Ethanol and Sleep Loss: A "Dose" Comparison of Impairing Effects

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Study Objectives: Studies to assess the risks associated with sleep loss relative to the well-documented risks of alcohol are limited in number and design. This study compared the "dose"-related sedative, performance-impairing, and amnestic effects of sleep loss to those of ethanol ingestion.
Design: Mixed-design experiment with random assignment to a sleep loss (n=12) or ethanol (n=20) group, with each participant assessed under 4 conditions.
Participants: Thirty-two healthy normal adult volunteers, aged 21 to 35 years.
Interventions: In sleep loss, participants had 8, 6, 4, and 0 hours time in bed, producing 0, 2, 4, and 8 hours of sleep loss. For ethanol, participants ingested 0.0 g/kg, 0.3 g/kg, 0.6 g/kg, and 0.9 g/kg ethanol from 8:30 AM to 9:00 AM after 8 hours of time in bed the previous night. Each participant received his or her 4 doses of ethanol or sleep loss in a Latin square design with 3 to 7 days between doses.
Measurements: All subjects completed the Multiple Sleep Latency Test (MSLT) at 9:30 AM, 11:30 AM, 1:30 PM, 3:30 PM, and 5:30 PM and a performance battery at 10:00 AM, 12:00 NOON, 2:00 PM, and 4:00 PM consisting of memory, psychomotor vigilance, and divided attention tests.
Results: Ethanol and sleep loss reduced the average daily sleep latency on the MSLT, both as a linear function of dose, with sleep loss in hours being 2.7 times more potent than ethanol in grams per kilogram. Ethanol and sleep loss also slowed reaction time on the psychomotor vigilance test in a linear dose-related function with the 2 being equipotent in their impairing effect. On the divided attention test, tracking deviations were increased by both ethanol and sleep loss in an equipotent and linear dose-related function. Memory recall was reduced in a linear dose-related function by both ethanol and sleep loss with ethanol being slightly more potent. Finally, sleep loss doses produced a linear decrease in self-rated quality of performance, while only at the highest ethanol dose was performance rated as poorer.
Conclusions: At the studied doses, sleep loss was more potent than ethanol in its sedative effects but comparable in effects on psychomotor performance. Ethanol produced greater memory deficits, and subjects were less aware of their overall performance impairment.
Key Words: Ethanol, sleep loss, MSLT, psychomotor performance, memory
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INTRODUCTION

THE SEDATIVE, AMNESTIC, AND PERFORMANCE-DISRUPTIVE EFFECTS OF ETHANOL AND SLEEP LOSS HAVE BEEN WELL DOCUMENTED SCIENTIFICALLY. Studies of the sedative effects of ethanol using direct physiologic measurement (ie, Multiple Sleep Latency Test [MSLT]) have shown a dose-related increase in sleepiness.[1] Similarly, parametric reduction in nocturnal bedtime for a single night is followed by a linear increase in sleepiness the next day.[2] Dose-related amnestic effects of ethanol have been reported for visual, verbal, and digit memory tasks.[3] Sleep loss due to reduced time in bed (TIB) is also associated with memory impairment, although a limited number of studies have performed parametric analyses.[4] Finally, the "dose"-related performance-impairing effects of ethanol and sleep loss have each been extensively studied.[1,2,5,6]

As yet, comparative studies of ethanol and sleep loss on any of these measures are limited in number and design. Studies have compared single doses of each,[7] multiple doses of ethanol to a single dose of sleep loss,[8] or multiple doses of sleep loss to a single dose of ethanol.[9] Other studies have compared a single dose of ethanol at multiple time points over the rise or decline in breath ethanol concentration (BrEC) to accumulating hours of wakefulness.[10-12] No study has compared multiple doses of ethanol to multiple doses of sleep loss, each administered in a separate session, to assess the relative potency of the 2 for impairment.

Comparative studies are highly critical for public health reasons. The public generally recognizes the risks associated with the use of ethanol, but risks associated with sleep loss, particularly moderate amounts, are not appreciated. Studies comparing the 2 on a common metric are important to further understand the relative risks and to overcome the perception that ethanol is impairing but that acute sleep loss is not. Thus, this study assessed the dose effects of sleep loss and ethanol over doses that included the previously studied high dose of each (ie, 1 night of sleep loss and legal intoxification in the United States—BrEC of 0.8% to 0.10%) and fractions thereof.

METHODS

Participants

Thirty-two men and women, aged 21 to 35 years, participated in the study. They had normal physical, psychiatric, and laboratory test results and a normal screening nocturnal polysomnogram and MSLT the following day. They were randomly assigned to an ethanol or sleep-loss group (see Table 1 for group demographics and sleep characteristics). This study was reviewed and approved by the Human Rights Board of the institution. All subjects signed a written informed consent and received payment for participation.

Study Design

The study was conducted in a mixed design with a between-subject factor, ethanol or sleep loss, and a within-subject factor, dose of either ethanol or sleep loss. The ethanol arm of the study was conducted in a double-blind fashion. Participants were randomly assigned to 1 of the experimental groups, ethanol or sleep loss (see Table 1). The within-subject comparison was 3 doses of ethanol (0.3 g/kg, 0.6 g/kg, and 0.9 g/kg) or sleep loss (2 hours, 4 hours, and 8 hours of sleep loss) and the place-

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bo dose for the assigned group (0.0 g/kg of ethanol or 0 hours of sleep loss, ie, 8 hours of TIB). The order in which subjects underwent the ethanol or sleep-loss doses was determined by a Latin square design with 3 to 7 days for recovery between doses.

Table 1—Demographic and Sleep Characteristics of the Study Groups

Characteristic	Intervention	
	Sleep Loss	Ethanol
No.	12	20
Women / Men	5 / 7	8 / 12
Age, y*	27.5 (5.37)	26.0 (3.68)
Sleep efficiency, %*†	92.2 (3.35)	90.6 (3.21)
Sleep latency, min*‡	11.0 (2.36)	12.5 (3.21)

*Data are presented as mean (SD)
†Sleep efficiency was recorded at baseline and is calculated as the time asleep/time in bed
‡The Multiple Sleep Latency Test was used to determine the sleep latency.

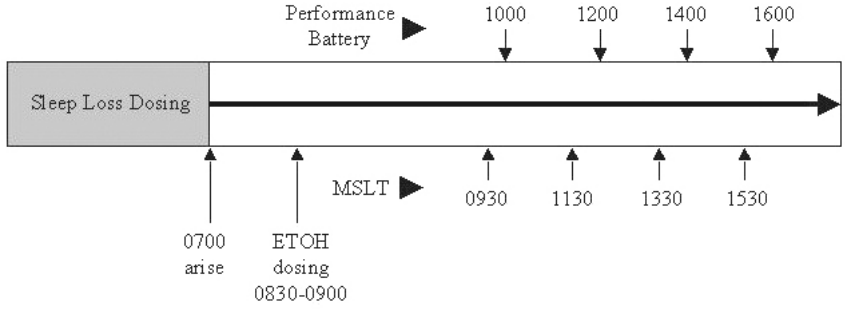

Figure 1—The timeline of experimental days. The sleep-loss group arose at 7:00 AM after going to bed at 11:00 PM, 1:00 AM, or 3:00 AM or remained awake, for the 8, 6, 4, and 0 hours of time in bed, producing the 0, 2, 4, and 8 hrs sleep loss doses. The ethanol (ETOH) group was in bed from 11:00 PM to 7:00 AM and, in the morning, consumed 0.0 g/kg, 0.3 g/kg, 0.6 g/kg, or 0.9 g/kg ethanol from 8:30 AM to 9:00 AM. The Multiple Sleep Latency Test (MSLT) and performance testing battery were completed as indicated.

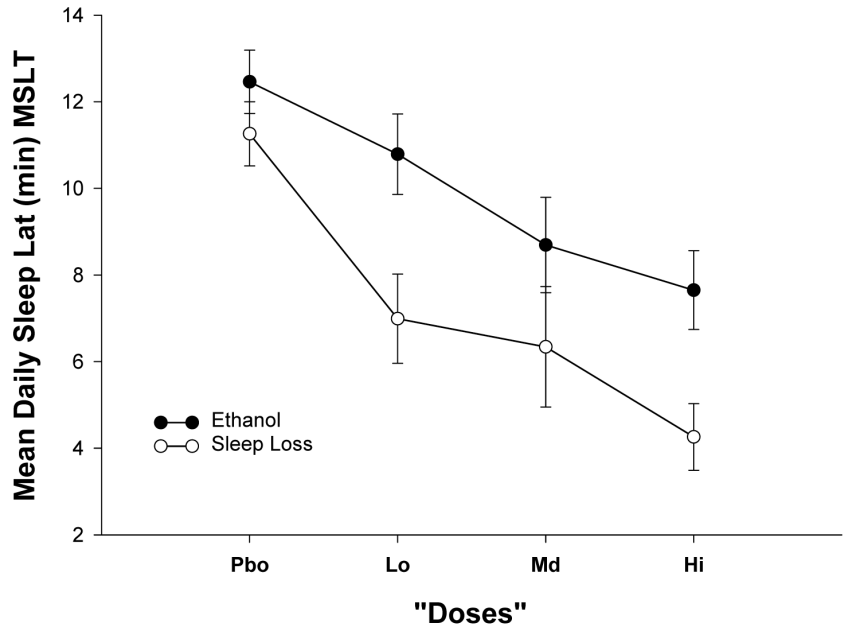

Figure 2—Mean daily sleep latency, in minutes, on the Multiple Sleep Latency Test for the placebo (Pbo), low (Lo), medium (Md), and high (Hi) doses of sleep loss and ethanol. The placebo was 8 hours of time in bed (TIB) or 0.0 g/kg ethanol, the low dose was 6 hours of TIB or 0.3 g/kg ethanol, the medium dose was 4 hours of TIB or 0.6 g/kg ethanol, and the high dose was 0 hours of TIB or 0.9 g/kg ethanol.

General Procedures

Respondents to advertisements for healthy volunteers were screened in a telephone interview that questioned potential subjects regarding habitual sleep time and schedule. Those reporting less than 7 hours of nightly sleep, more than 2 hours of variation in bedtime, and bedtimes later than 1:00 AM were excluded. Those volunteers passing the telephone screen were invited to the laboratory to provide a medical and drug-taking history and undergo a physical examination and standard laboratory blood and urine tests. Participants were excluded if they had acute or chronic medical conditions or were currently taking drugs that act on the central nervous system. The urine tests were also used to screen for current use of illicit drugs. Participants with a current or past history of psychiatric disorders; drug addiction; alcoholism; or consuming more than 200 mg of caffeine (from either beverage or food sources) daily, more than 14 alcohol drinks (standard 1-oz drinks) weekly, and any nicotine were excluded.

Finally, to qualify, participants had to show a sleep efficiency of greater than 85% on their screening nocturnal polysomnogram and no evidence of primary sleep disorders.[13,14] On the MSLT the following day, performed according to the standard protocol at 9:30 AM, 11:30 AM, 1:30 PM, 3:30 PM, and 5:30 PM, the subjects had an average daily sleep latency, with sleep latency defined as the first 30-second epoch scored as sleep, of more than 8 minutes and no sleep-onset rapid eye movement periods.[15]

A performance battery, consisting of memory, psychomotor vigilance, and divided attention tests was administered at 10:00 AM, 12:00 noon, 2:00 PM, and 4:00 PM. The screening day was used to train participants on the performance battery. The memory assessment was a 4-item paired-associate test in which the 4 paired words were presented for 1 minute and a probed recall for the missing member of each pair was tested 10 minutes later.[16] The psychomotor vigilance test (PVT) was administered on a hand-held device. It is a simple visual reaction-time task with a 10-minute duration.[17] The 15-minute divided attention test (DAT) required tracking a moving target across a video screen using a joystick and simultaneously pressing a button in response to the appearance of a white circle in the periphery of the screen or in the middle of the moving target.[18,19] Performance was self rated after each test session on a 7-point scale for its quality (good-poor) and the effort expended (a little-a lot).

Participants underwent the 4 doses of ethanol or TIB according to their random group assignment. Each dose consisted of 2 days, a baseline day with an 8-hour TIB that preceded the dose test day. On the baseline night, participants reported to the laboratory at 9:30 PM and went to bed at 11:00 PM and arose at 7:00 AM. The daytime MSLT and performance testing schedule is illustrated in Figure 1. Time of arising was always set to 7:00 AM, that is in both the ethanol and sleep loss groups and on both baseline and test days of each dose. Upon arising, participants toileted and were provided a light breakfast consisting on a noncaffeinated beverage and a roll or toast. They then underwent the MSLT and performance assessment as outlined.

Ethanol Procedures

Participants assigned to the ethanol group received placebo or ethanol from 8:30 AM to 9:00 AM. The 4 ethanol doses were 0.0 g/kg, 0.3 g/kg, 0.6 g/kg, and 0.9 g/kg. The ethanol was prepared in a 1:4 ratio with 80-proof vodka added to chilled tonic water, and the placebo

consisted of the chilled tonic water (in equal volume to the ethanol) with 3 drops of ethanol floated on the surface. The BrEC was measured at 8:30 AM before ethanol intake and immediately before each MSLT test with an Alco-Sensor III Intoxometer (Intoxometers Inc, Richmond, VA) that was calibrated weekly.

Sleep-loss Procedures

Participants assigned to the sleep-loss group received 8 hours, 6 hours, 4 hours and 0 hours of TIB with lights out adjusted to achieve the given TIB and the time of arising held constant at 7:00 AM. Thus, lights out was at 11:00 PM, 1:00 AM, and 3:00 AM for the 8, 6, and 4 hours of TIB and no TIB to produce the 0 hours, 2 hours, 4 hours, and 8 hours of sleep loss, respectively. Participants were not told which sleep-loss condition they were scheduled to undergo prior to entering the laboratory on the baseline night.

Study Instructions and Requirements

On the 3 to 7 days at home between doses, participants were asked to maintain an 11:00 PM to 7:00 AM sleep schedule. During the day on which participants entered the laboratory at 9:30 PM to begin each of the 4 dose sessions, participants were asked to refrain from drinking alcohol and caffeine after 5:00 PM. Throughout the study, participants were told to avoid the use of any over-the-counter or prescribed medications. Females assigned to the ethanol group were instructed to maintain their birth control methods throughout the study and were required to show negative pregnancy-test results on each entry to the laboratory.

Analyses

To reduce variability, the performance measures were converted to change scores from the prior baseline day for each dose. The mean change score for the 10:00 AM and 12:00 noon tests were the data analyzed, given the BrEC results presented below. Unlike the performance data, average daily sleep latency (the mean of the 4 tests) was the dependent measure of the MSLT analyzed. Studies have shown that at least 3 latency tests are necessary to produce a reliable MSLT, and our previous studies with similar ethanol doses have shown continued sedation on the MSLT after the BrEC has reached 0.[20,21]

To evaluate the presence and nature of the treatment effects, dose was compared within the ethanol and sleep-loss groups alone by 1-factor multivariate analysis of variance (MANOVA). Significant treatment effects were then further evaluated by trend analyses and Duncan posthoc comparisons. To compare ethanol and sleep loss, mixed-design MANOVAs with group (ethanol vs sleep loss) as the between-factor and dose as the within-subject factor were used. Conservative *P* levels were used for the dose factor in all analyses. Finally, relative potencies between ethanol and sleep loss were estimated from the 3 active doses when the data satisfied the criteria for a valid parallel-line bioassay.[22] The criteria require the dose-effect curves to be linear (eg, as seen on the trend analyses) and approximately parallel (eg, as evident by the absence of a group by dose interaction in the mixed-design MANOVAs). Given that these assumptions of a valid parallel-line bioassay are fulfilled, the analysis allows for differing intervals between doses and different units of measure. This analysis computes regression lines over the lowest to highest doses of each treatment, compares the regression lines between treatments to determine the magnitude of the difference, and tests the likelihood of overlap between lines.

RESULTS

Breath Ethanol Concentrations

The BrECs differed among the 3 ethanol doses. On average it was .09%, .04%, and .02%, for the 0.9 g/kg, 0.6 g/kg, and 0.3 g/kg doses at 30 minutes after consumption (9:30 AM test) ($F = 9.44$, $P < .004$); declined to .07%, .03%, and .00% ($F = 31.74$, $P < .001$) at 2.5 hours (11:30 AM test); and reached .02%, .00%, and .00% at 4.5 hours after consumption (1:30 PM test). At the 3:30 PM test (6.5 hours after consumption) the BrEC was 0 for all doses.

Multiple Sleep Latency Test

The average sleep latency on the MSLT for placebo and the 3 doses of ethanol and sleep loss is presented in Figure 2. Ethanol reduced the sleep latency ($F = 8.80$, $P < .001$), with the 0.3-g/kg dose differing from the 0.9-g/kg dose and the 0.6-g/kg dose being intermediate. Reduced TIB also reduced sleep latency ($F = 07.46$, $P < .001$), with 8 hours of TIB differing from 0 hours of TIB and 4 and 6 hours being intermediate. The functions relating sleep loss ($F = 51.68$, $P < .001$) and ethanol ($F = 11.99$, $P < .001$) to dose were both linear, but sleep loss also yielded nonlinear ($F = 4.92$, $P < .05$ - cubic) order effects. Sleep loss reduced sleep latency to a greater extent than did ethanol ($F = 7.23$, $P < .01$). There was no group by dose interaction (eg, necessary for a valid parallel-line bioassay). Finally, sleep loss (in hours) was 2.7 times more potent in reducing sleep latency than was ethanol (g/kg) in the parallel-line bioassay.

Memory

Change from baseline for number of items recalled for doses of sleep loss and ethanol are presented in Table 2 (negative scores reflect fewer items recalled). The ingestion of ethanol reduced recall ($F = 3.40$, $P < .02$), with the 0.0-g/kg dose differing from the 0.9-g/kg dose, and the 0.3-g/kg and 0.6-g/kg doses being intermediate but not different from placebo. Sleep loss reduced recall ($F = 3.03$, $P < .04$) with both 8 hours and 6 hours of TIB differing from the 0 hours of TIB. A linear function described the dose relation for ethanol ($F = 17.9$, $P < .001$) and sleep loss ($F = 11.9$, $P < .005$). Ethanol produced a greater impairment than did sleep loss ($F = 4.49$, $P < .04$), with no group by dose interaction. On the relative potency analyses, ethanol was slightly more potent (0.03 times) than sleep loss, but significantly so ($P < .01$).

Psychomotor Vigilance Performance and DAT

Table 2 presents PVT change scores for number of lapses and the 10% fastest reaction times (negative scores reflect more lapses and slower reaction times). Sleep loss produced an increase in lapses ($F = 3.19$, $P < .04$) with the 8 hours of TIB differing from 0 hours of TIB. Ethanol did not affect lapses, and sleep loss and ethanol did not differ in effects on lapses. Thus relative potency analyses were not conducted on this parameter. Ethanol slowed the 10% fastest reaction times ($F = 3.40$, $P < .02$), with the 0.0-g/kg and 0.3-g/kg doses differing from the 0.9-g/kg dose.

Table 2—"Dose" Effects of Ethanol and Sleep Loss on Memory and Performance

	Ethanol, g/kg				Sleep loss, h			
	0.0	0.3	0.6	0.9	0	2	4	8
Memory recall # correct	0.00 (0.16)	-0.35 (0.18)	-0.54 (0.14)	-0.80 (0.17)	0.08 (0.21)	0.08 (0.24)	-0.19 (0.18)	-0.44 (0.22)
PVT # Lapses	-0.16 (0.40)	-0.49 (0.57)	-1.22 (0.64)	-1.35 (0.58)	0.57 (0.58)	-1.10 (0.63)	-0.98 (0.57)	-2.63 (0.83)
PVT 10% Fastest RTs	0.99 (2.82)	1.86 (3.32)	-6.13 (6.52)	-17.9 (6.78)	7.74 (5.91)	-5.95 (4.47)	-6.39 (5.27)	-18.1 (4.63)
DAT central RT	-0.04 (0.02)	-0.05 (0.04)	-0.07 (0.03)	-0.10 (0.03)	0.01 (0.02)	0.01 (0.02)	-0.08 (0.03)	-0.15 (0.14)
DAT tracking deviations	-3.15 (2.28)	0.78 (2.77)	-7.34 (2.89)	-13.3 (6.23)	2.60 (0.97)	-1.52 (1.34)	3.73 (4.48)	-8.33 (2.71)
Performance rating	-0.02 (0.20)	0.05 (0.19)	0.12 (0.15)	-0.80 (0.30)	0.08 (0.23)	-0.29 (0.30)	-0.58 (0.26)	-1.15 (0.38)

Data are change scores (baseline minus test day except for Memory recall, where test day minus baseline was used) (± SEM). Negative scores reflect poorer performance: fewer memory items recalled, greater number of lapses, longer reaction times, greater tracking deviations, and poorer self-rated performance. PVT refers to psychomotor vigilance test; DAT, divided attention test; RT, reaction time

Sleep loss also slowed the 10% fastest reaction times (F = 4.11, $P < .01$), with 8 hours of TIB differing from 0 hours of TIB. A linear function described the dose effects for both ethanol (F = 10.89, $P < .01$) and sleep loss (F = 8.12, $P < .01$). Ethanol and sleep loss did not differ in effects on the 10% fastest reaction times, which the relative potency analyses also reflected (NS).

The DAT measures—tracking deviations and central reaction times—are presented in Table 2. Central reaction time was slowed by sleep loss (F = 6.20, $P < .002$), with 8 hours and 6 hours of TIB differing from 0 hours of TIB. The ingestion of ethanol did not alter central reaction time. Thus, relative potency analyses were not conducted on this parameter. Tracking deviations were increased by sleep loss (F = 4.35, $P < .01$), with 8 hours of TIB differing from 0 hours of TIB. Ethanol ingestion also increased tracking deviation (F = 3.00, $P < .04$), with the 0.3-g/kg dose differing from the 0.9-g/kg dose. Both ethanol (F = 9.25, $P < .01$) and sleep loss (F = 4.32, $P < .05$) produced linear dose effects. Ethanol ingestion and sleep loss did not differ in effects on tracking deviations, which the relative potency analyses also reflected (NS).

Self Ratings of Performance

Finally, self-rated performance data are presented in Figure 3. Sleep loss reduced self ratings of performance (F = 3.42, $P < .03$), with 8 hours and 6 hours of TIB differing from 0 hours of TIB. The relation of sleep loss to performance ratings was linear (F = 17.3, $P < .001$). Ethanol ingestion reduced ratings (F = 4.10, $P < .02$), with the 0.0-g/kg dose differing from the 0.9-g/kg dose. For ethanol ingestion, the dose-effect relation was quadratic only (F = 6.39, $P < .02$). Impaired performance was perceived only at the highest ethanol dose. Sleep loss produced a borderline reduction relative to ethanol ($P < .09$) in self ratings of performance, and the absence of linear dose effects for ethanol precluded a valid relative potency comparison.

Table 3—Sleep Loss and Ethanol Dose Equivalence for Sedative Effects

Sleep loss (time in bed), h	Dose	Beer, no.*	BrEC%†
8 (0)	2.16 g/kg	10-11	0.190%
6 (2)	1.07 g/kg	7-8	0.102%
4 (4)	1.0 g/kg	5-6	0.095%
2 (6)	0.5 g/kg	2-3	0.045%

*Number of 12-oz beers in the United States
†Approximate breath ethanol concentration (BrEC) at peak

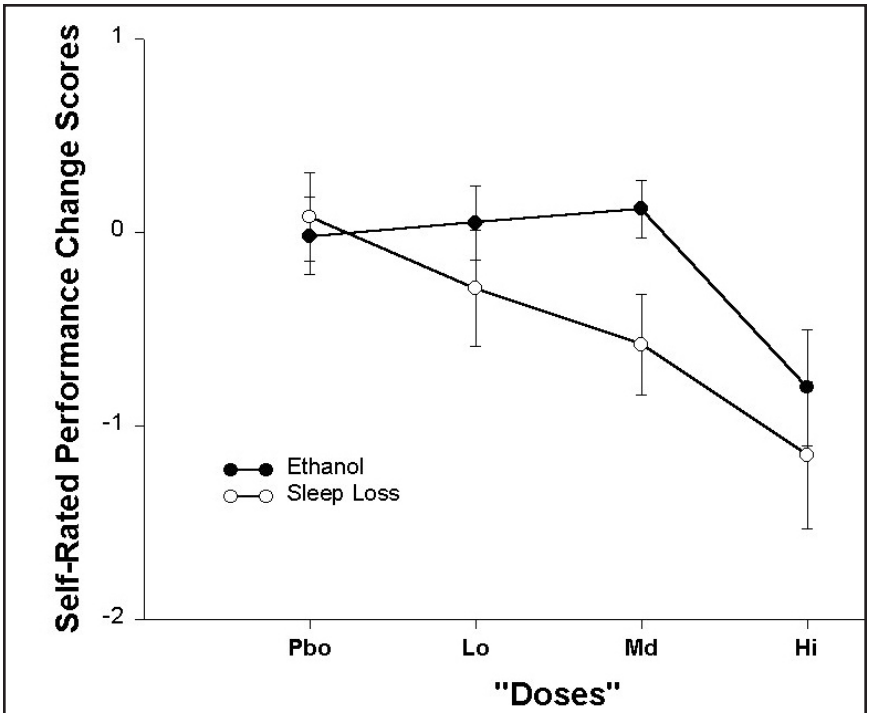

Figure 3—Change in self-rated performance from the baseline day preceding each experimental day for the placebo (Pbo), low (Lo), medium (Md), and high (Hi) doses of sleep loss and ethanol. The placebo was 8 hours of time in bed (TIB) or 0.0 g/kg ethanol, the low dose was 6 hours of TIB or 0.3 g/kg ethanol, the medium dose was 4 hours of TIB or 0.6 g/kg ethanol, and the high dose was 0 hours of TIB or 0.9 g/kg ethanol.

DISCUSSION

The major findings of this study are that, at the studied doses, sleep loss is at least as potent as ethanol in its performance-impairing and amnestic effects and is significantly more potent in its sedative effects. In terms of sedative effects as measured by the MSLT, sleep loss was 2.7 times more potent, meaning that 8 hours of sleep loss is equivalent to 2.16 g/kg of ethanol and 2 hours of sleep loss is equivalent to 0.54 g/kg. Table 3 presents the sleep loss and ethanol equivalence for sedative effects as measured by the MSLT by converting the grams per kilogram of ethanol dosing to the number of United States beers and approximate BrECs. In vigilance and divided-attention performance, sleep loss and ethanol were equipotent. In memory, ethanol was slightly more potent. Finally, while potency analyses were not conducted on self ratings of performance, the dose-effect curves were dramatically different. Increasing sleep loss was perceived as increasingly impairing, while only the highest ethanol dose was rated as impairing.

The differential relative potencies between the sedative and performance (ie, more potent vs equipotent) effects of sleep loss and ethanol may reflect an enhanced sensitivity of the MSLT and/or a reduced sensitivity of the performance measures to these 2 impairing manipulations. While both performance tests used in this study have previously been shown to be sensitive to sleep-deprivation effects, these tests are relatively short (10 and 15 minutes), and longer tests may have revealed a greater potency of sleep loss compared to ethanol. As to MSLT sensitivity, in previous studies from this laboratory, the MSLT has consistently been found to be more sensitive to the effects of ethanol compared to performance testing.[6,23]

The slightly greater ethanol potency in amnestic effects despite the greater sedative effects with sleep loss is interesting. The memory test used in this study was a relatively simple and short test, comprising 4 paired associates for recall. A more exacting test may have revealed a greater difference in potency. The direction of the difference (ie, ethanol ingestion being greater than sleep loss) is consistent with the benzodiazepine literature. While, benzodiazepine-associated amnesia is related to level of sleepiness, there are studies in which amnesia in the absence of sleepiness has been shown.[4,24] It is argued that this amnesia is due to the direct GABA-related inhibitory effect of benzodiazepine at the hippocampus. Ethanol is also known to facilitate GABA function and thereby could also have direct amnestic effects at the hippocampus, beyond the amnestic effects due to the sedative effects of ethanol.[25]

Ethanol and sleep loss were equipotent in impairing psychomotor performance at the studied doses. Tracking deviations on the DAT were increased to the same extent by both ethanol and sleep loss, while the reaction-time parameters on this task did not show consistent effects. Subjects often concentrate on 1 component of the task at the expense of the other, which in this case was the tracking component. On the PVT, which does not require divided attention, reaction times were affected. But, interestingly, on this task, both ethanol and sleep loss slowed the fastest reaction times, parenthetically to the same degree and in a dose-related linear fashion, while lapses and the slowest reaction times were not consistently affected. This is not supportive of the "lapse" hypothesis of sleep-deprivation effects, which suggests lapses in performance occur as one becomes sleepier. What these data show is that best performance is degraded.

Laboratory studies have clearly shown the public health implications of these findings. This study found that 2 hours of sleep loss has an equipotency to a 0.54-g/kg ethanol dose. A previous study from this laboratory assessed the sedative effects, as measured with the MSLT, and the simulated-driving effects of ethanol.[23] The ethanol was administered to produce BrECs of .05%, and it reduced average daily sleep latency on the MSLT to 6.3 minutes. In the present study, the average daily sleep latency was 6.9 minutes in the 2 hours of sleep-loss condition. In the earlier study, the .05% BrEC impaired simulated driving. In this study, the 2 hours of sleep loss, with its 0.54-g/kg ethanol equivalency and potential BrEC of approximately .05%, would certainly have impaired simulated driving.

Finally, the differential perception of performance impairment across doses of sleep loss and ethanol must be discussed. While sleep loss and ethanol produced equal impairment on the performance tests, at the low and medium ethanol doses, participants did not perceive that impairment. Only at the high dose was performance impairment perceived. This finding is consistent with traffic-safety data that show ethanol-related automobile crashes in persons showing relatively low BrECs.[26] While a number of factors contribute to this public health problem, these data clearly indicate that poor perception of impairment is another contributory variable.

In summary at the studied doses, sleep loss was more potent than ethanol in its sedative effects, but sleep loss and ethanol were comparable in their effects on psychomotor performance. Ethanol produced greater memory deficits, and participants were less aware of their impairment.

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REFERENCES

1. Roehrs T, Roth T. Sleep, sleepiness, sleep disorders and alcohol use and abuse. Sleep Med Rev 2001;5:287-97.
2. Drake C, Roehrs T, Burduvali E, Bonahoom A, Rosekind M, Roth T. Effects of rapid versus slow accumulation of eight hours of sleep loss. Psychophysiology 2001;38:979-87.
3. Mintzer MZ, Guarino J, Kirk T, Roache JD, Griffiths RR. Ethanol and pentobarbital: Comparison of behavioral and subjective effects in sedative drug abusers. Exp Clin Psychopharm 1997;3:203-15.
4. Roehrs T, Roth T. Sleep-wake state and memory function. Sleep 2000;23:S64-8.
5. Bonnet M. Sleep deprivation. In: Kryger M, Roth T, Dement WC, eds. Principles and Practice of Sleep Medicine. 3rd ed. Philadelphia, Pa: WB Saunders Co; 2000:53-71.
6. Roehrs T, Zwyghuizen-Doorenbos A, Knox M, Moskowitz H, Roth T. Sedating effects of ethanol and time of drinking. Alcoh Clin & Exp Res 1992;16:553-7.
7. Hack MA, Choi SJ, Vijayapalan P, Davies RJO, Stradling JR. Comparison of the effects of sleep deprivation, alcohol and obstructive sleep apnoea (OSA) on simulated steering performance. Respir Med 2001;95:594-601.
8. Krull KR, Landgrave T, Smith LD, Parsons OA. The influence of alcohol and sleep deprivation on stimulus evaluation. Alcohol 1992;9:445-50.
9. Fairclough SH, Graham R. Impairment of driving performance caused by sleep deprivation or alcohol: a comparative study. Hum Fact 1999;41:118-28.
10. Williamson AM Feyer AM. Moderate sleep deprivation produces impairments in cognitive and motor performance equivalent to legally prescribed levels of alcohol intoxication. Occup Environ Med 2000;57:649-55.
11. Dawson D, Reid K. Fatigue, alcohol, and performance. Nature 1997;388:23-9.
12. Arnedt TJ, Wilde GJS, Munt PW, MacLean AW. How do prolonged wakefulness and alcohol compare in the decrements they produce on a simulated driving task? Accid Anal Prevent 2001;33:337-44.
13. Rechtschaffen A, Kales A. A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects. Washington DC: U.S. Government Printing Office, USPHS; 1968.
14. American Sleep Disorders Association. The International Classification of Sleep Disorders. Rochester, MN: American Sleep Disorders Association; 1992.
15. Carskadon MA, Dement WC, Mitler MM. Guidelines for the Multiple Sleep Latency Test (MSLT): a standard measure of sleepiness. Sleep 1986;9:519-24.
16. Dinges DF, Kribbs NB, Bates BL, Carlin MM. A very brief probed memory task: sensitivity to sleep loss. Sleep Res 1993;22:330.
17. Dinges DF, Pack F, Williams K, et al. Cumulative sleepiness, mood disturbance, and psychomotor vigilance performance decrements during a week of sleep restricted to 4-5 hours per night. Sleep 1997;20:267-77.
18. Roehrs T, Rosenthal L, Koshorek G, Mangano RM Roth T. Effects of zaleplon or triazolam with or without ethanol on human performance. Sleep Med 2001;2:323-32.
19. Roehrs T, Papineau K, Rosenthal L, Roth T. Sleepiness and the reinforcing and subjective effects of methylphenidate. Exp Clin Psychopharm 1999;7:145-50.
20. Roehrs T, Claiborue D, Knox M, Roth T. Residual sedating effects of ethanol. Alcohol Clin Exp Res 1994;18:831-4.
21. Zwyghuizen-Doorenbos A, Roehrs T, Schaefer M, Roth T. Test-retest reliability of the MSLT. Sleep 1988;11:562-5.
22. Finney DJ. Statistical Method in Biological Assay. 3rd ed. London: Charles Griffin & Co; 1978.
23. Roehrs T, Baere D, Zorick F, Roth T. Sleepiness and ethanol effects on simulated driving. Alcohol Clin Exp Res 1994;18:154-8.
24. Roth T, Roehrs T, Wittig R, Zorick F. Benzodiazepines and memory. Brit J Clin Pharmacol 1984;18:45S-9S.
25. Roehrs T, Roth T. Alcohol-induced sleepiness and memory function. Alcohol Health Res World 1995;19:130-5.
26. Moskowitz H, Burns MM, Williams AF. Skills performance at low blood alcohol levels. J Stud Alcohol 1985;46:482-5.