Effects of a Moderate Evening Alcohol Dose. II: Performance

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Background: This second of a pair of papers investigates the effects of a moderate dose of alcohol and staying up late on driving simulation performance and simple visual reaction time (RT) at a known circadian phase in well-rested young adults.

Methods: Twenty-nine adults (9 males), ages 21 to 25 years, spent 1 week on an at-home stabilization schedule of 8.5 to 9 hours, followed by 3 nonconsecutive nights in-lab: adaptation, placebo, and alcohol. Performance task practice occurred on 3 occasions before the study. Alcohol (vodka; 0.54 g/kg men; 0.49 g/kg women mixed with tonic) was consumed over 30 minutes ending 1 hour before normal bedtime; the same quantity of beverage was given on placebo. Driving simulation (with drive-only and dual-task drive and subtract components) and psychomotor vigilance task (PVT) testing occurred before and after alcohol/placebo ingestion. Breath alcohol concentration (BrAC) readings were taken before all test sessions. Saliva samples were taken approximately every 30 minutes to determine circadian phase.

Results: Driving simulation and PVT variables significantly deteriorated with increasing time awake. Driving simulator lane variability was worse with alcohol compared with placebo at 15.5 hours awake. No PVT variable showed an effect of alcohol.

Conclusions: Driving simulation performance deteriorated with extended waking and with alcohol; driving was most impaired at the peak alcohol level. The PVT, less complex than the driving simulation, did not show effects of alcohol, a finding consistent with previous literature that disruptive effects of low alcohol concentrations increase with task complexity. Overall, simulated driving performance is significantly impaired late at night when even a moderate dose of alcohol is consumed.

Key Words: Extended Waking, Alcohol, Driving Simulation, Reaction Time, Circadian Timing.

REPORTS OF DRIVING fatalities and injuries due to sleepiness or to alcohol have been well documented, and the risk of accidents from each is the greatest between the hours of midnight and 3:00 AM (National Center for Statistics, 2004). The interactive effects of staying up late and consuming alcohol, specifically low, legal doses of alcohol, are less clear. This is the second of 2 papers examining this issue, here investigating the effects of a moderate dose of evening alcohol and staying up late on simulated driving performance and visual reaction time (RT). A companion paper (Effects of a Moderate Alcohol Dose. I: Sleepiness) describes the effects of evening alcohol on sleepiness measures from the same study.

The 2-process model of sleep (described also in Part I) includes Process S, an exponential function reflecting sleep homeostasis, and Process C, a sinusoidal function reflecting the influence of the circadian rhythm (Borbély and Achermann, 1992). Process S is generally conceived of as a simple reservoir in which performance capacity increases exponentially during sleep and decays during wakefulness. Johnson et al. (2004), however, argue for a modulated concept of Process S that includes its capacity as varying as a function of the amount of sleep obtained during many previous days. This modulated model highlights the importance of controlling sleep for many days before experimental manipulation, as was done in the current study.

Current laboratory research on sleep deprivation and performance increasingly distinguishes between paradigms of sleep restriction versus extended waking. According to Van Dongen et al. (2003), "cumulative sleep loss and cumulative wake extension are different constructs that can have different quantitative values depending on the manner in which sleep loss occurs. The build-up of neurobehavioral deficits may not be caused by reduction of sleep time per se, but rather by excessive wakefulness beyond a maximum duration during which stable wakefulness can be maintained." "Excess wakefulness" is

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defined as waking time beyond a hypothetical critical period of about 16 hours (Van Dongen et al., 2003). The effects on performance of extended waking and of extended waking with alcohol are the focus of the current study.

Most sleep-focused studies of the effects of alcohol on driving simulation and RT have examined sleep restriction rather than wake extension (see, Rupp et al., Part I, for additional description). The few studies that used a wake-extension paradigm have focused on comparative effects of extended waking and alcohol consumption independently. For example, one study (Dawson and Reid, 1997) used a computer-tracking task (test of hand–eye coordination) to compare impairment secondary to alcohol with impairment produced by sustained wakefulness. Their results equated performance levels after 19 hours of sustained wakefulness (3:00 AM) to performance impairment seen with a breath alcohol concentration (BrAC) of 0.05%.

In a similar study, Williamson and Feyer (2000) examined the effects of extended waking or alcohol on a range of measures, including tasks involving cognitive and motor speed, accuracy, coordination, and attention. When awake for 17 to 19 hours, performance levels were judged low enough to be incompatible with safe driving. The deficit occurred between 22:00 and 00:00—well before the trough of the circadian rhythms—and were judged to be equivalent to BrAC of 0.05% at approximately noon.

Additionally, a study with afternoon/early evening alcohol ingestion compared with extended wakefulness showed impairments in simulated driving (speed and position maintenance) at modest BrAC levels (0.05%) comparable to that of 18.5 hours of wakefulness (Arnedt et al., 2001). Although this study examined wake extension, the results were based on 2 separate studies and did not compare directly the combined effects of wake extension and alcohol. Finally, another study by Arnedt et al. (2000) did examine extended waking combined with alcohol, but only included male participants and had a relatively high dose of alcohol (target BrAC of 0.08%). In this study, the greatest impairment of simulated driving performance occurred in the combined alcohol-extended waking group during the elimination phase of alcohol metabolism when BrAC was 0. No interaction effects, however, achieved statistical significance (Arnedt et al., 2000).

When considering the effects of both alcohol and sleep deprivation, task complexity is an important factor. Generally, the likelihood of performance decrements with small doses of alcohol is greater with increasing task complexity (e.g., Drew et al., 1958). According to Kerr and Hindmarch (1991), alcohol appears to produce increased focus on the primary component of complex skills, while secondary tasks are discarded and attentional capacity declines (Kerr and Hindmarch, 1991). Similarly, complex task performance after sleep deprivation places greater demands on attentional capacity than after normal sleep. For example, one neuroimaging study comparing brain activation while performing a divided attention task showed that performance required more attentional resources (evidenced by increased activation) in the sleep-deprived state (Drummond et al., 2001). One goal of the present study is to examine the effects of alcohol and extended waking using tasks with different levels of complexity.

The aim of this study was to extend the findings in the literature by examining how a moderate extension of waking (i.e., late night) with and without a moderate evening dose of alcohol affects driving simulation performance and RT. We hypothesized that cognitive performance impairments from the combined effects of extended waking and moderate evening alcohol would be exacerbated compared with extended waking alone. In addition, we hypothesized that we would see less impairment on the psychomotor vigilance task (PVT), a simple RT task, than on a more complex driving simulation task.

MATERIALS AND METHODS

Methods are described in detail in a companion paper, Rupp et al., Part I. To summarize briefly, 9 men and 20 women with valid drivers' licenses, ages 21 to 25 ($M = 22.6$ years, $SD = 1.2$), successfully completed the study. Nine participants were classified as having a positive parental history (PH+) of alcohol abuse or dependence (3 males, 6 females) based on a structured telephone interview with biological parents inquiring about their experiences with alcohol (structured clinical interview for DSM-IV, First et al., 1995); 17 were classified as having a negative parental history (PH−; 4 males, 13 females), and 3 participants' parental histories could not be determined due to parents' unwillingness to complete the interview.

Participants completed a minimum of 10 nights on a stabilized sleep schedule (8.5 or 9 hours; details on stabilization schedule provided in Rupp et al., Part I) before in-lab sessions began and for a minimum of 5 nights between experimental nights, confirmed by actigraphy (Mini Motionlogger BMA-32, Ambulatory Monitoring Inc., Ardsley, NY), sleep diary, and call-ins to a time-stamped answering machine. The study protocol included an adaptation night and 2 nonconsecutive, randomized nights on which alcohol or placebo were administered. Alcohol (vodka; 0.54 g/kg men; 0.49 g/kg women mixed with tonic) was consumed over 30 minutes ending 1 hour before normal bedtime; the same quantity of beverage was given on placebo.

Performance task practice was provided on 3 occasions: during an orientation visit (about 1-hour driving simulation practice), an actigraph exchange visit (30 minutes of driving simulation practice), and on the adaptation night (30 minutes of driving simulation practice, 10 minutes of PVT practice). The timing of the procedures on each of the experimental nights is detailed in Table 1.

Saliva samples were taken for each participant across the night at approximately 30-minute intervals (details provided in Rupp et al., Part I). Dim-light melatonin onset (DLMO) phase was computed from these samples for each participant to provide a marker of circadian timing.

Breath alcohol concentration in gram percent (g%) was measured at arrival and approximately every 20 to 30 minutes throughout the night following beverage administration (placebo or alcohol ingestion) with an Alco-Sensor IV Breathalyzer (Intoximeters Inc., St. Louis, MO).

Table 1. Timeline of Procedures

Hours awake	Time[a]	Event/form
9.5	−360	ARRIVE, BrAC
10	−330	Meal
10.5	−300	Electrode application
12	−210	**PVT1**
13.5	−150	**DRIVE1**
14	−90	**DRINK START**
14.5	−60	**DRINK END**, BrAC
15	−30	BrAC
15.5	0	STABILIZED BEDTIME, BrAC
		DRIVE2
16	30	BrAC
16.5	60	BrAC
17	90	BrAC
		PVT2
17.5	120	BrAC
18	150	BrAC
18.5	180	BrAC
		DRIVE3
19	210	BrAC
19.5	240	BrAC
		LIGHTS OUT

[a]Minutes relative to stable bedtime.
BrAC, breath alcohol concentration; PVT, psychomotor vigilance task.

Apparatus and Task Parameters

The PVT (Dinges and Powell, 1985) was used as a "gold standard" of vigilance (e.g., Doran et al., 2001) and simple RT as compared with the driving simulation task. The PVT is a 10-minute simple visual RT task: using a book-sized, hand-held device, the participant was instructed to respond with a button press as quickly as possible with his/her dominant thumb each time a number appeared on a screen at various interstimulus intervals (varied randomly from 2 to 10 seconds in 2-second increments), providing measures of RT in milliseconds. Psychomotor vigilance task variables were derived using REACT software (Ambulatory Monitoring Inc.).

The driving simulation task runs on a personal computer using the Drivesim 3.00 computer software (York Computer Technologies, Kingston, ON, Canada) with peripheral steering wheel, and gas and brake pedals (Logitech Wingman Formula, Freemont, CA). The program operates using Windows 98, 200 Mhz clock speed, 15 in. monitor, and Direct X 6.0 software installed. The task display is a 2-lane road with speed limit signs, lane dividers, and small trees. In addition, the driver sees an outline of the car's front end, a digital speedometer, and—in the dual task—the numbers for the subtract task. Participants are instructed to stay in the center of the right-hand lane and to maintain a fixed speed while "driving" on the straight "road" with no other vehicles. The participant's car is pushed by "wind gusts" to the right of the lane, left of the lane, or not at all (randomly determined). The strength of the wind gusts is measured as the number of road units the wind moves the simulated car with no steering correction. Lane position (0 = left edge to 100 = right edge) and speed are stored for each 10th of a second. The following task parameters were set based on previous work with well-rested young adults (Rupp et al., 2004): speed = 50 mph (determines speed limit posting); wind-gust interval = 20 seconds; and wind-gust offset = 30 U. Road width was 100 U.

On each test session, a single 15-minute drive-only task is immediately followed by a dual task (driving plus subtraction by sevens). For the dual task, a 3-digit number is displayed for 1 second in the center of the driving background on the computer screen, followed by a 1.4-second interstimulus interval. Participants respond to indicate whether the current 3-digit number displayed is 7 less than the preceding number by pressing the right-hand (correct) or left-hand (incorrect) button of the steering wheel as quickly as possible using their thumbs. The computer provides the correct result (previous number minus 7) on 75% of trials, and otherwise, an incorrect number either 6 or 8 less than the previous number.

Analysis

Breath alcohol concentration values were used to gauge participants' levels of intoxication.

Psychomotor vigilance task variables including mean RT (ms), mean fastest 10% RT (ms), and PVT lapses (count of RTs $\geq$ 500 ms) were assessed. Variables on both the single and dual driving tasks include lane variability [standard deviation of the road position deviation in units from lane center (50)], speed variability (standard deviation of speed deviation from 50 mph), and off-road events (count of times the car went beyond the edges of the lanes). In addition to the driving variables, variables specific to the subtraction task included mean RT in milliseconds and percentage of correct responses.

The statistical package SPSS® was used for statistical analyses (version 8.02 for Macintosh, SAS Institute Inc., Cary, NC). We analyzed driving simulation and PVT measures for time awake as well as for differences within conditions (placebo vs alcohol). Variables were analyzed using repeated measures ANOVAs with within-subject factors time awake and condition (placebo or alcohol). Post hoc tests were performed using paired sample *t*-tests for significant main effects of time awake for driving simulation variables. Analyses for the main effect of condition and the interaction of condition and time awake did not include baseline measures, because placebo/alcohol ingestion occurred after baseline. Paired sample *t*-tests were used for PVT variables to assess the main effects of condition. A Greenhouse–Geisser adjustment (Geisser and Greenhouse, 1958) was used for all driving simulation analyses and the level of significance for all tests was set at 0.05; effect sizes (partial η^2) for variables were determined for all significant effects. Partial η^2 values of 0.2, 0.5, and 0.8 are indicative of small, medium, and large effect sizes, respectively.

Secondary separate analyses for the effects of sex and PH (positive or negative) were performed using the same analyses described above with the addition of sex and PH as between-subject factors. Parental history unclassified subjects ($n = 3$) were excluded from the analyses.

Mean prior sleep duration (actigraph minutes time in bed) and DLMO phase were examined for differences between conditions using paired samples *t*-tests.

RESULTS

Prior Sleep and Circadian Phase

The mean (SD) estimate sleep durations (actigraph minutes between sleep onset and sleep offset) for the 5 nights on the stabilized sleep schedules before in-lab alcohol and placebo conditions were 467 (19) and 473 (17), respectively, and did not differ significantly between conditions.

Analyses of salivary melatonin showed that the mean circadian phase was the same for assessments under both conditions (Rupp et al., 2007). The mean (SD) DLMO phase occurred 39.7 ($\pm$66) minutes before the end of beverage administration in the placebo condition and 36 ($\pm$65) minutes before the end of beverage administration in the alcohol condition.

Breath Alcohol Concentration

Breath alcohol concentration taken upon arrival and departure from the sleep lab confirmed a BrAC of 0 g% for all participants, as did BrAC readings from the placebo night. Mean (SD) of BrAC levels before each driving simulation and PVT test are indicated in Figs. 1 and 2 (all well below the legal driving limit for most states of 0.08 g%).

PVT Performance

Table 2 summarizes descriptive data and significant statistical effects for the PVT variables. One participant's (female, PH −) data from the alcohol condition were missing due to technical error and her data were removed from all PVT analyses.

All PVT variables showed a main effect of time awake: mean RT and fastest 10% RT were slower after 17 hours awake than after 12 hours and more lapses occurred. As shown in Fig. 1, participants' RTs were less stable at 17 hours awake, with more RTs $\geq$500 ms, reflecting lapses in attention. No effects of condition were found for any PVT variable. Separate analyses for sex (9 males, 19 females) and PH (9 PH+, 16 PH −) differences found no sex or PH differences for any PVT variable.

Driving Simulation

Table 3 summarizes descriptive data and significant statistical effects for the driving simulation task. Results are described separately for the drive-only and dual-task components.

Drive Only. Three participants' (1 male, 2 females, 1 PH+, 1 PH − , 1 unknown PH) data were excluded for the drive-only driving simulation component and all drive-only analyses: 2 due to technical problems and 1 due to participant illness on the placebo night.

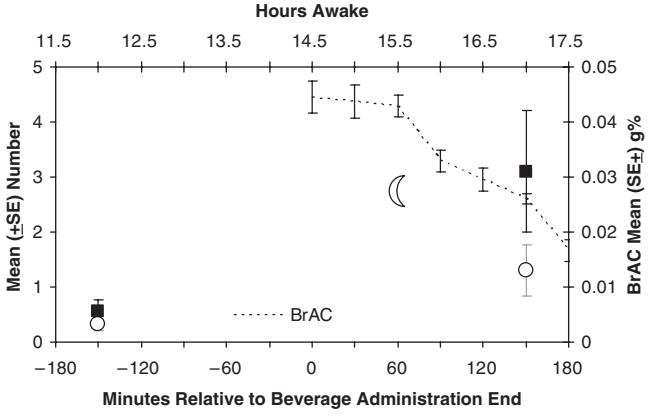

Fig. 1. Mean and standard deviation of attentional lapses are displayed for placebo (○) and alcohol (■) conditions for the psychomotor vigilance task. Values for lapses are labeled on the left *y*-axis and for breath alcohol concentration (BrAC) on the right *y*-axis. Time on the top *x*-axis is labeled in hours as waking and the lower *x*-axis is labeled in minutes relative to the end of beverage administration (time 0); the stabilized home bedtime is +60 min. The crescent moon symbol indicates stabilized bedtime.

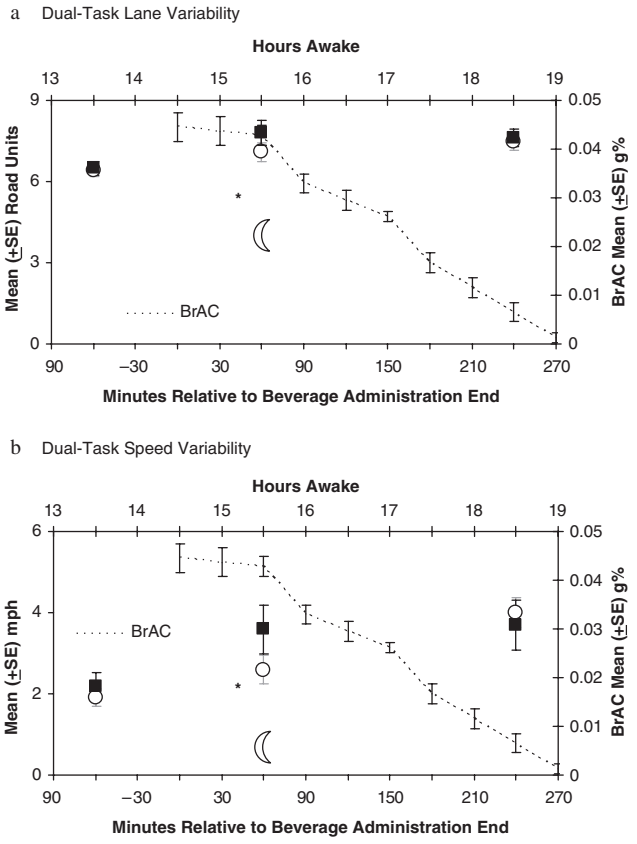

Fig. 2. Mean and standard deviation of lane variability (**A**) and speed variability (**B**) are displayed for placebo (○) and alcohol (■) conditions for the dual-task driving simulation. Values for lane variability and speed variability are labeled on the left *y*-axis and for breath alcohol concentration (BrAC) on the right *y*-axis. Time on the top *x*-axis is labeled in hours as waking and the lower *x*-axis is labeled in minutes relative to the end of beverage administration (time 0); the stabilized home bedtime is +60 min. *Significant ($p<0.05$) differences with alcohol compared with placebo. The crescent moon symbol indicates stabilized bedtime.

Significant main effects of time awake for lane variability and speed variability indicated that participants had more difficulty maintaining a stable lane position and constant speed with increasing time awake. Post hoc *t*-tests showed significant differences between the baseline and subsequent driving tests. A similar main effect of time awake for off-roads showed that participants had more difficulty staying in their lane for tests at 15.5 and 18.5 hours awake compared with baseline. In addition, a main effect of condition was found for lane variability, showing less stable performance after alcohol ingestion as compared with after placebo. No interactions were significant for drive-only task variables.

Finally, no main effects or interactions of sex (8 males, 18 females) or PH (8 PH+, 16 PH −) were found for the drive-only task.

Dual-Task. Three participants' (1 male, 2 females, 2 PH+, 1 unknown PH) data were excluded for the dual-task driving simulation analyses due to technical problems and 1 due to participant illness on the placebo night.

Table 2. Mean (SD) Psychomotor Vigilance Task Variables

	Hours awake	
	12 (baseline)	17
Mean RT (ms)[a]		
Placebo	240 (26)	262 (40)
Alcohol	245 (27)	263 (40)
Mean fastest 10% RT (ms)[b]		
Placebo	190 (19)	199 (19)
Alcohol	191 (15)	202 (23)
Lapses (count)[c]		
Placebo	0.32 (0.55)	1.3 (2.5)
Alcohol	0.55 (1.2)	3.1 (5.9)

Significant effects from analysis of variance and paired *t*-test.
[a]Time awake ($F_{1,27} = 25.88$, $p < 0.01$, partial $\eta^2 = 0.49$); 12 < 17 h.
[b]Time awake ($F_{1,27} = 9.01$, $p < 0.01$, partial $\eta^2 = 0.25$); 12 < 17 h.
[c]Time awake ($F_{1,27} = 28$, $p < 0.01$, partial $\eta^2 = 0.51$); 12 < 17 h.
RT, reaction time.

We found significant main effects of time awake for lane variability and speed variability on the dual task; participants had more difficulty maintaining a stable lane position and speed on tests at 15.5 and 18.5 hours awake compared with baseline. A similar main effect of time awake for off-road events indicated that participants had more difficulty staying in their lane with increasing time awake.

A significant main effect for condition indicated that lane variability was worse in the alcohol condition compared with placebo. Significant interactions of time awake and condition for lane variability and speed variability are shown in Fig. 2, with worse performance in the alcohol condition compared with placebo at 15.5 hours awake.

Analysis of the scores from the dual-task subtract component resulted in a significant main effect for time awake for percent correct. Scores were worse at 15.5 and 18.5 hours awake versus baseline. No significant main effects or interactions were found for response RT for the subtraction component.

A main effect of sex (8 males, 18 females) was found on the dual task for percent correct on the subtraction task, such that females had greater accuracy on the subtraction task overall. No other sex differences or interactions were found for any other dual-task variable. For percent correct, there was also a significant 3-way interaction of PH (7 PH+, 17 PH −), time awake, and condition so that PH − participants had the fewest correct at 18.5 hours awake on the placebo night.

Table 3. Mean (SD) Driving Simulation Variables

		Hours awake	
Task	13.5 (baseline)	15.5	18.5
Drive only			
Lane variability[a*†]			
Placebo	5.8 (1.0)	6.3 (1.1)	6.5 (1.4)
Alcohol	5.9 (1.0)	6.7 (1.5)	6.7 (1.0)
Speed variability[b*]			
Placebo	1.7 (1.0)	2.3 (1.5)	2.4 (1.8)
Alcohol	1.8 (.99)	2.9 (2.6)	2.5 (1.4)
Off-roads[c*]			
Placebo	0.42 (.95)	0.77 (1.9)	1.6 (2.9)
Alcohol	0.38 (.94)	1.6 (4.6)	1.8 (2.6)
Dual-task drive			
Lane variability[a*†‡]			
Placebo	6.4 (1.1)	7.1 (1.8)	7.5 (1.7)
Alcohol	6.5 (1.3)	7.8 (2.4)	7.6(1.7)
Speed variability[b*‡]			
Placebo	1.9 (1.1)	2.6 (1.8)	4.0 (1.9)
Alcohol	2.2 (1.6)	3.6 (3.0)	3.7 (3.1)
Off-roads[c*]			
Placebo	0.89 (2.0)	2.3 (5.3)	3.8 (5.0)
Alcohol	0.81 (1.3)	3.0 (9.6)	3.8 (6.0)
Dual-task subtract			
% Correct[*§¶]			
Placebo	88.7 (16.6)	87.8 (17.5)	82.9 (21.3)
Alcohol	88.9 (17.9)	85.2 (18.3)	84.4 (19.1)
Response RT (ms)			
Placebo	0.74 (0.18)	0.75 (0.21)	0.74 (0.22)
Alcohol	0.74 (0.20)	0.74 (0.21)	0.76 (0.20)

[a]Road units.
[b]MPH.
[c]count.
Significant effects from analysis of variance; significant differences from post hoc tests.
[*]Time awake: 13.5 < 15.5, 18.5 h.
Drive-only: lane variability ($F_{2,50} = 18.82$, $p < 0.01$, partial $\eta^2 = 0.43$); speed variability ($F_{2,50} = 5.93$, $p = 0.01$, partial $\eta^2 = 0.19$); off-roads ($F_{2,50} = 4.33$, $p = 0.03$, partial $\eta^2 = 0.15$).
Dual-task: lane variability ($F_{2,50} = 12.35$, $p < 0.01$, partial $\eta^2 = 0.33$); speed variability ($F_{2,50} = 17.06$, $p < 0.01$, partial $\eta^2 = 0.41$); off-roads ($F_{2,50} = 4.7$, $p = 0.02$, partial $\eta^2 = 0.15$); % correct, ($F_{2,50} = 7.7$, $p < 0.01$, partial $\eta^2 = 0.24$).
[†]Condition: placebo < alcohol.
Drive-only: lane variability ($F_{1,25} = 8.46$, $p = 0.01$, partial $\eta^2 = 0.25$).
Dual-task: lane variability ($F_{1,25} = 6.19$, $p = 0.02$, partial $\eta^2 = 0.20$).
[‡]Time awake-by-condition interaction: placebo < alcohol at 15.5 h.
Dual-task: lane variability ($F_{1,25} = 5.1$, $p = 0.03$, partial $\eta^2 = 0.17$); speed variability ($F_{1,25} = 5.2$, $p = 0.03$, partial $\eta^2 = 0.17$).
[§]Sex: females > males.
Dual-task: % correct ($F_{1,25} = 4.5$, $p < 0.01$, partial $\eta^2 = 0.17$).
[¶]Parental history (PH)-by-time awake-by-condition interaction: placebo PH − at 18.5 h < all others.
Dual-task: % correct ($F_{1,23} = 5.8$, $p = 0.03$, partial $\eta^2 = 0.17$).
RT, reaction time.

DISCUSSION

We examined the effects of extended waking with and without alcohol on driving simulation and RT. Both measures generally showed performance deterioration with late nights. Alcohol exacerbated the effect on driving simulation variables at 15.5 hours after waking, when alcohol was at its peak. For the simpler RT test—the PVT—performance also deteriorated as the night progressed, although with no differences on the alcohol versus placebo nights.

The current study controlled for prior sleep/wake history and time of day in examining alcohol's effects on performance. We observed no significant differences in time asleep for 5 days before each experimental condition, and indeed, participants obtained 8.5 to 9 hours time in bed for an additional week before that, ensuring that they

were reasonably well rested. Controlling prior sleep is important, especially considering Johnson et al.'s (2004) assertion that Process S varies as a function of the amount of sleep obtained during many previous days. By controlling for homeostatic and circadian factors, we more accurately assess the effects of wake extension and alcohol on performance than previous studies that have not utilized these controls.

We describe our findings in the context of a wake extension paradigm. Our findings align with Van Dongen et al.'s (2003) assertion that there is a build-up of neurobehavioral deficits by excessive wakefulness beyond a maximum period during which stable neurobehavioral functioning cannot be maintained (Van Dongen et al., 2003). Thus, both PVT and driving simulation performances showed marked deterioration from the point of "excess wakefulness" (~16 hours), in some cases equal to that produced by alcohol, worsening as the night progressed.

We compare our findings with the findings of Arnedt et al. (2001), who also studied extended waking and alcohol and driving simulation using a higher target BrAC of 0.08 g% and found a trend for the greatest impairment during the elimination phase of alcohol. In contrast, we found the greatest impairment on several measures when alcohol was at its peak, rather than during the elimination phase. It is possible that the higher dose of alcohol in Arnedt et al.'s (2001) study produced a greater effect of residual sedation (described in more detail in Part I; Roehrs et al., 1994).

For task complexity, our results support previous work suggesting that the effects of small doses of alcohol on performance become stronger with increasing task complexity. Thus, while performance deteriorated with time awake on all measures, only the driving simulation task variables supported our hypothesis that alcohol would exacerbate this deterioration, while the PVT was unaffected by alcohol in this study. Our data extend previous findings by Dawson and Reid (1997) regarding task complexity and extended waking (without alcohol) that showed that the effects of sustained waking on performance increased with greater task complexity.

We also examined the role of sex and PH. Only the subtraction component of the complex dual driving simulation task showed sex and PH differences; females had higher percent correct than males and PH − participants had the fewest correct with 18.5 hours awake with placebo. Previous literature suggests that a low level of response to alcohol is a heritable trait and is found in as many as 60% of men and women with a family history of alcoholism (Eng et al., 2005). No study has examined individuals with a PH+ and their performance with acute alcohol and extended waking. If, indeed, PH+ participants do respond less to acute alcohol, we might predict that they would perform better on the performance tasks than those participants with a PH − . Our findings did not support this prediction; however, our small sample size of PH+ participants limits our interpretations.

In addition to sample size considerations, the study has several limitations; menstrual phase was not taken into consideration when scheduling female participants for in-lab assessment. Although limited evidence suggests that menstrual phase may affect alcohol pharmacokinetics, for our purposes we assume that menstrual phase may add noise as opposed to a systematic bias in one direction or another. A further limitation was that only a single dose of alcohol was used to compare the effects of alcohol on performance and so examination of dose response is not possible.

In conclusion, the current study demonstrated that staying up only a few hours later than usual may have significant effects on performance. In the real world, our demonstrated increase in attentional lapses (PVT RT >500 ms) could translate into increased accident risk if operating machinery or driving a car. In addition, the increased number of driving simulation off-roads with wake extension could translate into potentially fatal consequences in a real-world driving situation. Consuming even low to moderate amounts of alcohol within the legal limit exacerbates certain of these effects. These results indicate the need for increased public awareness and education about the risks and impairment associated with even low levels of alcohol late at night, specifically with regard to driving.

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REFERENCES

Arnedt JT, Wilde GJ, Munt PW, MacLean AW (2000) Simulated driving performance following prolonged wakefulness and alcohol consumption: separate and combined contributions to impairment. J Sleep Res 9:233–241.

Arnedt JT, Wilde GJ, Munt PW, MacLean AW (2001) How do prolonged wakefulness and alcohol compare in the decrements they produce on a simulated driving task? Accid Anal Prev 33:337–344.

Borbély AA, Achermann P (1992) Concepts and models of sleep regulation: an overview. J Sleep Res 1:63–79.

Dawson D, Reid K (1997) Fatigue, alcohol and performance impairment. Nature 388:235.

Dinges D, Powell JW (1985) Microcomputer analyses of performance on a portable, simple visual RT task during sustained operations. Behav Res Methods Instrum Comput 17:652–655.

Doran SM, Van Dongen HP, Dinges DF (2001) Sustained attention performance during sleep deprivation: evidence of state instability. Arch Ital Biol 139:253–267.

Drew GC, Colquhoun WP, Long HA (1958) Effect of small doses of alcohol on a skill resembling driving. Med Leg J 26:94–108.

Drummond SP, Gillin JC, Brown GG (2001) Increased cerebral response during a divided attention task following sleep deprivation. J Sleep Res 10:85–92.

Eng MY, Schuckit MA, Smith TL (2005) The level of response to alcohol in daughters of alcoholics and controls. Drug Alcohol Depend 79: 83–93.

First MB, Spitzer RL, Gibbon M, Williams JB (1995) *Structured Clinical Interview for DSM-IV Axis I Disorders (SCID)*. New York Psychiatric Institute, Columbia University, New York.

Geisser S, Greenhouse SW (1958) An extension of Box's results on the use of the F distribution in multivariate analysis. Ann Math Stat 29:885–891.

Johnson ML, Belenky G, Redmond DP, Thorne DR, Williams JD, Hursh SR, Balkin TJ (2004) Modulating the homeostatic process to predict performance during chronic sleep restriction. Aviat Space Environ Med 75 (suppl): A141–A146.

Kerr JS, Hindmarch I (1991) Alcohol, cognitive function and psychomotor performance. Rev Environ Health 9:117–122.

National Center for Statistics (2004) *2004 Traffic Safety Fact Sheets*. National Highway Safety and Traffic Administration, Washington, DC.

Roehrs T, Claiborue D, Knox M, Roth T (1994) Residual sedating effects of ethanol. Alcohol Clin Exp Res 18:831–834.

Rupp T, Acebo C, Carskadon MA (2007) Evening alcohol suppresses salivary melatonin in young adults. Chronobiol Int 24:1–8.

Rupp T, Arnedt JT, Acebo C, Carskadon MA (2004) Performance on a dual driving simulation and subtraction task following sleep restriction. Percept Mot Skills 99 (part 1): 739–753.

Van Dongen HP, Maislin G, Mullington JM, Dinges DF (2003) The cumulative cost of additional wakefulness: dose-response effects on neurobehavioral functions and sleep physiology from chronic sleep restriction and total sleep deprivation. Sleep 26:117–126.

Williamson AM, Feyer AM (2000) Moderate sleep deprivation produces impairments in cognitive and motor performance equivalent to legally prescribed levels of alcohol intoxication. Occup Environ Med 57: 649–655.