast to controlled laboratory studies, they accurately reflect real life drinking.

Results from naturalistic studies

Lyvers and Tobias-Webb (2010) investigated executive cognitive functioning in a social setting where alcohol consumption was self-regulated. A relationship between BAC and persisting errors in the Wisconsin Card Sorting Test was observed. The effects of acute alcohol consumption have also been investigated in the field; two studies (Tiplady and Degia, 2004; Degia *et al.*, 2006) have used portable testing equipment at music festivals where alcohol consumption is ubiquitous. Degia *et al.* (2006) revealed that alcohol impaired divided attention and sustained attention. Tiplady and Degia (2004) observed overall impairment on the hand-held two-choice tester, which is a prototype tester for detecting driver impairment at the roadside.

As with the investigation of acute alcohol consumption, attention has been directed towards investigating the effects of a naturally occurring hangover. Naturalistic studies have investigated the next-day effects of alcohol consumption, when the alcohol consumption (quantity, pace and environmental setting) has been under personal control (Mc Kinney and Coyle, 2004; Finnigan *et al.*, 2005; Mc Kinney and Coyle, 2006). The findings reveal poorer memory, psychomotor performance and attention the morning after alcohol consumption.

To the authors' knowledge, there are no direct comparisons of the acute and next-day effects of alcohol consumption on performance. Therefore, the present study was designed to facilitate a direct comparison between performance when participants have consumed a quantity of alcohol to raise their BAC to the legal limit for driving (0.08%) and the naturally occurring next-day effects of a 'usual' night's alcohol consumption. On the basis of previous research, it is expected that during the alcohol conditions (either acute or the next day), performance will be significantly worse than when in the no alcohol state. However, it is not known if performance with a BAC of 0.08% will differ from performance during the morning after a normal night's drinking, when BAC is zero.

Alcohol hangover develops after excessive drinking when BAC returns to zero and is characterised by a range of symptoms (i.e. thirst, fatigue, drowsiness, nausea and headache). In the present Design and Results sections, 'hangover' is used simply to refer to the morning after a normal night's alcohol consumption and not the hangover state *per se*.

METHOD

Participants

A total of 84 healthy volunteers were recruited for the study. Forty-eight volunteers (15 men and 33 women) were allocated to the next day 'hangover' study, and 36 volunteers (18 men and 18 women) were allocated to the acute study. Volunteers were non-pregnant social drinkers who drank on average six units 2.5 times per week. Participants were free to terminate the study without reason at any time. All participants were students recruited from the University of Ulster student accommodation building. Testing also took place in their accommodation building; hence, participants did not have to travel to the testing venue. Participants were given both written and verbal information about the nature of the study, and written informed consent was obtained. The study was approved by the research ethics board of the University of Ulster and was carried out in accordance with the Helsinki Declaration.

Design

Study 1. Each participant was tested twice; they carried out tasks the morning after a normal night's drinking (hangover) and the morning after no alcohol consumption. The two sessions were approximately one week apart, and the order of testing was counterbalanced.

Study 2. Each participant was tested after consuming alcohol to attain a BAC of 0.08% and also tested with no alcohol, neither hangover nor acute. The two sessions were approximately one week apart, and the order of testing was counterbalanced.

Participants in both studies were randomly allocated to an order of testing and a time of testing (09:00, 11:00 or 13:00 h). Time of testing was not expected to impact on the acute alcohol testing but was held constant between the acute and hangover study where testing in the morning was considered to be an important factor. BAC reaches zero around 08:00–11:00 h after consuming the amount of alcohol required for a hangover state to be experienced (Chapman, 1970; Ylikahri, *et al*., 1974; Rohsenow, *et al*., 2010). Participants in the present post intoxication study were required to stop drinking at 02:00 h, the first testing session occurred 8 h after alcohol consumption ended (09:00–10:00 h) and the last group were tested 11 h after the cessation of alcohol consumption (13:00–14:00 h).

The studies followed a mixed-factor, counterbalanced, repeated measures design with test condition (hangover/acute) and order of testing, either acute or hangover/no alcohol versus no alcohol/either acute or hangover, and time of testing (09:00, 11:00 or 13:00 h) as between-participants factors. The factor of state, alcohol condition (either acute or hangover) versus no alcohol condition, was a within-participants factor.

Procedure

Pretesting requirements. Participants were required to consume no alcohol for the 24 h preceding testing for the acute session and the no alcohol sessions. On all testing days, participants were required to eat their breakfast of choice at 08:00 h and not to consume any caffeinated beverages. When participants arrived for their test session, they confirmed compliance with the pretesting requirements. Following this, participants' blood alcohol levels were recorded using a Lion breath alcohol meter (Lion Laboratories Limited, Barry, UK), which had been calibrated prior to the study.

Hangover group. For the hangover session, participants informed the researcher on the day they intended to drink alcohol, and arrangements were made for testing the following morning. The counterbalanced repeated measures design used a naturalistic drinking environment to facilitate the investigation of participants' usual volume of consumption of preferred beverage in the chosen company. Participants were requested to consume their usual quantity and type of alcoholic drink between 22:00 and 02:00 h and to eat no food after alcohol consumption.

Acute study. The acute alcohol session began with the participant drinking vodka mixed with chilled, caffeine-free, diet Coke. The calculated quantity of ethanol administered was based on total body water, according to Watson *et al*. (1981). The drinks were made up of 2.13 mL vodka (37.5% alcohol by volume) per litre body water mixed with caffeine-free diet coke to a volume of 500 mL. The participants were instructed to finish the drink within 10 min. When the drink was finished, each participant completed a set of questionnaires. Approximately 20 min after the drink was completed, and prior to the first computerised task, a breath reading was taken. Subsequent breath readings were obtained at approximately 10-min intervals.

Task battery of subjective and objective measures. The participants completed questionnaires on demographic information, drinking practices, hangover signs and symptoms (Myrstein *et al*., 1980), hangover experiences (Newlin and Pretorius, 1990), and sleep quality and quantity (in hours). Questionnaires assessing mood (Herbert *et al*., 1976), anxiety (Spielberger *et al*., 1970), perceived stress (Cohen *et al*., 1983), cognitive interference (Sarason *et al*., 1986) and extroversion (Eysenck Personality Inventory, 1963) were also

DOI: 10.1002/hup

completed. Following the completion of the questionnaires, a battery of tasks, memory (immediate and delayed recall), reaction time, divided attention, selective attention, spatial attention and Stroop interference, were administered in a standard order.

Memory tasks. For the free recall task, 20 words, selected from cluster 7 of the Handbook of Semantic Word Norms (Toglia and Battig, 1978), were presented on a computer screen. Participants wrote down as many of the words as they could remember. The delayed recognition task, which was presented 60 min after the free recall task, comprised a list of 40 words, consisting of the 20 free recall list, previously presented, among 20 distracters. Participants were required to indicate any word that had been presented on the computer screen at the start of the task battery. The participants were given different word lists on each test occasion.

Reaction time tasks. The two simple reaction time tasks required participants to respond, by pressing the space bar, as quickly as possible when an 'X' appeared in the centre of the screen. In the regular reaction time task, the five blocks of 10 stimuli had a constant interstimulus interval of 2000 ms. In the irregular condition, the interstimulus intervals had increments of 500 ms within the range of 500–5000 ms. The 10 different interstimulus intervals were equally represented and randomized within each of the five blocks.

Attention tasks

Divided attention task (Tedstone and Coyle, 2004). Single digits were presented at a rate of 600 ms/item in the centre of the screen. The participant was required to respond to two even digits appearing consecutively and a solid square that randomly appeared in four positions (directly above, below, left and right) around the centrally positioned number task.

Selective Attention Test (Eriksen and Eriksen, 1974). The Eriksen Flanker Test was used as a selective attention task (Eriksen and Eriksen, 1974). The targets and distracters comprised the letters A and B, and distracters were compatible or incompatible with the target. The flankers appeared near (1.0 cm, 0.6° visual angle) the target or far (3.4 cm, 1.9° visual angle) from the target. Participants responded to the target by pressing the key 'A' for target A and the key 'L' for target B. At the beginning of each trial, the word 'READY' appeared in the centre of the screen. After 1000 ms, three asterisks appeared, indicating the position of the target and non-target. Following 500 ms, the spatial priming cues were replaced with the target and non-targets, which remained on screen until the participant made a response or the time limit of 2000 ms was reached.

The Stroop Test (Stroop, 1935). The Stroop Test was used to measure selective attention (Stroop, 1935). In this task, the word–colour card and colour–word card consist of a 4×28 matrix of words printed in incongruous colours (e.g. 'red' printed in blue ink). The response to the word–colour card is to read aloud the word; in contrast, the response to the colour–word card is to read aloud the colour of the ink the colour word is written in. The time taken to read the last 100 items on each card was recorded.

Spatial attention task (Broadbent et al., 1989). This task was developed by Broadbent *et al.* (1989) to measure aspects of selective attention and choice reaction time. Each trial started with the appearance of the word 'READY', which remained on the screen for 1000 ms. This was replaced by two asterisks (priming cues) spaced either 2.04 or 5.20 degrees apart. Following 500 ms, the spatial priming cues were replaced. One of the target letters, A or B, was presented alone in half the trials and was accompanied by the digit 5 in the other half. The participant did not know which of the asterisks the target would replace. Participants responded to the target by pressing the appropriate key. A left hand key press of the 'A' key was required for target A. A right hand key press of the 'L' key for the letter B. Half the trials led to compatible responses (i.e. the letter A on the left of the screen or the letter B on the right of the screen), whereas the others were incompatible (i.e. the letter A on the right of the screen or the letter B on the left of the screen). In half the trials, the priming cues were close together (2.04 degrees apart), and in the other half, the priming cues had a far spacing (5.20 degrees apart). Ten blocks of eight trials were presented.

Statistical analysis

Statistical analyses were performed using SPSS statistical Package (version 17). Analysis of variance for repeated measures was conducted to test for significant differences. The between-participants factors were alcohol group (laboratory-induced acute or naturalistic-induced hangover) and order of testing (alcohol (either acute or hangover)/no alcohol, no alcohol/alcohol (either acute or hangover)), and the repeated measures factor of State (alcohol condition (either acute or hangover) versus no alcohol condition) were tested for significance. Only main effects and interactions with alcohol group and

DOI: 10.1002/hup

state were considered. The reported number of drinks was converted to units by the researcher.

RESULTS

The sample of participants in the acute intoxication group did not differ significantly from the sample of participants in the hangover group on age and drinking characteristics (Table 1).

None of the personality scales revealed significant differences between the two groups, except for extroversion ($t(69) = 2.07$, $p < 0.05$) acute group mean 15.12, SD 3.14 and hangover group mean 13.59, SD 3.05. Partial correlational analysis revealed no relationship with any measure of task performance in the task battery.

Blood alcohol concentration after controlled administration of alcohol

The mean peak BAC was 40.73 μg/dL (SD 11.63) with a range of 25–65, that is, 0.081% BAC. The mean time to consume the beverage, 13.14 min (SD 5.69) with a range of 5–35min, was longer than the requested 10 min per drink. However, drink time was not significantly correlated with the peak BAC obtained ($r = -0.015$, $p > 0.05$) and will not be considered further. No gender differences were observed between time taken to consume the beverage ($t(34) = 1.714$, $p > 0.05$) and peak BAC ($t(31) = -0.722$, $p > 0.05$).

Hangover group

The night before testing, participants drank on average 11.84 (SD 7.72) units of alcohol (range 3–24.5 units). Men reported consuming on average 14.7 (SD 3.76) units, and females, 10.5 (SD 7.14) units the night before testing. All BACs were zero the morning after participants' normal alcohol consumption, except for two participants who had low readings of 50 mg/L on arrival. Participants allocated to the three testing times and two orders did not differ on units per occasion and units consumed the night before testing; these drinking variables did not differ between men and women. Participants reported their sleep as being less satisfying, less restful and less refreshing the morning after alcohol consumption; there was no difference in how good or how deep participants found their sleep the morning after alcohol consumption (see Mc Kinney and Coyle, 2006). Each sleep quality index obtained in the hangover and no hangover conditions was entered into a bivariate correlation with the performance measures. This failed to reveal any significant relationship between reported sleep quality and performance on tasks. The quantity of sleep the night before both testing sessions failed to show any relationship with any aspect of performance on the cognitive tasks.

Analysis of alertness and tranquillity

The participants were significantly ($F(1, 76) = 48.16$, $p < 0.001$) less alert the morning of the alcohol session (either acute or hangover), mean 561.19 (SD 188.11), compared with the morning of the no alcohol condition, mean 375.21 (SD 180.58). The main effect of alcohol group (acute versus hangover) reached significance ($F(1, 76) = 10.06$, $p < 0.001$) as did the interaction alcohol group by alcohol condition ($F(1, 76) = 17.17$, $p < 0.001$), indicating that participants were significantly less alert during the morning after a normal night's drinking (mean 640.00, SD 160.42) compared to the rising limb of the blood alcohol curve (mean 442.97, SD 164.65).

The participants were significantly ($F(1, 75) = 9.05$, $p < 0.01$) less tranquil the morning of the alcohol session (either acute or hangover) (mean 224.62, SD 110.01) compared with the morning of the no alcohol condition (mean 173.20, SD 89.5). The main effect of alcohol group (acute versus hangover) reached significance ($F(1, 75) = 7.54$, $p < 0.01$). The significant interaction of alcohol group by alcohol condition (either acute or hangover) ($F(1, 75) = 14.82$, $p < 0.001$)

Table 1. Age and drinking variables of participants allocated to either the acute alcohol group or the hangover group

	Acute alcohol group ($N = 36$) Mean (SD)	Hangover group ($N = 48$) Mean (SD)	t value p value
Age	24.50 (5.77)	23.38 (5.26)	0.93 $p = 0.35$
Frequency of alcohol consumption per week	2.47 (0.654)	2.42 (0.647)	0.88 $p = 0.69$
Usual quantity of alcohol per occasion	5.82 (2.73)	5.37 (2.27)	00.52 $p = 0.61$
Hangover experience	33.03 (10.901)	33.73 (8.15)	−0.34 $p = 0.74$

DOI: 10.1002/hup

indicates that participants were significantly less alert during the morning after a normal night's drinking (mean 263.35, SD 103.141) compared with when at acute alcohol intoxication (mean 164.65, SD 93.16) or no alcohol conditions.

Cognitive and psychomotor tests

An overview of the results is presented in Table 2.

Memory tasks

The results of the free recall task revealed no significant differences between the no alcohol session and the alcohol sessions nor any differences between acute versus hangover.

Performance on the delayed recognition task revealed a significant main effect of alcohol ($F(1, 80) = 10.16$, $p < 0.005$, η^2 0.113), indicating poorer recognition in the alcohol condition (mean 13.00, SD 3.036) compared with the no alcohol condition (mean 14.11, SD 3.00). The significant main effect of alcohol group (acute versus hangover) ($F(1, 80) = 6.04$, $p < 0.01$, η^2 0.07) indicates higher recognition in the acute group (mean 14.25, SD 2.54) compared with the hangover alcohol group (mean 12.82, SD 2.67). The lack of interaction between alcohol group and alcohol condition, when the two main effects reached significance, may indicate group differences. To elucidate this, the difference between the acute and hangover groups was further analysed by looking at the difference between the alcohol-related performance and the no alcohol-related performance and comparing the acute participants with the hangover participants.

Analysis of the difference scores (acute intoxication — acute group no alcohol performance) versus (hangover — hangover group no hangover performance) revealed no significant difference, $t(82) = 0.219$, $p > 0.05$.

Reaction time tasks

Results from the regular reaction time task are depicted in Figure 1. Performance on the regular interstimulus reaction time task revealed significantly ($F(1, 79) = 8.98$, $p < 0.005$, η^2 0.102) slower reaction time when in the alcohol condition (either hangover or acute intoxication), mean 267.66 (SD 6.46), compared with the no alcohol condition, mean 249.10 (SD 4.34). The main effect of alcohol group ($F(1, 79) = 12.46$, $p = 0.001$, η^2 0.136) reached significance, indicating slower reaction time for the hangover group (mean 274.48, SD 49.10) compared with the acute group (mean 242.29, SD 25.58). The interaction of alcohol condition and alcohol group ($F(1, 79) = 4.02$, $p < 0.05$, η^2 0.048) revealed that reaction times were significantly ($t(70.57) = 3.74$, $p < 0.001$) slower in the morning after naturalistic alcohol consumption (mean 311.95, SD 82.69) compared with the laboratory-induced acute consumption (mean 261.20, SD 38.89).

Table 2. Results of the cognitive and psychomotor tests

	Comparing the alcohol state with the no alcohol state			Comparing the acute state with the hangover state		
	Alcohol condition (either acute or hangover)	No alcohol condition	F value significance level	Hangover	Acute	Hangover/acute
Task	$N = 84$ Mean (SE)	$N = 84$ Mean (SE)	$N = 48$ Mean (SE)	$N = 36$ Mean (SE)		
Delayed recognition	13.00 (0.34)	14.07 (0.32)	10.16**	12.82 (0.38)	14.25 (0.44)	6.05*
Regular interstimulus reaction time	286.90 (7.61)	262.11 (4.83)	10.98***	293.51 (6.79)	255.45 (7.76)	13.58**
Irregular interstimulus reaction time	360.52 (7.47)	333.00 (4.19)	14.92***	352.82 (6.42)	340.70 (7.41)	ns
Stroop	76.46 (1.28)	73.62 (1.37)	8.02**	74.16 (1.57)	75.95 (1.90)	ns
Divided attention	487.87 (6.48)	476.25 (6.15)	4.33*	486.21 (7.25)	477.92 (8.71)	ns
Selective attention state × distance	467.90 (73.08)	450 (72.14)	4.27*	–	–	ns

Comparing the alcohol condition (either acute or hangover) with the no alcohol condition and comparing acute with hangover when there was a significant difference due to alcohol (either acute or hangover).
*$p < 0.05$, **$p < 0.01$ and ***$p < 0.001$.

DOI: 10.1002/hup

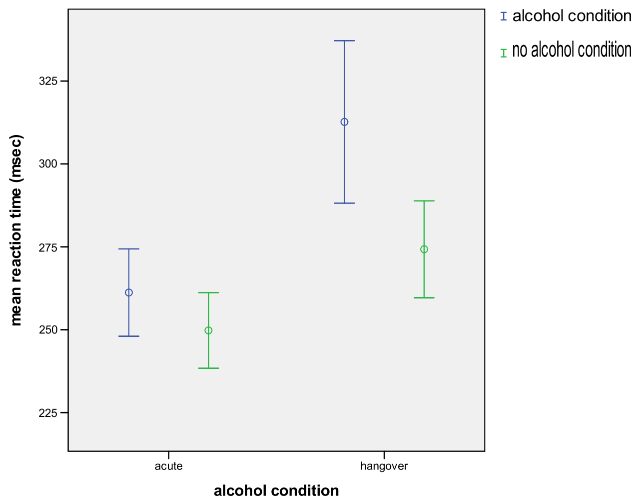

Figure 1. The mean reaction time (ms) for the regular interstimulus reaction time task, during the alcohol and no alcohol conditions, for both the acute and hangover alcohol groups

Analysis of the mean irregular reaction time also revealed a main effect of alcohol ($F(1, 80) = 14.92$, p 0.001, η^2 0.157), indicating slower reaction time when in the alcohol condition (either hangover or acute intoxication), mean 360.52 (SD 69.53), compared with the no alcohol condition, mean 333.00 (SD 38.85). Hangover and acute performance did not differ in this task.

Attention tasks

The results of the spatial attention task revealed no significant differences between acute versus hangover, nor any differences between the no alcohol session and the alcohol session.

Divided attention

The divided attention task yielded a main effect of alcohol ($F(1, 74) = 4.33$, $p < 0.05$, η^2 0.055), revealing slower reaction time in the alcohol condition, mean 488.81 (SD 56.27), compared with the no alcohol condition, mean 476.67 (SD 54.05). Hangover and acute performance did not differ in this task.

Stroop

Results indicated a significant main effect of alcohol condition (no alcohol/alcohol (acute or hangover)), $F(1, 77) = 8.02$, $p < 0.01$, η^2 0.094. The no alcohol condition (mean 73.04, SD 12.97) was significantly faster than the alcohol condition (mean 76.27, SD 11.80). The first-order interaction of alcohol by Stroop ($F(1, 77) = 5.708$, $p < 0.05$, η^2 0.102) indicates that alcohol had no effect on the reading task ($p > 0.05$) but caused a slowing of the colour word task ($t(80) = -3.36$, $p < 0.001$). Performance did not differ between acute and hangover states.

Selective attention task

The observed alcohol × distance interaction ($F(1, 74) = 4.27$, $p < 0.05$, η^2 0.055) revealed that alcohol consumption (both acute and hangover) caused significantly ($t(77) = -2.234$, $p < 0.05$) slower response to far distractors (mean 467.90, SD 73.08) compared with the no alcohol condition (mean 450.50, SD 72.14) as illustrated in Figure 2. The difference did not reach significance for the near distractors (Figure 2.) Performance during acute intoxication did not differ from performance the morning after a normal night's drinking on this task.

DISCUSSION

This study showed that performance impairment during alcohol hangover, when BAC has returned to zero, is similar to the effects seen with a BAC of 0.08%, the UK legal limit for driving a car. Cognitive and psychomotor performance is similarly impaired after both acute alcohol consumption and post alcohol consumption the morning after a normal night's drinking. The strength of the current study was the explicit comparison of acute and next day performance on memory, reaction time and tests of attention. The results indicate that different types of tasks are differentially sensitive to alcohol; however, the effects are similar during both the acute and hangover states. In the present study, the same pattern of deficits emerged for both the state of acute intoxication and during the morning after a normal night's alcohol consumption on tasks of attention. Acute and next day performance differed from each other on the delayed recognition task and in the regular interstimulus interval task, with next day performance being more affected than acute.

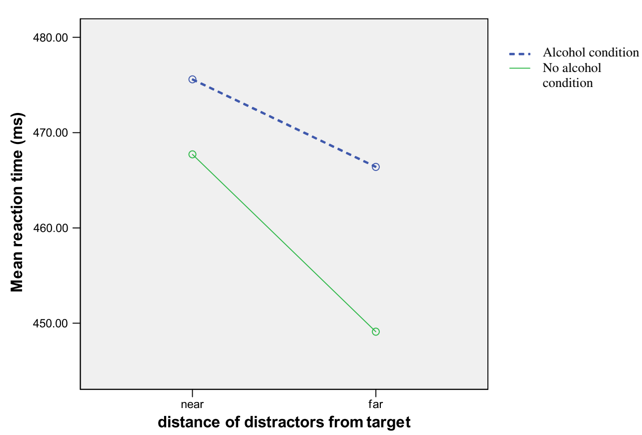

Figure 2. The mean reaction time for response to target flanked with near and far distractors in both the alcohol and no alcohol conditions

The detrimental effects of alcohol on memory, vigilance, attention and planning capacity are well established (Maylor *et al*., 1990; Curran and Weingartner, 2002; Weissenborn and Duka, 2003; White, 2003; Geroge *et al*., 2005); in contrast, the next-day effects of alcohol are less clear. Affectation has been observed in more recent studies (Verster, *et al*., 2003; Mc Kinney and Coyle, 2004; Finnigan *et al*., 2005; Mc Kinney and Coyle, 2006); however, no impairment has also been observed (Bowden, Walton, and Walsh, 1988; Lemon *et al*., 1993; Chait and Perry, 1994; Finigan *et al*., 1998; Rohsenow *et al*., 2006). In these studies, there exist several methodology differences, including dose of ethanol administered, time of cognitive function assessment after ethanol consumption and task used. The present study employed a naturalistic design and observed that the real life hangover results in the same performance decrements as observed when at the legal limit for driving on tasks of reaction time, divided attention, selective attention and Stroop interference.

Tasks that indicated performance at a lower level during the morning after alcohol consumption compared with when at the legal limit for driving were a simple reaction time task and a delayed recognition task. The reaction time tasks employed in the present investigation were employed by Smith *et al*. (1995). The results obtained in the present study support the findings of Smith *et al*. (1995) in that the irregular interstimulus task was sensitive to the effects of acute alcohol and the regular interstimulus task was not; however, the present study revealed that the regular interstimulus task was also sensitive to the next-day effects of alcohol. The second task that showed a differential effect between acute and next day performance was the delayed recognition task. It was observed that alcohol had no effect on the immediate free recall task but had a detrimental effect on the delayed recognition task. This supports and extends the findings that intoxicated subjects are typically able to repeat new information immediately after its presentation (see Ryback, 1971, for an early review). In contrast, alcohol impairs the ability to store information across delays longer than a few seconds if they are distracted between presentation and testing (for a review, see White, 2003). The present results are in line with those of other alcohol hangover studies that failed to find significant impairment on tests of immediate free recall (Chait and Perry, 1994; Verster *et al*., 2003) but observed an effect on delayed recognition (Verster *et al*., 2003), thus suggesting that both hangover and acute alcohol intoxication have similar effects on the stages of memory processing, alcohol primarily affects the transfer of information from short-term to long-term memory and this process is disrupted more during the morning after alcohol consumption compared with when at a BAC of 0.08%.

The present study found no significant relationship between sleep the night before and performance on tasks of memory, attention and reaction time. This can be seen to support the finding by Rohsenow *et al*. (2010) that alcohol effects on sleep correlated with hangover but did not mediate the effects on performance.

A potentially significant limitation of the present study is the use of standardized psychological tasks that may not translate to the real world. There is a need to compare the acute and hangover effects on tasks related to real world activities such as driving and on-the-job performance. There is also the issue of expectancy effects that needs to be taken into consideration in all alcohol research. The present study employed non-blind testing to facilitate as natural an investigation as possible. In hangover research, achieved BAC levels are of such magnitude that blinding is very difficult because subjects are familiar with and will experience at least some alcohol intoxication effects. It is, therefore, a matter of debate: is adequate blinding wanted or needed (Verster *et al*. 2010). The present paper was not concerned with gender differences, but rather was high in ecological validity and investigated cognitive and psychomotor performance of both men and women as experienced the morning after a normal night's drinking in comparison with performance on the same tasks when at peak blood alcohol. It may be of interest to future research to investigate any gender differences in performance due to the habitual alcohol consumption levels that appear to be increasing for women to a similar level as men. There is the issue that impulsive personality traits are related to performance on tasks of executive functions (Dolan, Bechara and Nathan, 2008) and that impulsivity may be one factor in the experiences of hangover, possibly because impulsive individuals are less adept at regulating their drinking (Field *et al*., 2010; Robertson & Piasecki, 2011) and subsequently consume larger amounts of alcohol. The use of naturalistic designs in investigating alcohol-related performance should take these personality characteristics into account especially when investigating executive function.

In conclusion, the effect on cognition during naturalistic hangover appears to have a similar pattern as those observed at acute alcohol consumption to the level allowed to drive a car. Thus, there is a need to raise awareness that the performance decrements evident at acute alcohol intoxication may last beyond the period in which alcohol is detectable in the breath and that caution should be taken when in the hangover state and carrying out everyday tasks (e.g. driving a car).

DOI: 10.1002/hup

CONFLICT OF INTEREST

No conflict of interest declared.

REFERENCES

Bowden SC, Walton NH, Walsh KW. 1988. The hangover hypothesis and the influence of moderate social drinking on mental ability. *Alcohol Clin Exp Res* **12**: 25–29.

Broadbent DE, Broadbent MHP, Jones JL. 1989. Time of day as an instrument for the analysis of attention. *Eur J Cognit Psychol* **1**: 69–94.

Chait LD, Perry JL. 1994. Acute and residual effects of marijuana, alone and in combination, on mood and performance. *Psychopharmacology* **115**: 340–349.

Chapman LF. 1970. Experimental induction of hangover. *Q J Stud Alcohol* **5**: 67–86.

Cohen S, Kamarck T, Mermelstein R. 1983. A global measure of perceived stress. *J Health Soc Behav* **24**: 385–396.

Collins WE, Chiles WD. 1980. Laboratory performance during acute alcohol intoxication and hangover. *Hum Factors* **22**: 445–462.

Curran HV, Weingartner H. 2002. Psychopharmacology of memory. In *The Handbook of Memory*, Baddeley A, Wilson B, Kopelman M (eds). Wiley: New York; 123–141.

Degia A, Meadows R, Johnsen S, Dixon P, Hindmarch I, Boyle J. 2006. Investigation into the suitability of a portable psychometric device to be used in the field: an illicit drugs field investigation. *J Clin Forensic Med* **13**: 242–246.

Dolan SL, Bechara A, Nathan PE. 2008. Executive dysfunction as a risk marker for substance abuse: the role of impulsive personality traits. *Behavioral Sciences and the Law* **26**: 799–822.

Eriksen BA, Eriksen CW. 1974. Effects of noise letters upon identification of target in a non-search task. *Percept Psychophys* **16**: 143–149.

Ferrara SD, Zancaner S, Giorgetti R. 1994. Low blood alcohol concentrations and driving impairment. A review of experimental studies and international legislation. *Int J Legal Med* **4**: 169–177.

Field M, Wiers R, Christiansen P, Fillmore M, Verster JC. 2010. Acute alcohol effects on inhibitory control and implicit cognition: implications for loss of control over drinking. *Alcohol Clin Exp Res* **34**(8): 1346–1352.

Finigan F, Hammersley RH. 1992. Effects of alcohol on performance. In *Handbook of Human Performance. Health and Performance*, Vol. 2, Jones DM, Smith AP (eds). Academic Press: London; 73–126.

Finigan F, Hammersley RH, Cooper T. 1998. An examination of next-day hangover effects after a 100 mg/100 ml dose of alcohol in heavy social drinkers. *Addiction* **12**: 1829–1838.

Finnigan F, Schulze D, Smallwood J, Helander A. 2005. The effects of self-administered alcohol-induced ‘hangover’ in a naturalistic setting on psychomotor and cognitive performance and subjective scale. *Addiction* **100**: 1680–1689.

Geroge S, Rogers RD, Duka T. 2005. The acute effect of alcohol on decision making in social drinkers. *Psychopharmacology* **182**: 160–169.

Hamilton MJ, Bush MS, Peck AW. 1984. The effect of bupropion, a new antidepressant drug, and alcohol and their interaction in man. *Eur J Clin Pharmacol* **27**: 75–80.

Herbert M, Johns MW, Dore C. 1976. Factor analysis of analogue scales measuring subjective feelings before sleep and after sleep. *Br J Med Psychol* **49**: 373–379.

Howland J, Rohsenow DJ, Greece J, et al. 2010. The effects of binge drinking on college students’ next-day academic test-taking performance and mood state. *Addiction* **105**: 655–665.

Kruisselbrink LD, Martin KL, Megeney M, Fowles JR, Murphy RJL. 2006. Physical and psychomotor functioning of females the morning after consuming low to moderate quantities of beer. *J Stud Alcohol* **67**: 416–420.

Laurell H, Törnos J. 1983. Investigation of alcoholic hangover effects on driving performance. *Blutalkohol* **20**: 489–499.

Lemon J, Chester G, Fox A, Greeley J, Nabke C. 1993. Investigation of the “hangover” effects of an acute dose of alcohol on psychomotor performance. *Alcohol Clin Exp Res* **17**: 665–668.

Ling J, Stephens R, Heffernan HM. 2010. Cognitive and psychomotor performance during alcohol hangover. *Curr Drug Abuse Rev* **3**: 80–87.

Lyvers M, Maltzman I. 1991. Selective effects of alcohol on Wisconsin Card Sorting Test performance. *Br J Addict* **86**: 399–407.

Lyvers M, Tobias-Webb J. 2010. Effects of acute alcohol consumption on executive cognitive functioning in naturalistic settings. *Addict Behav* **35**: 1021–1028.

Maylor EA, Rabbitt PM, James GH, Kerr SA. 1990. Comparing the effects of alcohol and intelligence on text recall and recognition. *Br J Psychol* **81**: 299–313.

Mc Kinney A, Coyle K 2004. Next day effects of a normal night’s drinking on memory and psychomotor performance. *Alcohol Alcohol*. **39**. 509–513.

Mc Kinney A, Coyle K. 2006. Alcohol hangover effects on measure of affect the morning after a normal night’s drinking. *Alcohol Alcohol* **41**: 54–60.

Miller RC. 1984. The effect of low doses of alcohol on human behaviour. PhD Thesis, University of Texas at Austin. Ref from Keolega HS. 1995.

Moskowitz H, Robinson CD. 1987. Effects of low doses of alcohol on driving related skills: a review of the evidence. Tech Rep US Dept Transp, Washington DC.

Myrstein AL, Rydberg U, Idstrom CM, Lamble R. 1980. Alcohol intoxication and hangover: modification of hangover by chlormethiazole. *Psychopharmacology* **69**: 117–125.

Myrsten AL. 1971. Effects of alcohol on psychological functions: experimental studies on non-alcoholic subjects. *Reports from the Psychological Laboratories University of Stockholm. Supplement 7.*

Newlin D, Pretorius M. 1990. Sons of alcoholics report greater hangover symptoms than sons of non-alcoholic: a pilot study. *Alcohol Clin Exp Res* **14**: 713–716.

Piasecki TM, Robertson BM, Epler AJ. 2010. Hangover and risk for alcohol use disorders: existing evidence and potential mechanisms. *Drug Abuse Rev* **3**: 92–102.

Prat G, Adan A, Pérez-Pàmies M, Sànchez-Turet M. 2008. Neurocognitive effects of alcohol hangover. *Addict Behav* **33**: 15–23.

Robertson BM, Piasecki TM. 2011. Exploring the relation between impulsivity and alcohol hangover: evidence from survey and diary measures. 34th Annual Scientific Meeting of the Research-Society-on-Alcoholism.

Roehrs T, Roth T. 2001. Sleep, sleepiness, and alcohol use. *Alcohol Res Health* **25**: 101–109.

Rohsenow DJ, Howland J, Minsky SJ, Arnedt JT. 2006. Effects of heavy drinking by maritime academy cadets on hangover, perceived sleep, and next day ship power plant operation, *J Stud Alcohol* **67**: 406–415.

Rohsenow DJ, Howland J, Arnedt JT, et al. 2010. Intoxication with bourbon versus vodka: effects on hangover, sleep, and next-day cognitive performance in young adults. *Alcohol Clin Exp Res* **34**: 509–518.

Ryback RS. 1971. The continuum and specificity of the effects of alcohol on memory: a review. *Q J Stud Alcohol* **32**: 995–1016.

Sarason IG, Sarason BR, Keefe DE, Hayes BE, Shearin EN. 1986. Cognitive interference: situational determinants and traitlike characteristics. *J Pers Soc Psychol* **51**: 215–226.

Seppala T, Leino T, Linnoila M, Huttumen M, YlikahrI R. 1976. Effects of hangover on psychomotor skills related to driving: modifications by fructose and glucose. *Acta Pharmacol Tox* **38**: 209–218.

Smith AP, Whitney H, Thomas M, Perry K, Brockman P. 1995. Effects of regular alcohol intake and stress on mental performance, mood and cardiovascular function. *Hum Psychopharmacol* **10**: 423–431.

Spielberger CD, Gorsuch RL, Lushene RE. 1970. *The State Trait Anxiety Inventory (Test Manual)*. Consulting Psychologists Press: Palo Alto, CA.

Stephens R, Ling J, Heffernan TM, Heather N, Jones K. 2008. A review of the literature on the cognitive effects of alcohol hangover. *Alcohol Alcohol* **43**: 163–170.

Stroop JR. 1935. Studies of interference in serial verbal reactions. *J Exp Psychol* **18**: 643–662.

Sullivan EV, Pfefferbaum A. 2005. Neurocircuitry in alcoholism: a substrate of disruption and repair. *Psychopharmacology* **180**: 583–594.

Tedstone D, Coyle K. 2004. Cognitive impairments in sober alcoholics: performance on selective and divided attention tasks. *Drug Alcohol Depend* **75**: 277–286.

Tiplady B, Degia A. 2004. Evaluation of tests of driver Impairment suitable for roadside use. http://www.icadts.org/T2004/pdfs/O111.pdf

Toglia MP, Battig WF. 1978. *Handbook of Semantic Word Norms*. Erlbaum: Hillsdale, NJ.

Tornros J, Laurell H. 1991. Acute and hang-over effects of alcohol on simulated driving performance. *Blutalkohol* **28**: 24–30.

Tzambazis K, Stough C. 2000. Alcohol impairs speed of information processing and simple and choice reaction time and differentially impairs higher-order cognitive abilities. *Alcohol Alcohol* **35**(2): 197–201.

Verster JC. 2007. Alcohol hangover effects on driving and flying. *Int J Disabil Hum Dev* **6**(4): 361–367.

Verster JC. 2008. The alcohol hangover: a puzzling phenomenon. *Alcohol Alcohol* **43**: 1–3.

Verster JC, van Duin D, Volkerts ER, Schreude AH, Verbaten MN. 2003. Alcohol hangover effects on memory functioning and vigilance performance after an evening of binge drinking. *Neuropsychopharmacology* **28**: 740–746.

Verster JC, Stephens R, Penning P, et al. 2010. The Alcohol Hangover Research Group consensus statement on best practice in alcohol hangover research. *Curr Drug Abuse Rev* **3**: 116–126.

Watson PE, Watson ID, Batt RD. 1981. Prediction of blood alcohol concentrations in human subjects: updating the Widmark equation. *J Stud Alcohol* **42**: 547–556.

Weissenborn R, Duka T. 2003. Acute alcohol effects on cognitive function in social drinkers: their relationship to drinking habits. *Psychopharmacology* **165**: 306–312.

White AM. 2003. What happened? Alcohol, memory blackouts, and the brain. *Alcohol Res Health* **27**: 186–196.

Ylikahri RH, Huttunen MO, Eriksson CJP, Nikkilä EA. 1974. Metabolic studies on the Pathogenesis of Hangover. *Eur J Clin Invest* **4**: 93–100.

DOI: 10.1002/hup