Effects of Alcohol on Sleep and the Sleep Electroencephalogram in Healthy Young Women

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Background: Although the association between sleep and alcohol has been of interest to scientists for decades, the effects of alcohol on sleep and sleep electroencephalogram (EEG) have not been extensively studied in women. Our specific aim was to determine whether sleep stage variables and/or spectral characteristics of the sleep EEG are altered by alcohol administration in women.

Methods: Changes of sleep and the sleep EEG were investigated after administration of a moderate dose of alcohol (0.49 g/kg) in the hour before bedtime compared with placebo in young healthy women. After approximately 2 weeks at home on a fixed 8.5- or 9-hour stabilization sleep schedule, sleep was continuously recorded by polysomnography for 3 consecutive nights [adaptation, placebo, alcohol (mean breath alcohol concentration 0.043 g/% before bedtime)] in the laboratory in 7 women (ages 22–25, mean = 23.5, SD = 1 year). Sleep stages were scored according to conventional criteria. Electroencephalogram power spectra of the bipolar derivations Fz/Cz (anterior) and Pz/Oz (posterior) were calculated using a fast Fourier transform routine.

Results: Only few changes in sleep and the sleep EEG were observed. Across the entire night rapid eye movement (REM) sleep decreased, while minutes of stage 4 sleep were increased in the first 2-hour interval on alcohol nights compared with placebo nights. Spectral analysis of the EEG showed increased power in the α range (9–11 Hz) during all-night non-REM (NREM) sleep in anterior derivations after alcohol compared with placebo. Differences in spectral EEG power were also present in 2-hour intervals of NREM sleep; in particular, EEG power was increased on the alcohol night for frequency bins within the α range in anterior derivations and within the δ range (3–4 Hz) in posterior derivations during the initial part of the night.

Conclusions: A moderate dose of alcohol just before bedtime resulted in a short-lived increase in sleep intensity. A limitation of the study, however, was that only a single dose of alcohol was used to examine the effects of alcohol on sleep.

Key Words: Sleep, Alcohol, Women, EEG Spectral Power.

THE ASSOCIATION BETWEEN sleep and alcohol has been of interest to scientists for decades. As summarized by Roehrs and Roth (2001) sleep studies in the 1960s and 1970s examining acute effects of alcohol consumption (doses ranging from 0.16 to 1.0 g/kg) on sleep of healthy nonalcoholic humans showed a number of consistent effects on subsequent sleep: decreased sleep latency, increased light sleep (stage 1) in the second half of the sleep episode, increased slow-wave sleep [SWS, non–rapid eye movement (NREM) sleep stages 3 and 4] in the first half of the night, and a dose-dependent decrease in rapid eye movement (REM) sleep in the first half of the night with a subsequent increase in REM sleep in the second half of the night. Chronic alcohol use and abuse were also shown to disrupt sleep, and furthermore, sleep remains severely disrupted in abstinent alcoholics (Drummond et al., 1998; Lester et al., 1973). Taken together, previous studies consistently demonstrate that acute and chronic use of alcohol alters the "macroarchitecture" of sleep as determined from visual assessment of sleep stages.

Electroencephalogram (EEG) spectral analysis examines the "microarchitecture" of sleep. Although spectral characteristics of the sleep EEG are correlated with sleep stages [e.g., SWS and slow-wave activity (SWA; EEG spectral power less than 4 Hz; Borbely et al., 1981)], the examination of sleep using both sleep staging and spectral EEG power analysis provides an in-depth assessment of sleep combining qualitative (i.e., sleep staging) and quantitative (i.e., spectral analysis) techniques.

Two studies by the group of Borbely have examined differences in spectral characteristics of the sleep EEG following alcohol administration. First, Dijk et al. (1992) administered 0.6 g/kg alcohol to 8 men (mean age = 24.3, range = 22–28) 35 minutes before bedtime. They reported

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an increase in spectral power in the slowest frequencies (0.25–1 Hz) and reductions in the 13.25- to 17-Hz range in the first 2 hours of NREM sleep. Second, Landolt and coworkers (1996) examined the spectral EEG during sleep in 10 healthy middle-aged men (mean age = 61.6 years) when alcohol (dose 0.55 g/kg) was administered 6 hours before bed. Although participants went to bed with a breath alcohol concentration (BrAC) of 0, Landolt and colleagues found a modest increase of EEG power in the slow δ (2 Hz or slower) frequencies in both NREM and REM sleep from placebo to alcohol nights, which was more prominent in frontal and central EEG derivations compared with parietal and occipital derivations. Strong conclusions regarding the effect of alcohol on sleep EEG spectra, however, are difficult to construct from these data due to the broad age range of participants and differences in the dosage schedule of alcohol administration and data analytic approaches.

We note that previous studies have investigated the effects of alcohol on sleep only in men. Clearly, men and women differ in the response to alcohol on a number of physiological parameters (Mumenthaler et al., 1999). For example, women absorb and metabolize alcohol differently compared with men, which may ultimately contribute to higher blood alcohol concentration levels on average in women compared with men, even when factors (i.e., body weight) known to affect blood alcohol levels are controlled (Mumenthaler et al., 1999). Alcohol and its effects on sleep and sleep EEG have not been extensively studied in women. A notable exception is the study of Williams et al. (1983) who examined sleep stage variables of 11 healthy young women (ages 18–21) across several in-lab nights in response to 2 doses of alcohol (0.5 and 0.75 g/kg) compared with placebo consumed approximately 1 hour before bedtime. These authors reported a significant dose-dependent increase in SWS in the first half of the night and a decrease of SWS in the second half, consistent with the findings of studies examining the effects of alcohol in men. They also observed a dose-dependent decrease in minutes in latency to sleep onset and in REM sleep percent for the entire night, an increase of percent stage 1 for the entire night, as well as increased minutes of stage 1 in the second half of the night and increased wake in the final hour of the night.

To date, no studies have reported spectral sleep EEG features in women in response to alcohol. Assessment of spectral EEG following presleep alcohol administration in women may be of interest because physiological effects (i.e., BAC levels are higher in women after an equivalent alcohol dose compared with men) of alcohol are stronger in women compared with men (Mumenthaler et al., 1999).

The goal of this analysis, therefore, was to examine the effects of a moderate dose of alcohol (0.49 g/kg) on sleep stages and spectral characteristics of the EEG in women. Our specific aim was to determine whether sleep stage variables and/or spectral characteristics of the sleep EEG are altered by alcohol administration in women.

MATERIALS AND METHODS

Participants

Eligibility Requirements and Screening. Individuals were recruited using flyers, as well as radio and newspaper advertisements. All potential participants were screened initially using a brief telephone questionnaire that asked questions regarding current or past medical/psychiatric conditions, drinking practices, and sleep habits. Participants were required to report consuming alcoholic beverages on at least 2 occasions per month and at least 2 drinks per occasion, averaging no more than 10 drinks per week. Personal and family history of alcohol abuse or dependence were prescreened with a Family History Screen (FHS; Weissman et al., 2000) and assessed according to *Diagnostic and Statistical Manual*—fourth edition (DSM-IV) criteria in all participants and their parents using a structured interview with relevant modules (DSM-IV alcohol abuse/dependence) from the structured clinical interview for DSM-IV (SCID; First et al., 1995). Volunteers classified with past or current alcohol abuse/dependence and participants with a positive parental history of alcohol abuse/dependence were excluded from the study.

Paper and pencil self-report inventories, including the Revised Symptom Checklist-90 (SCL-90-R; Derogatis, 1975) and Beck Depression Inventory-II (BDI-II; Beck, 1972), were used to exclude individuals with current major depression or a personal history of psychopathology (e.g., schizophrenia or bipolar disorder). Telephone interviews and questionnaires were used to exclude individuals with chronic medical conditions (e.g., diabetes or cancer), neurological disorders, or a family history of psychopathology. Blood tests were used to exclude pregnant women and individuals with abnormal liver function. Further, volunteers with a known sensitivity to alcohol or who were taking medications or drugs that affect the sleep/wake cycle or who smoked were excluded. Telephone screening interviews and questionnaires were used to assess current sleep habits and sleep disorders. Individuals with current sleep problems, irregular sleep patterns, travel beyond 2 time zones within 3 months before the scheduled in-lab nights, excessive daytime sleepiness, and/or a personal or family history of narcolepsy were excluded.

Seven healthy young women [ages 22–25, mean = 23.5, SD = 1 year and body mass index (BMI) 23.8–29.7, mean = 26.5, SD = 2.4] passed the screening procedures and participated in this study. The Lifespan Institutional Review Board for the Protection of Human Subjects approved the protocol for this study, and informed consent was obtained from all participants. Participants received monetary compensation for their participation.

Procedures

At-Home Protocol. All participants slept on a fixed 8.5- or 9-hour stabilization sleep schedule for approximately 2 weeks at home before coming into the laboratory. Schedules were determined based on the participants' usual sleep pattern and any social restrictions, such as hours of work or school. The 8.5-hour sleep schedule was the minimum allotted sleep time; the planned hours of sleep were extended for participants who reported usually sleeping longer. Adherence to the schedule was confirmed by actigraphy, sleep diary, and evening and morning phone calls to the lab's time-stamped answering machine; these data were examined after 1 week and on the first study day. Participants were asked to abstain entirely from alcohol, medications, drugs, and from caffeine within 12 hours before bedtime for the entire course of the study. Urine was collected for toxicology on the first in-lab night to confirm compliance. All participants meet compliance criteria.

In-Lab Protocol. Study participants came into the lab for 3 consecutive nights (adaptation, placebo, alcohol) after sleeping on the fixed schedule at home. Breath alcohol concentration levels were obtained using hand-held breath analyzer (AlcoSensor IV; Intoximeters, Inc,

St. Louis, MO) upon arrival each evening to confirm that participants had a BrAC of 0. All participants took a home-pregnancy test on the first in-lab night to confirm that they were not pregnant. Participants arrived approximately 5 hours before their scheduled bedtime on each in-lab night. Directly after arrival each evening, participants ate a meal (pizza) and were oriented to the study protocol. No food was consumed after the scheduled meal, and water was provided ad libitum throughout the protocol. Electrodes were attached to participant's head and face, and upon completion of the hookup on nights 2 and 3, participants went to their bedrooms to begin the placebo or alcohol protocol.

The first in-lab night (adaptation night) was used to screen for sleep disorders and to allow participants to adapt to sleeping in the laboratory. The second and third in-lab nights were used to compare sleep following placebo or alcohol administration. To control for participants' expectancies of effects of alcohol consumption, participants were not told they would receive a placebo beverage, but instead were told they would receive either a low or moderate dose of alcohol. Night 2 was always a placebo night and night 3 always an alcohol night, because alcohol may affect sleep even after it has been metabolized (see Landolt et al., 1996). Alcohol (night 3) or placebo (night 2) administration began 90 minutes before bedtime, as established for the at-home sleep schedule. The alcohol beverage was vodka (Smirnoff 80 proof) mixed with chilled tonic water in a 1:4 ratio, with a wedge of lime placed in the drink. The "placebo" beverage was a chilled tonic and lime drink of the same volume with 3 drops of vodka floating on the surface. The dose of alcohol was calculated taking into account body weight (0.49 g/kg) and was formulated to achieve a breath alcohol concentration of 0.05 g%. The dose of alcohol used in this protocol is considered to be a moderate dose equivalent to about 2 or 3 standard drinks.

During the alcohol administration, participants sat in bed at a 70° angle with no social interaction and were monitored with polysomnography (PSG) and video monitoring. Participants rinsed their mouths with mouthwash before they began drinking in an attempt to keep participants' blind to whether or not they had an alcoholic or placebo beverage. For both conditions (placebo and alcohol) the beverages were distributed equally into 3 glasses, and participants were instructed to drink these beverages within a 30-minute window (10 minutes for each drink). Participants were blind to alcohol or placebo condition. In the hour before bedtime following alcohol administration BrACs were taken at approximately 20-minute intervals.

Polysomnographic Recordings and Analysis

Sleep was continuously recorded by PSG in the laboratory for 3 consecutive nights (adaptation, placebo, alcohol) and monitored closely by a trained technician. Polysomnography included central and occipital referential EEG derivations (C3/A2 or C4/A1 and Oz/A2 or O2/A1) and frontal/central and parietal/occipital bipolar EEG derivations (Fz/Cz and Pz/Oz), along with right and left electrooculogram (EOG), electromyogram (EMG; mentalis, submentalis), and electrocardiogram (ECG). The locations of EEG electrode placements were determined using the international 10-20 system (Jasper, 1958). Respiration (oral/nasal thermocouple) and leg EMG were also recorded on the adaptation night to detect occurrence of sleep disorders related to breathing abnormalities or periodic limb movements. None was detected. Electroencephalogram signals were filtered with Grass Model 8 amplifiers (high-pass EEG filter, 0.3 Hz; low-pass EEG filter, 35 Hz; notch filter 60 Hz). The Albert Grass Heritage System (Astromed, Grass, West Warwick, RI) was used for all digital PSG recordings; EEG signals were digitized on-line (12 bit AD converter; butterworth filter, −12 dB/octave; low-pass filter, −6 dB at 35 Hz; time constant 1.0 second; storage—resolution of 128 Hz for the EEG).

Sleep stages were scored visually off-line in 30-second epochs using C3/A2, EOG, and EMG according to the criteria of Rechtschaffen and Kales (1968). Sleep stage variables assessed include: Time In Bed (TIB; minutes from lights off to lights on); Sleep Period Time (SPT; minutes from sleep onset to sleep offset); Total Sleep Time (TST; minutes of sleep scored within minutes of TIB); Latency to Sleep Onset (SOL; minutes from lights out to the first of 3 consecutive epochs of stage 1 sleep); Latency to Slow Wave Sleep Onset (minutes from sleep onset to first epoch of stage 3 or 4 sleep); REM latency (minutes from sleep onset to first epoch of REM sleep); Wake After Sleep Onset (WASO; minutes of wake within SPT); minutes of stages 1, 2, 3, 4, and REM; minutes of SWS (stages 3 and 4); number of awakenings (transitions from sleep to wake); Sleep Efficiency (TST/SPT×100). Sleep stage variables are presented in Tables 1 and 2. Time in bed was 508 (SD = 2) minutes and 509 (SD = 2) minutes on placebo and alcohol nights, respectively. For the analyses of 2-hour intervals, the first interval began at lights out.

Electroencephalogram spectral analysis was preformed after all 30-second epochs containing EEG artifacts (e.g., eye blinks, movement artifacts, or signal noise) were excluded by careful visual inspection of the EEG signals. The derivations Fz/Cz and Pz/Oz were subjected to spectral analysis off-line using a fast Fourier transform routine (FFT; Matlab, The MathWorks Inc., Natick, MA). Power spectra of consecutive 30-second epochs (Hanning window, averages of six 5-second epochs) were computed, resulting in a frequency resolution of 0.2 Hz. Power spectral data (μV^2) for 0.2-Hz bins were then averaged into 1-Hz bins from 1 to 25 Hz. The lowest 2 frequency bins (0.2 and 0.4 Hz) were excluded from the final 1-Hz bin analysis because of possible contamination by slow-frequency artifacts. Power spectral data were aligned with sleep stage data, and EEG power spectra were computed for the entire night, for NREM and REM sleep epochs across the night.

An additional analysis examined NREM sleep within 2-hour intervals. Sleep interval analysis examined EEG power spectra separately for NREM sleep within each of the first four 2-hour sleep intervals of the night. A sleep interval began with the first epoch of stage 2 sleep and included only epochs staged as NREM sleep (stages 2–4) within the 2-hour interval.

Statistical Analysis

The statistical package SPSS® was used for statistical analyses of sleep stage data. (Version 8.02, SAS Institute Inc., Cary, NC). All-night sleep stage variables (variables listed in Table 1) were analyzed using paired *t*-tests to compare all night stage variables on placebo night to those on alcohol night. Analyses for main effects of condition (alcohol, placebo), NREM sleep within a 2-hour interval (intervals 1–4), and the interactions of condition×interval were computed using MANOVA with condition and interval as within-subjects factors. Post hoc tests were performed using paired *t*-tests to detect significant differences for main effects of interval. A significance level was set at $p < 0.05$ for all analyses.

The statistical package SAS® was used for statistical analyses. Analyses for main effects of condition (alcohol, placebo), derivation (anterior, posterior), or 2-hour interval (1–4) and the interactions condition×derivation or condition×interval were computed using 2-way repeated-measures analysis of variance (rANOVA) with condition as within-subject factors separately for each frequency bin (1–25 Hz). All analyses of EEG spectral data were performed on log-transformed data. Post hoc tests were performed using 1-way rANOVA to detect significant differences on alcohol versus placebo nights for each frequency bin (1–25 Hz) and to detect significant differences for main effects of interval. A significance level was set at $p < 0.05$ for all analyses with Huynh–Feldt corrected degrees of freedom, but original *F* values are reported. Type I errors were avoided by considering contiguous bins that achieved statistical significance.

Table 1. Sleep Stage Variables (Minutes) for Young Women ($n = 7$) Following Alcohol or Placebo Administration 90 Minutes Before Bedtime. (See Methods for Definitions.)

	Mean (SD)	
	Placebo	Alcohol
Time in bed	508 (2)	509 (2)
Sleep period time	498 (14)	504 (3)
Total sleep time	478 (17)	478 (18)
Latency to sleep onset	4 (3)	5 (3)
Latency to SWS onset	14 (3)	11 (3)
REM latency	80 (21)	99 (43)
Wake after sleep onset	17 (15)	23 (17)
Stage 1	43 (40)	46 (18)
Stage 2	217 (44)	218 (39)
Stage 3	29 (13)	28 (11)
Stage 4	72 (23)	78 (30)
Slow-wave sleep	101 (30)	106 (35)
REM	116 (17)[a]	109 (17)[a]
Number of awakenings	7.3 (4.3)	6 (2.3)
% Sleep efficiency	96 (3)	95 (4)

[a]Paired *t*-tests: $t(6) = 2.6$; $p = 0.039$.

RESULTS

Breath Alcohol Levels

Breath alcohol concentration taken upon arrival and departure from the sleep lab confirmed a BrAC of 0 for all participants, as did BrAC readings from the placebo night. Scheduled BrAC levels closest to bedtime ranged from 0.031 to 0.053 g/% ($M = 0.043$ g/%, SD = 0.009 g/%) on the alcohol nights. The mean BrACs relative to bedtime on the alcohol night for the entire group ($n = 7$) (with the exception of the BrAC value at time 0, which includes only 5 participants) were 0.042 g/% (SD = 0.01 g/%,), 0.043 g/% (SD = 0.01 g/%,), 0.043 g/% (SD = 0.009 g/%), and 0.044 g/% (SD = 0.007 g/%), respectively, for 1 hour and 30, 15, and 0 minutes (i.e., bedtime) before bedtime.

All-Night Sleep Stage Variables

Visually scored sleep variables for placebo and alcohol nights are summarized in Table 1. The sleep stage variables from the placebo night in these participants are similar to published values for this age group (Acebo et al., 1996). Within-group analyses showed a statistically significant effect of alcohol for only one sleep stage variable: REM sleep amount was greater on the placebo ($M = 116$, SD = 17 minutes) than on the alcohol ($M = 109$, SD = 17 minutes) nights ($p = 0.039$).

Sleep Stage Variables for 2-Hour Intervals

Visually scored sleep variables for 2-hour intervals on placebo and alcohol nights are summarized in Table 2. No main effect of condition was found for any sleep stage 2-hour variable. A main effect of interval was found for minutes of stages 2–4 and REM sleep. These expected effects showed more SWS in early intervals and more stage 2 and REM sleep in later intervals. Post hoc tests comparing specific intervals are described in Table 2. An interaction of interval×condition was present only for minutes of stage 4 sleep: post hoc tests confirmed an increase in minutes of stage 4 sleep during the first 2-hour interval on the alcohol night compared with the first interval on the placebo night.

NREM Sleep EEG Spectra as a Function of Alcohol Administration and Derivation

Figure 1 shows absolute power density values presented for each night separately and then for the alcohol night scaled relative to the placebo night for all-night NREM sleep in anterior and posterior EEG derivations for placebo and alcohol nights. A main effect of derivation was found for frequency bins 1–3 Hz, reflecting greater spectral

Table 2. Stage Variables in Minutes for the First 4 Consecutive NREM Sleep Within 2-Hour Intervals Across the Night Following Alcohol or Placebo Administration 90 Minutes Before Bedtime ($n = 7$)

Stage variable (min)	Placebo mean (SD)				Alcohol mean (SD)			
Two-hour intervals	1	2	3	4	1	2	3	4
Stage 1	8 (7)	11 (13)	10 (13)	14 (16)	5 (4)	7 (5)	12 (7)	11 (8)
Stage 2[a]	39 (11)	54 (19)	58 (15)	46 (17)	39 (13)	57 (11)	53 (14)	62 (24)
Stage 3[b]	10 (5)	9 (8)	6 (6)	4 (2)	7 (3)	9 (7)	6 (4)	4 (4)
Stage 4[c,d]	46 (6)	19 (10)	7 (9)	6 (11)	58 (16)	11 (10)	7 (13)	6 (9)
REM[e]	10 (10)	21 (7)	30 (13)	47 (11)	5 (6)	30 (13)	31 (9)	35 (19)
SWS[f]	56 (8)	28 (10)	13 (13)	10 (12)	65 (17)	20 (14)	13 (14)	10 (12)
Wake	7 (7)	5 (9)	8 (16)	3 (6)	5 (5)	4 (5)	11 (13)	1 (2)

Two-way repeated measures ANOVA factor interval and condition for each stage variable.

[a]Main effect of interval for stage 2 sleep ($F = 8.25$, $p = 0.001$); Stage 2 interval 1< intervals 2, 3, and 4 ($p = 0.005$, $p = 0.003$, $p = 0.013$, respectively).

[b]Main effect of interval for stage 3 sleep ($F = 3.91$, $p = 0.026$); Stage 3 interval 1> interval 4 ($p = 0.043$) and interval 2> interval 4 ($p = 0.016$).

[c]Main effect of interval for stage 4 sleep ($F = 53.42$, $p < 0.001$); Stage 4 interval 1> intervals 2, 3, and 4 ($p < 0.001$ for all comparisons).

[d]Significant interval×condition interaction ($F = 3.54$, $p = 0.036$); Stage 4 interval 1 on Alcohol night >Stage 4 interval 1 on Placebo night.

[e]Main effect of interval for rapid eye movement (REM) sleep ($F = 21.77$, $p < 0.001$); REM interval 1< intervals 2, 3, and 4 ($p = 0.003$, $p = 0.001$, $p < 0.001$, respectively) and interval 4> intervals 2 and 3 ($p = 0.041$, $p < 0.001$, respectively).

[f]Main effect of interval for Slow Wave Sleep (SWS) ($F = 60.2$, $p < 0.001$); SWS interval 1> intervals 2, 3, and 4 ($p = 0.022$, $p < 0.001$, $p = 0.003$, respectively) and SWS interval 2> intervals 3 and 4 ($p = 0.016$ and $p = 0.009$, respectively).

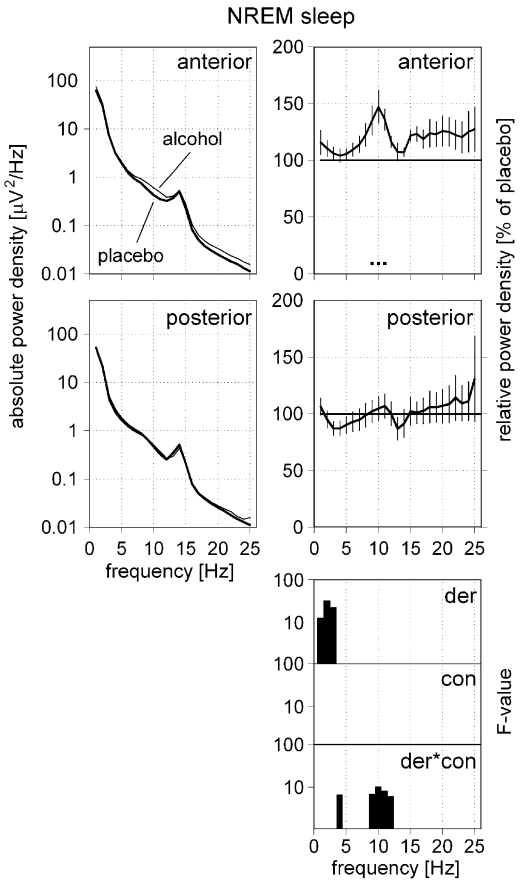

Fig. 1. *Left*: Mean all-night absolute electroencephalogram (EEG) power spectra for non–rapid eye movement (NREM) sleep (stages 2–4). Power density values are plotted for 1- to 25-Hz bins with the anterior lead in the upper panel and posterior lead in the lower panel; placebo night data is the thick line and alcohol night the thin line. *Right*: Mean NREM all-night EEG power spectra for each 1-Hz frequency bin of alcohol night expressed relative to the corresponding bin for the placebo night (placebo = 100%) in anterior (upper panel) and posterior (lower panel) derivations. Vertical lines represent ± 1 SD. Squares at the bottom of the figure indicate frequency bins in which absolute power density of alcohol night was significantly increased compared to placebo night ($p < 0.05$; 1-way repeated-measures ANOVA on log-transformed values). *Bottom Right*: *F*-values for frequency bins in which a significant ($p < 0.05$) main effect of derivation (der) or condition (con) and interaction of derivation × condition occurred.

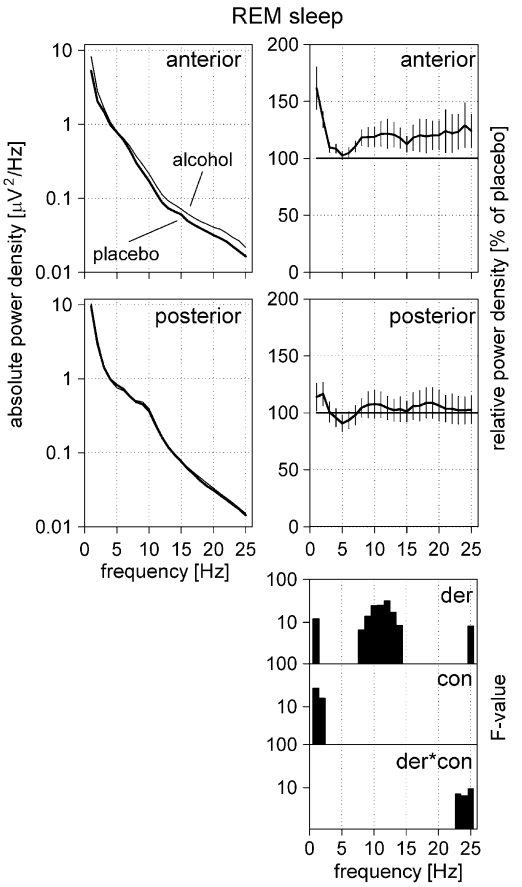

Fig. 2. Mean all-night absolute (*left*) and relative (*right*) electroencephalogram (EEG) power spectra in rapid eye movement sleep. Data are displayed as in Fig. 2.

power in the anterior than posterior derivation. No main effect of alcohol condition was found for any frequency bin. An interaction of derivation×condition was present for the 3-Hz bin and for the 8- to 11-Hz bins. Post hoc tests performed for these frequency bins showed no condition effects for the posterior EEG, but confirmed statistically significant increases of EEG power in NREM sleep for alcohol versus placebo in the anterior derivation for several frequency bins. Thus, EEG power was greater with alcohol in bins 9 to 11 Hz relative to placebo.

REM Sleep EEG Spectra as a Function of Alcohol Administration and Derivation

Figure 2 illustrates absolute power spectra values presented for each night separately and then for the alcohol night scaled relative to the placebo night for REM sleep on alcohol and placebo nights in anterior and posterior derivations. A main effect of derivation emerged for frequency bins at 1, 8 to 14, and 25 Hz, with higher spectral power for the more posterior derivation. A 2-way rANOVA with factors condition and derivation showed a main effect of condition for the 1- and 2-Hz bins due to an increase in spectral power on alcohol nights. An interaction of derivation × condition was also present for the 23- to 25-Hz bin; however, post hoc tests showed no significant bins for the interaction of derivation×condition.

NREM Sleep EEG Spectral Power in the Anterior Derivation Within 2-Hour Intervals

Interval analyses were carried out separately for anterior and posterior derivations using 2-way rANOVAs with factors interval (1–4) and condition (placebo, alcohol) for each frequency bin. The left panels of Fig. 3 illustrate power spectra data for each interval from the anterior derivation. A main effect of interval for the 1- to 13- and 15-Hz bins was observed. A main effect of condition was found for frequency bins 9–12, 15–17, and 19 Hz, reflecting greater power with alcohol. Further, a significant interaction of

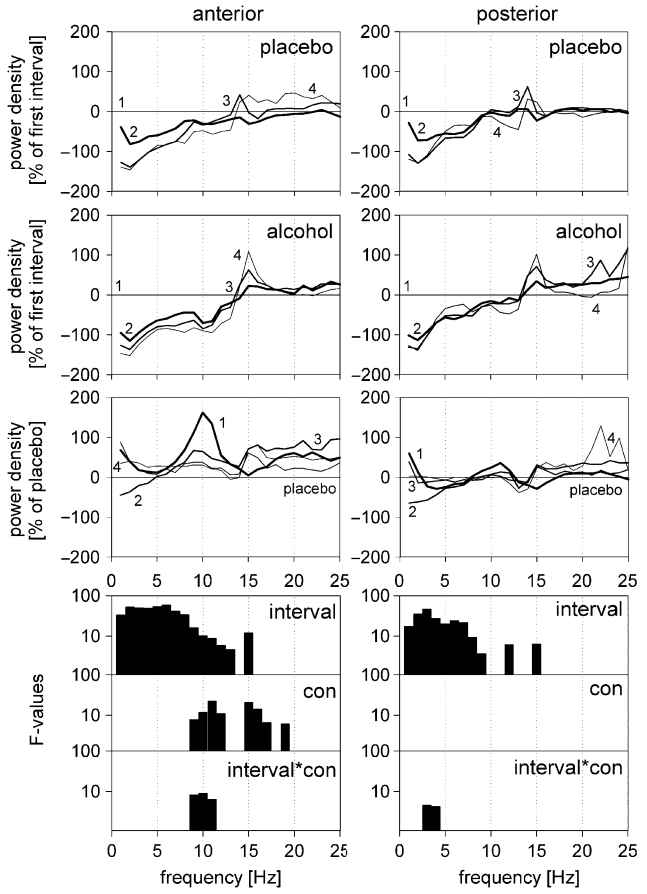

Fig. 3. Mean electroencephalogram (EEG) power spectra for the first 4 consecutive 2-hour non–rapid eye movement (NREM) sleep intervals. Power density values are plotted for 1- to 25-Hz bins. *Upper and second row panels*: NREM sleep within 2-hour sleep intervals 2, 3, and 4 expressed relative to the first intervals (Interval 1 = 100%) in anterior (*left panel*) and posterior (*right panel*) derivations on placebo night (*upper panels*) and alcohol night (*second row panels*). *Third row panels*: 2-hour NREM spectral EEG for intervals 1, 2, 3, and 4 on alcohol night relative to the corresponding interval on placebo night (100%) in anterior (*left panel*) and posterior (*right panel*) leads. *Lower panels*: *F*-values for frequency bins in which a significant ($p<0.05$) main effect of interval, condition, and interaction of interval×condition occurred for anterior (*left*) and posterior (*right*) derivations. Significant bins for main effects of interval for anterior derivation: bins 2 to 7 and 9 to 10 Hz interval 1 > 2, 3, 4; bins 11 and 15 Hz interval 1 > 2; bin 8 Hz interval 1 > 3 and 4; bin 13 Hz interval 1 > 4; bins 2 to 8 Hz interval 2 > 3, 4; bin 9 Hz interval 2 > 4; bin 15 Hz interval 4 > 2. Significant bins for main effects of interval for posterior derivation: bins 2 to 8 Hz interval 1 > 2,3,4; bin 1 Hz interval 1 > 3, 4; bin 14 Hz interval 1 < 3, 4; bins 1 to 5 and 7 Hz interval 2 > 3; bin 14 interval 3 > 2; bins 1 to 4 and 12 Hz interval 2 > 4; bins 6 and 14 Hz interval 4 > 2; bins 5 to 7 Hz interval 4 > 3; bin 12 Hz interval 3 > 4.

interval × condition was present in the 9- to 11-Hz bins. Post hoc tests performed on these bins revealed an increase on the alcohol night in power for frequency bins within the α range (9–11 Hz) in the first 2 hours of sleep and the 9-Hz bin for the second 2 hours of sleep (Fig. 3).

NREM Sleep EEG Spectral Power in the Posterior Derivation Within 2-Hour Intervals

An identical analysis was performed to assess the effects of NREM sleep within 2-hour intervals on the posterior derivation (Fig. 3, right panels). A main effect of interval was shown in the 1- to 9-, 12-, and the 15-Hz bins. No main effect of condition was found in the posterior derivation for any frequency bin. Further, an interaction of interval×condition was present in the 3- and 4-Hz bins; post hoc tests performed on these bins revealed a significant increase on placebo night in the 3- and 4-Hz bins for the second 2 hours of sleep (Fig. 3).

DISCUSSION

The purpose of the current study was to investigate changes in sleep and the sleep EEG after alcohol administration compared with placebo in young healthy women. Few changes in visually scored sleep stage variables were observed in our study. Across the entire night, a decrease in minutes of REM sleep was seen after alcohol administration. Examining sleep variables in 2-hour intervals, we found an increase in minutes of stage 4 sleep in the first 2-hour interval on alcohol nights compared with placebo nights. Spectral EEG analyses also demonstrated effects of a moderate dose of alcohol. Thus, an increase in EEG power during NREM sleep was shown in the α range (9- to 11-Hz bins) in anterior derivations after alcohol compared with placebo, and alcohol-related differences in spectral EEG were also present in 2-hour intervals of NREM sleep.

Our data in part confirm the previous literature. For example, the modest suppression of minutes of REM sleep on the alcohol night compared with placebo is consistent with the findings of Williams et al. (1983). Moreover, Dijk et al. (1992) reported an increase in minutes of stage 4 sleep in men in the first 2 hours of sleep following alcohol with a similar alcohol dose, time of administration, and age range of participants. Other studies examining the effects of alcohol on sleep in men, however, reported alcohol affecting several other sleep variables, such as sleep latency, light sleep, and SWS. One possible explanation for our finding that the sleep stage variables were only minimally changed following alcohol compared with studies in men is that alcohol dose and BrAC levels in our study were lower than in other studies (MacLean and Cairns, 1982; Rundell et al., 1972; Yules et al., 1967). For instance, these studies demonstrating a change in sleep stage variables with alcohol (even in women) used doses in the range of 0.75 g/kg, compared with 0.45 g/kg for the current study. Furthermore, the differences may also reflect methodological changes that exist between studies done in the 1960s through the 1980s and studies done today. We note that inadequate sleep can markedly change sleep stage variables, such as SWS (Borbely et al., 1981). In the present sleep study, participants kept a fixed at-home sleep schedule before the in-lab portion of the study in an attempt to eliminate changes in sleep stage variables and spectral characteristics as a result of inadequate sleep. Most of the early studies either did not stabilize, monitor, or describe prestudy sleep schedule.

Spectral EEG analyses revealed alcohol-related effects for NREM sleep and REM sleep across the night and at intervals during the night. An increase in EEG spectral power in association with alcohol was seen for the anterior derivation for frequency bins within the α range (9–11 Hz) for NREM sleep, and this finding of increased α activity during NREM sleep following alcohol administration is novel. Scheuler et al. (1993) proposed that NREM α activity is distinct from α activity observed during REM sleep and waking. This presumption was based on several observations including the predominance of NREM α in frontal brain regions compared with the occipital regions during waking (Finelli et al., 2001; Scheuler et al., 1993), an absence of change in NREM α activity in response to external stimuli in contrast to prearousal α activity, which was highly responsive (Scheuler et al., 1993), and dissipation of NREM α activity across sleep similarly to that of δ activity (δ is thought to be a physiological marker of sleep intensity), indicating that NREM α may be regulated by the same systems involved in the regulation of NREM sleep as opposed to those regulating arousal (Scheuler et al., 1993). In addition, Pivik and Harman (1995) have noted that frontocentral α is more indicative of sleep maintenance than sleep disruption. Further support for the idea that NREM α is regulated by the same systems that regulate NREM sleep was demonstrated by increased NREM α activity (specifically 9–10.75 Hz) in response to the administration of GABAergic substances specifically during the first cycle of sleep. GABAergic substances are also known to promote slow wave sleep and δ frequencies (Jones, 2000). Interestingly, GABA is thought to be involved in sedation following acute alcohol intoxication (Fromme and D'Amico, 1999); thus, the effects of alcohol on GABA may help to explain the increase in NREM α activity following a moderate dose of alcohol before bedtime.

Further, the finding that alcohol affected anterior derivations and not posterior derivations is consistent with the regional differences in neurotransmitter systems in the brain that are sensitive to the effects of alcohol (Fromme and D'Amico, 1999) and also supports the findings of Landolt et al. (1996). In addition, an increase in spectral power for frequency bins within the α range was present in NREM sleep within the first 2-hour interval of the sleep episode for the anterior derivation, which likely reflects greater alcohol activity early in the sleep episode.

Finally, we note that sleep EEG spectra differed as a function of derivation independent of alcohol administration. As observed by Werth et al. (1997) in healthy young men, we also found a higher NREM sleep EEG power in the frequency range from 1 to 3 Hz in anterior compared with posterior derivations, indicating a distinctive involvement of the frontal cortex in sleep homeostasis in women (Werth et al., 1997).

This study was not without limitations. Data from a small sample ($n = 7$) were used to examine the effects of alcohol on sleep. Another limitation is that menstrual phase was not taken into consideration when scheduling women for in-lab sleep assessment. In addition, the nights of sleep on which placebo and alcohol were administered before bedtime were consecutive, doses were not counterbalanced (i.e., placebo was always administered on the night before alcohol), and only a single dose of alcohol was used in this study to compare the effects of alcohol on sleep and sleep EEG in women. Women in this study were enrolled for participation as part of a larger study and to make it easier for them to complete the entire study consecutive nights were used. Alcohol was never given before placebo to avoid any residual effects of alcohol on the following night's sleep.

In sum, only few changes in sleep and the sleep EEG were observed. We are confident enough to conclude that a moderate dose of alcohol just before bedtime does not have profound effects on subsequent sleep, although the increase in frontal α EEG power early in the night indicates an enhancement of sleep intensity.

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