

SOSI Final Report

**AN OLFACTORY STIMULUS MODIFIES SUBSEQUENT SLEEP IN YOUNG MEN
AND WOMEN**

Namni Goel, Ph.D.

Department of Psychology

207 High Street, Judd Hall

Wesleyan University

Middletown, CT 06459

Names and Definitions of Sleep Parameters.

Sleep Measures	Definitions
Total Sleep Time (TST)	Amount of actual sleep time in the night
Sleep Period Time (SPT)	Total duration of time from sleep onset to final awakening
Total Wake Time (TWT)	Total duration of time spent awake in the night
Sleep Efficiency (SE)	Measure of overall efficiency of sleep
Sleep Maintenance Efficiency (SME)	Measure of efficiency of sleep after falling asleep initially
Sleep Onset	First three 30-second epochs of Stage 1 or first 30-second epoch of any other sleep stage
Sleep Onset Latency (SOL)	Duration to reach sleep onset after lights out; good measure of sleep initiation
Wake after Sleep Onset (WASO)	Total time spent awake after sleep onset; good measure of sleep maintenance
Stage 1 sleep	Transition stage from wakefulness to sleep; drowsiness
Stage 2 sleep	Light sleep
Stage 3 sleep	Initial stage of deep, restorative sleep
Stage 4 sleep	Deepest stage of sleep
Slow-wave sleep (SWS)	Deepest sleep (Stages 3+4 combined)
NREM sleep	Non-rapid-eye movement sleep (Stages 1-4 combined)
REM sleep	Rapid-eye movement sleep (stage when dreaming most often occurs; follows NREM sleep at beginning of the night)
NREM-REM cycle	NREM sleep followed by REM sleep; cycles are typically 90-120 minutes
Latency to REM	Time to reach first REM episode
Duration of each sleep stage or wake (minutes)	Amount of time (in minutes) spent in each of the sleep stages (or wake) from lights off to final awakening
Percentage of each sleep stage or wake	Amount of time spent in each of the sleep stages (or wake) as a percentage of SPT
Latency to each sleep stage or wake	Amount of time elapsed (in minutes) from lights off to the first occurrence of each sleep stage or wake

ABSTRACT

Study Objectives: Aromatherapy is an anecdotal method for modifying sleep and mood.

However, whether olfactory exposure to essential oils affects nighttime objective sleep remains to be rigorously tested. Previous studies also demonstrate superior olfactory abilities in women.

Therefore, this study investigated an olfactory stimulus' effects on subsequent sleep and assessed gender differences in such effects.

Design: Three consecutive overnight sessions: one adaptation, one stimulus and one control night (latter two nights were counterbalanced).

Setting: Experimental sleep laboratory.

Participants: Thirty-one young, healthy sleepers (16 men and 15 women, ages 18-30, mean \pm SD, 20.5 ± 2.4 y).

Interventions: Intermittent presentation (first two minutes of each 10 minutes) of lavender oil or a control from 2310h-2340h.

Measurements and Results: Polysomnographic recordings and self-rated sleepiness and mood questionnaires. Lavender significantly increased the percentage of slow-wave sleep (deep sleep). Vigor scores on the Profile of Mood States were higher the morning after lavender exposure, corroborating the restorative slow-wave sleep increase. Lavender increased stage 2 (light sleep) and decreased rapid-eye movement sleep and also decreased the amount of time to reach wake after first falling asleep (wake after sleep onset latency) in women, with opposite effects in men. Across nights, women showed better sleep efficiency than men: women fell asleep faster (shorter sleep onset latency), spent more time asleep and less time awake.

Conclusions: Lavender increases slow-wave sleep in all subjects and differentially affects wake after sleep onset latency, stage 2 sleep and rapid-eye movement sleep in men and women.

Overall, women show better objective sleep quality than men. Lavender serves as a mild sedative and a novel, nonphotic method for promoting deep, restorative sleep in young men and women and producing gender-dependent sleep effects.

INTRODUCTION

The physiological and psychological effects of aromatherapy, particularly the use of pure plant oils, known as essential oils, have been acknowledged worldwide in folk medicine and among health care professionals.¹⁻⁴ In addition to aromatherapy's long-standing popularity, it claims to significantly affect sleep and mood.² However, evidence that such oils can produce changes has been predominantly anecdotal, deriving from small trials and case studies.^{1,5} This is particularly true for aromatherapy's effects on human sleep; thus far, this topic has not been tested rigorously in controlled laboratory conditions.

Olfactory Stimuli and Sleep

Olfactory stimuli such as peppermint and pyridine, when presented during sleep produce physiological responses in young adults, despite a reduced arousal threshold during sleep compared to waking.^{6,7} In addition, studies have found sleep-promoting effects of odors when administered before and during sleep. For example, a handful of studies have found improved sleep, including decreased time awake, increased total sleep time and reduced daytime sleepiness (as reported by medical and nursing staff) following lavender oil presentation before and during sleep in elderly and demented subjects.⁸⁻¹¹ A few studies also have found similar effects using other essential oils, including bitter orange, chamomile, marjoram and cedar essence, in young and older adults.¹²⁻¹⁵ However, such studies were uncontrolled with small sample sizes and an absence of objective evaluations. Therefore, further investigations are necessary to determine odors' effects on objective sleep.

Sedative Effects of Lavender

Beyond the aforementioned studies that suggest lavender produces soporific effects on sleep, lavender's sedative and calming effects have been noted in various physiological measures during the waking state. Lavender lowers heart rate¹⁶ and blood pressure^{16,17} and produces changes in EEG frequency and contingent negative variation,¹⁸ suggesting increased drowsiness. Lavender also increases beta activity,^{19,20} decreases alpha activity²¹ and increases theta activity.^{22,23} Such findings concur with studies that found more relaxed mood states, assessed by self-report mood questionnaires, following lavender exposure.^{19, 24-27} In addition, lavender produces slower reaction times²⁸⁻³⁰ and poorer performance on cognitive tasks.^{19,31}

Gender Differences in Olfaction

Gender differences in olfactory performance have been investigated widely (see reviews^{32,33}). Studies have found that women surpass men on nonbiological odor detection,³⁴⁻³⁸ discrimination³⁸ and identification tests.³⁹⁻⁴⁵ Various odors also produce greater physiological responses in women than men,⁴⁶⁻⁴⁹ although one study found no differences⁵⁰ and another found greater effects in men.⁵¹

Gender Differences in Sleep

Similarly, there are gender differences in adult objective sleep measures (see review⁵²). In middle-aged and elderly populations, women show better sleep quality than men, including more SWS, more REM and fewer nighttime awakenings.⁵³⁻⁵⁸ Elderly women also show longer REM latencies than men.^{58,59}

A few studies also have detected polysomnographic gender differences in younger populations. Women show significantly fewer awakenings,⁶⁰ more stage 2 sleep⁶⁰ and shorter

REM latencies⁶¹ than men. Women also have less SWS during the second half of the night and a greater decrease in SWS from the first to the second half of the night.⁶² A number of studies, however, have not found significant gender differences in polysomnographic sleep measures.⁶³⁻⁶⁸

Furthermore, by employing spectral EEG sleep analysis, many of the aforementioned studies found significant gender differences in delta power during NREM sleep in younger subjects, with women showing greater power than men.^{62,64,65, 67-69} Other studies demonstrated significantly higher power density in the delta, theta, low alpha and high spindle frequency ranges,⁶⁶ larger low-frequency EEG amplitudes⁷⁰ and a higher percentage of spindles in the left frontal channel⁷¹ in women.

The three goals of this study were as follows: 1. to examine the effects of an olfactory stimulus using commercially available lavender oil on subsequent polysomnographic (PSG) sleep; 2. to investigate gender differences in such effects; 3. to assess overall gender differences in PSG sleep. We tested three specific hypotheses: 1. the olfactory stimulus (lavender oil), but not the control stimulus (distilled water), will promote sleep by increasing slow-wave sleep and shortening sleep onset latency when presented prior to bedtime in young men and women; 2. the olfactory stimulus will have gender-specific effects on sleep, with larger responses in young women than men; 3. young women will show better PSG sleep quality than young men.

METHODS

Subjects

Sixteen men and fifteen women, ages 18-30 (mean age \pm SD, 20.5 \pm 2.4 y) participated in this study. All subjects were in good physical and psychological health, were healthy sleepers, and were without current use of central nervous system medications. Three women were taking

oral contraceptives. Subjects with extreme morningness or eveningness, measured by the Morningness-Eveningness Questionnaire (see below), with difficulty in smelling odors, or with asthma or sinus problems were excluded during the initial interview. Subjects maintained a stable wake-up time and bedtime, documented by sleep logs for one week before study entry. Wesleyan University's Institutional Review Board approved this protocol and all procedures conformed to the Declaration of Helsinki. Subjects received monetary compensation for participation and signed written informed consent was obtained before study entry.

Polysomnographic Recordings

Central and occipital electroencephalographic (EEG), electrooculographic (EOG), and submental electromyographic (EMG) recordings were carried out from 2400h (lights off) to 0800h (lights on). During the adaptation night, subjects were screened for sleep pathologies, including apneas, oxygen desaturation and periodic limb movements by monitoring respiratory effort, nasal airflow, arterial oxygen saturation level, bilateral anterior tibialis EMG, and heart rate (EKG).

Sleep records were visually scored in 30-second epochs according to Rechtschaffen and Kales' standard scoring criteria by two trained scorers blind to the experimental conditions.⁷² Sleep parameters for the entire night, and the first (2400h-0400h) and second (0400h-0800h) half of each night were analyzed.

Subjective Sleepiness Scales

The Stanford Sleepiness Scale (SSS⁷³) quantifies the progressive, subjective stages of the sleep-alertness continuum, with a 7-point scale from 1 (feeling active, vital, alert, or wide awake)

to 7 (sleep onset soon; lost struggle to remain awake). The Karolinska Sleepiness Scale (KSS⁷⁴), a transient self-rated 9-point scale ranging from 1 (extremely alert) to 9 (extremely sleepy), also quantifies subjective changes in the sleepiness-alertness continuum.

Morningness-Eveningness Questionnaire (MEQ)

The English language Morningness-Eveningness Questionnaire (MEQ⁷⁵) is a 19-item self-rated instrument that assesses the timing of preferred sleep habits and peak performance on various tasks, and classifies subjects on a morningness-eveningness continuum as chronotypes. Such chronotypes are differentiated by objective measures of circadian rhythms and sleep (see reviews⁷⁶⁻⁷⁸). Our study's chronotype distribution was as follows: definitely morning type (score range: 70-86, N=0); moderately morning type (59-69, N=8; 25.8%); neither type (42-58, N=14; 45.2%); moderately evening type (31-41, N=9; 29.0%); definitely evening type (16-30, N=0).

Profile of Mood States Questionnaire (POMS)

The Profile of Mood States Questionnaire (POMS⁷⁹) is a 65-item self-report scale that assesses transient affective states in response to various stimuli including olfactory cues.⁸⁰⁻⁸⁴ Furthermore, the POMS has been validated in many populations and study designs, including repeated measures (see review⁸³) and sleep studies.⁸⁵⁻⁸⁷ Each item is rated on a scale from 0 (not at all) to 4 (extremely), on one of six factors: depression-dejection (Depression), tension-anxiety (Tension), anger-hostility (Anger), confusion-bewilderment (Confusion), vigor-activity (Vigor), fatigue-inertia (Fatigue). The total score for each factor is calculated by adding together the respective set of adjectives corresponding to that factor. The total mood disturbance score

(TMD), a global estimate of affective state, derives from summing the factors together, with vigor-activity weighted negatively.

Participant Information Questionnaire (PIQ)

The PIQ assesses demographic information including age and smoking habits (smokers, N=4; non-smokers, N=27). Menstrual cycle dates and length, and birth control history also were assessed for women. All women were normally menstruating.

Odorant

The olfactory stimulus was commercially available lavender oil (International Fragrance and Technology, Inc., Canton, GA). The lavender oil contained a natural lavender base component to which constituents were added; it did not contain solvent materials (verified by gas chromatography). This particular lavender oil was validated externally as a sedative in a similar group of subjects derived from the same college population: in that study, lavender oil increased fatigue and confusion and decreased vigor on the POMS compared to distilled water.²⁴ Lavender has been rated as neutral to mildly pleasant and neutral on ratings of familiarity, intensity and irritability.^{30, 88-94}

Procedure

Subjects slept in the laboratory for three consecutive overnight sessions (see Figure 1). Each session lasted from approximately 2100h to 0800h. On the second and third intervening days, subjects were allowed to leave the laboratory between 0800h to 2100h and perform their habitual activities. On these study days, subjects refrained from napping and exercise, and from

alcohol or caffeine intake. In addition, subjects were not allowed to wear scented products (e.g., perfume, lotion) or to eat or drink for at least one hour before the testing sessions.

Electrode placement for polysomnographic recordings occurred at 2100h for all nights. After electrode placement, subjects engaged in recreational activities until bedtime (2400h) on the first night and until 2310h on the second and third nights. Polysomnographic recording of sleep was performed from 2400h to 0800h each night. Subjects remained in bed if they awakened before 0800h.

During the second and third nights, subjects received either the experimental or control session. The order of the experimental and control sessions was counterbalanced; furthermore, gender also was counterbalanced within the order assignment. Of the 31 subjects, 16 (9 men, 7 women) received the odor stimulus first, whereas 15 (7 men, 8 women) received the control first. During the experimental session, subjects received an intermittent olfactory stimulus from 2310h to 2340h. The stimulus was presented for the first two minutes of each 10-minute period (2310h, 2320h, 2330h and 2340h). Subjects held the lavender oil vial at chest level and breathed normally with their eyes closed. They were required to remain awake and no other competing stimuli were allowed during testing. The control session was identical to the experimental session except that subjects held a vial of distilled water. In the control session, subjects were told that they were receiving an odor that may be diluted as to be undetectable. The SSS and KSS were administered at 2350h and 0800h on all nights; the POMS was administered at 2300h, 2312h, 2342h and 0800h on the stimulus and control nights.

--Insert Figure 1 about here--

Statistical Analyses

Repeated measures analyses of variance, with gender and session order as between-subjects factors examined differences in PSG sleep measures, KSS/SSS scores and POMS scores across sessions. *Post-hoc* paired *t*-tests analyzed significant interactions for PSG measures and for POMS scores. The magnitude of between-group differences in PSG measures and POMS scores was expressed as effect size, *d*, the standardized difference between means (*d*=0.3, small; 0.5, medium; 0.8, large). Pearson product-moment correlation coefficient analyses (*r*) quantified the various relationships between KSS, SSS, POMS and PSG sleep measures. Data are presented as mean \pm SD; $P \leq .05$ was considered significant for all statistical analyses.

RESULTS

Polysomnographic (PSG) Sleep

Session Differences

Stimulus vs. Control. Across the whole night, lavender odor significantly increased SWS (stages 3 and 4) %SPT (6.94% \pm 4.11% vs. 5.72% \pm 4.24%; $F_{1,27}=10.41$, $P < .005$, $d=.29$; see Figure 2 and Table 1) and SWS duration compared to the control (32.47 \pm 19.43 vs. 26.69 \pm 20.04; $F_{1,27}=10.03$, $P < .005$, $d=.29$). More specifically, stage 3 duration (25.42 \pm 10.47 vs. 21.31 \pm 12.63; $F_{1,27}=7.22$, $P < .05$, $d=.35$), stage 3 %SPT (5.44% \pm 2.20% vs. 4.57% \pm 2.67%; $F_{1,27}=7.37$, $P < .05$, $d=.36$) and stage 4 %SPT (1.50% \pm 2.70% vs. 1.14% \pm 2.22%; $F_{1,27}=4.15$, $P=.05$, $d=.15$; see Table 1) were significantly higher in the stimulus than control session.

During the first half of the night, SWS duration (30.32 \pm 18.00 vs. 25.79 \pm 19.51; $F_{1,27}=5.19$, $P < .05$, $d=.24$), SWS %SPT (13.29% \pm 7.66% vs. 11.48% \pm 8.47%; $F_{1,27}=4.79$, $P < .05$, $d=.22$) and stage 4 %SPT (3.05% \pm 5.46% vs. 2.34% \pm 4.61%; $F_{1,27}=4.12$, $P=.05$, $d=.14$) were

significantly higher in the stimulus than control session. More specifically, SWS duration was significantly longer during the first NREM-REM cycle in the stimulus session (24.50 ± 16.00 vs. 20.35 ± 15.59 ; $F_{1,27}=4.33$, $P=.05$, $d=.26$).

Although there were no significant differences in sleep measures for the second half of the night, several measures showed trends toward significance in the same direction as those in the first half of the night: stage 3 %SPT ($.86\% \pm 1.75\%$ vs. $.35\% \pm .52\%$; $F_{1,27}=4.45$, $P=.08$, $d=.40$), SWS duration (2.08 ± 4.25 vs. $.86 \pm 1.25$; $F_{1,27}=3.17$, $P=.09$, $d=.39$) and SWS %SPT ($.86\% \pm 1.75\%$ vs. $.35\% \pm .52\%$; $F_{1,27}=3.24$, $P=.08$, $d=.40$).

--Insert Figure 2 and Table 1 about here--

First-Night Effects. Table 1 illustrates significant differences in whole night PSG sleep measures across the adaptation, stimulus and control nights for all subjects.

Session \times Gender Differences

For the whole night, there was a significant session \times gender interaction for WASO latency ($F_{1,10}=10.07$, $P <.01$): women showed decreased WASO latency while men showed increased WASO latency in the stimulus compared to the control session. There were no significant differences in WASO latency between the stimulus and control sessions for either gender (men, 245.33 ± 112.25 vs. 243.27 ± 112.97 ; $t_8=.38$, $P=.71$, $d=.02$; women, 94.90 ± 88.03 vs. 186.63 ± 156.68 ; $t_4=1.00$, $P=.38$, $d=.68$; see Figure 3A). Women, however, showed a significantly shorter WASO latency than men in the stimulus session ($F_{1,13}=6.62$, $P <.05$; $d=1.44$).

In addition, for the first half of the night, there were significant session \times gender interactions for stage 2 duration ($F_{1,27}=4.51$, $P <.05$) and stage 2 %SPT ($F_{1,27}=14.34$, $P <.001$).

There were no significant differences in stage 2 duration between the control and stimulus sessions for either gender (men, 161.41 ± 23.61 vs. 149.44 ± 23.66 , $t_{15}=1.58$, $P=.14$, $d=.51$; women, 158.87 ± 28.08 vs. 166.43 ± 22.22 , $t_{14}=1.47$, $P=.16$, $d=.30$). However, stage 2 %SPT was significantly lower in the stimulus than control session for men ($67.93\% \pm 9.05\%$ vs. $75.08\% \pm 6.73\%$; $t_{15}=4.41$, $P < .001$, $d=.90$; see Figure 3B), while it did not differ significantly between sessions for women ($71.53\% \pm 9.59\%$ vs. $69.16\% \pm 11.01\%$; $t_{14}=1.22$, $P=.24$, $d=.23$). There were no significant gender differences in stage 2 variables for either session night.

REM duration ($F_{1,27}=7.03$, $P < .05$) and REM %SPT ($F_{1,27}=7.25$, $P < .05$) also showed significant session \times gender interactions. Lavender significantly increased REM duration (32.44 ± 13.35 vs. 23.31 ± 17.86 ; $t_{15}=2.30$, $P < .05$, $d=.58$) and REM %SPT ($14.85\% \pm 6.35\%$ vs. $10.83\% \pm 8.52\%$; $t_{15}=2.10$, $P=.05$, $d=.54$; see Figure 3C) compared to the control in men, while these differences were not significant for women (REM duration, 29.60 ± 15.14 vs. 35.53 ± 9.79 , $t_{14}=1.24$, $P=.23$, $d=.47$; REM %SPT, $12.69\% \pm 6.44\%$ vs. $15.61\% \pm 4.79\%$, $t_{14}=1.42$, $P=.18$, $d=.51$; see Figure 3C). There were no significant gender differences in REM variables for either session night. In addition, there were no significant interactions in PSG measures for the second half of the night.

--Insert Figure 3 about here--

Gender Differences

There were a number of significant gender differences in sleep across all three nights. Women showed significantly longer TST (463.05 ± 12.92 vs. 444.18 ± 23.36 ; $F_{1,26}=6.10$, $P < .05$, $d=.98$; see Figure 4A), longer SPT (469.40 ± 8.36 vs. 453.90 ± 17.41 ; $F_{1,26}=8.01$, $P < .01$,

$d=1.11$; see Figure 4B) and better sleep efficiency than men ($96.64\% \pm 2.28\%$ vs. $92.98\% \pm 4.76\%$; $F_{1,26}=5.82$, $P < .05$, $d=.96$; see Figure 4C). Women also had significantly less TWT than men (15.77 ± 10.77 vs. 33.54 ± 22.76 ; $F_{1,26}=6.03$, $P < .05$, $d=.98$; see Figure 4D). Sleep onset latency was significantly shorter for women than men (9.31 ± 5.64 vs. 23.23 ± 16.27 ; $F_{1,26}=8.09$, $P < .01$, $d=1.11$; see Figure 4E), as were latencies to stage 1 (9.31 ± 5.64 vs. 23.35 ± 16.26 ; $F_{1,26}=8.22$, $P < .01$, $d= 1.12$), stage 2 (13.70 ± 6.75 vs. 28.09 ± 16.54 ; $F_{1,26}=8.05$, $P < .01$, $d= 1.11$) and stage 3 sleep (47.64 ± 17.24 vs. 71.00 ± 31.03 ; $F_{1,26}=5.21$, $P < .05$, $d= .91$). Beyond those reported for the whole night, there were no additional gender differences in sleep measures for the first or second half of the night.

--Insert Figure 4 about here--

Session Order Differences

There were no significant session order differences in PSG measures for the whole night, or for the first or second half of the night.

Subjective Sleepiness

SSS and KSS scores did not differ significantly across session nights or between morning and evening (see Figure 5). Men had significantly higher SSS scores than women across all three nights ($4.01 \pm .41$ vs. $3.50 \pm .71$; $F_{1,19}=5.38$, $P < .05$, $d=.89$), although men and women did not differ significantly in SSS and KSS scores between each corresponding session night or between morning and evening.

There were a number of positive, significant correlations between the morning and evening KSS and SSS scores for the control and stimulus sessions (see Table 2). In addition, there were positive, significant correlations in KSS scores between the adaptation and control evening sessions ($r=.48$, $P < .05$) and between the adaptation and stimulus evening sessions ($r=.43$, $P < .05$), but not between the stimulus and control evening sessions or between morning sessions. Similarly, there were positive, significant correlations in SSS scores between the adaptation and control evening sessions ($r=.39$, $P < .05$), the adaptation and stimulus evening sessions ($r=.38$, $P < .05$) and the control and stimulus morning sessions ($r=.42$, $P < .05$). However, there were no significant correlations between the stimulus and control evening sessions or between the other morning sessions.

--Insert Figure 5 and Table 2 about here--

Fatigue and vigor scores on the POMS showed significant positive and negative correlations, respectively with KSS and SSS scores. Higher vigor (SSS: $r=-.50$, $P < .01$) and lower fatigue (SSS: $r=.44$, $P < .05$; KSS: $r=.56$, $P < .01$) both significantly related to decreased sleepiness in the morning following the control session. Similar relationships to sleepiness were observed in the morning following the stimulus session for vigor (SSS: $r=-.55$, $P < .01$; KSS: $r=-.64$, $P < .01$) and fatigue (SSS: $r=.63$, $P < .01$; KSS: $r=.77$, $P < .01$) and in the evening of the stimulus session [vigor (SSS: $r=-.58$, $P < .01$; KSS: $r=-.77$, $P < .01$) and fatigue (SSS: $r=.39$, $P < .05$; KSS: $r=.51$, $P < .01$)]. However, these relationships were not found in the evening of the control session.

In addition, vigor in the morning following the stimulus negatively related to evening stimulus KSS ($r = -.47$, $P < .05$) and SSS ($r = -.41$, $P < .05$) scores. Higher anger ($r = .40$, $P < .05$) and more depression ($r = .49$, $P < .05$) in the morning following the stimulus session positively correlated with increased sleepiness on the SSS at the same time point. Similarly, higher confusion in the morning following the stimulus session positively correlated with increased sleepiness on the KSS at the same time point ($r = .38$, $P < .05$). Higher tension scores in the evening of the control session also positively correlated with SSS scores the next morning ($r = .38$, $P < .05$).

Table 3 illustrates significant correlations between POMS scores and sleep measures in the stimulus and control sessions. Similarly, Table 4 illustrates significant correlations between KSS and SSS scores and sleep measures in the stimulus and control sessions.

--Insert Tables 3 and 4 about here--

Subjective Mood: POMS Measures

Vigor. Vigor scores showed a significant session night \times time interaction ($F_{3,66} = 3.93$, $P < .05$). Vigor scores were significantly higher at 0800h in the stimulus than control session (5.72 ± 6.28 vs. 4.03 ± 3.83 ; $t_{26} = 2.44$, $P < .05$, $d = .32$; see Figure 6A). In the stimulus session, compared to 2300h (8.83 ± 5.60), all subjects were significantly less vigorous at 2312h (6.97 ± 4.92 ; $t_{29} = 3.30$, $P < .01$, $d = .35$), 2342 (4.94 ± 4.87 ; $t_{29} = 6.13$, $P < .001$, $d = .74$) and 0800h (5.72 ± 6.28 ; $t_{27} = 2.42$, $P < .05$, $d = .52$; see Figure 6A). All subjects also were significantly less vigorous at 2342h than 2312h ($t_{30} = 4.77$, $P < .001$, $d = .41$). Similarly, in the control session, compared to 2300h (9.81 ± 6.46), all subjects were significantly less vigorous at 2312h (6.97 ± 5.56 ; $t_{30} = 5.00$,

$P < .001$, $d = .47$), 2342h (4.90 ± 4.65 ; $t_{30} = 6.03$, $P < .001$, $d = .87$) and 0800h (4.03 ± 3.83 ; $t_{28} = 5.64$, $P < .001$, $d = 1.08$; see Figure 6A). All subjects also were significantly less vigorous at 2342h ($t_{30} = 3.53$, $P = .001$, $d = .43$) and 0800h ($t_{28} = 2.82$, $P < .01$, $d = .66$) than at 2312h. There were no significant gender or session order differences.

TMD. TMD scores showed a significant gender \times time interaction ($F_{3,66} = 2.77$, $P < .05$). Compared to 2300h (1.07 ± 10.24), men had significantly higher TMD scores at 2342h (5.94 ± 10.73 ; $t_{14} = 2.32$, $P < .05$, $d = .46$) and 0800h (10.32 ± 9.40 ; $t_{12} = 2.45$, $P < .05$, $d = .94$; see Figure 6B). Similarly, compared to 2312h (1.47 ± 10.52), men also had significantly higher TMD scores at 2342h ($t_{15} = 4.25$, $P = .001$, $d = .42$) and 0800h ($t_{13} = 2.67$, $P < .05$, $d = .88$). Compared to 2300h (-2.27 ± 11.50), women had significantly higher TMD scores at 2312h (3.47 ± 8.49 ; $t_{14} = 3.59$, $P < .01$, $d = 1.01$) and 2342h (8.77 ± 9.56 ; $t_{14} = 4.86$, $P < .001$, $d = 1.04$; see Figure 6B). Similarly, compared to 2312h, women also had significantly higher TMD scores at 2342h ($t_{14} = 3.99$, $P = .001$, $d = .59$). There were no significant session night or session order differences.

Depression. Depression scores showed a significant difference across time ($F_{3,69} = 5.38$, $P < .01$). Compared to 0800h ($.86 \pm .92$), all subjects were significantly more depressed at 2300h (1.92 ± 1.88 ; $t_{26} = 3.48$, $P < .01$, $d = .71$), 2312h (1.42 ± 1.90 ; $t_{27} = 2.12$, $P < .05$, $d = .37$) and 2342h (1.45 ± 1.73 ; $t_{27} = 2.42$, $P < .05$, $d = .42$; see Figure 7A). There were no significant gender, session night or session order differences.

Fatigue. Fatigue scores showed a significant difference across time ($F_{3,66} = 8.44$, $P < .001$). Compared to 2342h (8.60 ± 4.43), all subjects were significantly less fatigued at 2300h (5.38 ± 3.83 ; $t_{29} = 5.11$, $P < .001$, $d = .78$), 2312h (6.23 ± 4.03 ; $t_{30} = 5.91$, $P < .001$, $d = .56$) and 0800h (6.52 ± 4.65 ; $t_{26} = 2.91$, $P < .01$, $d = .46$; see Figure 7B). There were no significant gender, session night or session order differences.

Confusion. Confusion scores showed a significant difference across time ($F_{3,69}=4.24$, $P < .01$). Compared to 2300h ($.73 \pm 1.92$), all subjects were significantly more confused at 2312h (1.32 ± 2.11 ; $t_{29}=2.33$, $P < .05$, $d=.29$), 2342h (1.92 ± 2.35 ; $t_{29}=3.40$, $P < .01$, $d=.55$) and 0800h (2.55 ± 2.88 ; $t_{26}=2.66$, $P < .05$, $d=.75$; see Figure 7C). Similarly, all subjects were significantly more confused at 2342h than 2312h ($t_{30}=2.71$, $P < .05$, $d=.27$). There were no significant gender, session night or session order differences.

Anger. There were no significant gender, session night, session order or time differences in anger scores.

Tension. There were no significant gender, session night, session order or time differences in tension scores.

--Insert Figures 6 and 7 about here--

DISCUSSION

This study demonstrates sleep-promoting effects of lavender odor on subsequent sleep in young men and women. A significant increase in slow-wave sleep following evening presentation of lavender indicates that subjects spent more time in the deep, more restful stages of sleep compared to the no odor condition. Higher vigor scores on the Profile of Mood States in the morning after lavender exposure corroborate this restorative slow-wave sleep increase. In addition, the differential effects of lavender odor on sleep between men and women and overall gender differences in polysomnographic sleep underscore gender as an important variable in future sleep research using young populations.

Effects of Lavender Odor on Polysomnographic Sleep

The slow-wave sleep increase in this study corroborates previous findings of improved sleep quality in elderly and demented subjects following lavender presentation before and during sleep⁸⁻¹¹ and also following other odors.¹²⁻¹⁵ While these earlier studies contained methodological drawbacks, our results confirm lavender's sleep-promoting effects using a larger sample size and objective evaluations of sleep. The slow-wave sleep increase resulting from lavender exposure indicates that this odor has sedative effects, an interpretation supported by previous findings of reduced blood pressure and heart rate during lavender administration.^{16,17}

Contrary to our prediction, lavender did not shorten sleep onset latency. The absence of lavender's effects on sleep onset latency could be attributed to the fact that all subjects were healthy sleepers and on average, fell asleep approximately fifteen minutes after lights off in the control and stimulus sessions. Thus, differences in sleep onset latencies in our subjects may have been difficult to detect due to a basement effect.

Lavender would likely produce significant effects on sleep onset latency and other polysomnographic measures in subjects with initial insomnia or in older adults. Indeed, other mild nonpharmacologic sedatives, such as valerian, show individual differences in effectiveness, with greater responses in habitually poor or irregular sleepers, including the elderly.⁹⁵ Beyond these groups, lavender also may benefit depressed subjects who characteristically show a reduction in slow-wave sleep, among other sleep changes (reviewed in⁹⁶). Indeed, our laboratory recently demonstrated increased olfactory discrimination to lavender in depressed young adults.²⁴ Finally, lavender could be systematically used to promote sleep in critically ill or hospitalized patients, since both groups show anecdotal benefits from aromatherapy.⁹⁷⁻⁹⁹

Increases in slow-wave sleep duration, slow-wave sleep % sleep period time and stage 4 % sleep period time were found during the first, but not second half of the night. Moreover, such differences in slow-wave sleep duration were predominantly during the first non-rapid-eye movement/rapid-eye movement cycle. These results indicate that lavender's effects on subsequent restorative sleep do not persist across the night. Given its route of administration, lavender likely is absorbed quickly, exerting immediate, transient effects. Moreover, an absence of change in morning sleepiness scores suggests that lavender does not produce "hangover" effects the next day.

Slow-wave sleep changes conceivably could be due to prior sleep history, including prior wake duration.¹⁰⁰ Although there were differences in slow-wave sleep variables for the first night compared with the control and stimulus nights, the amount and percentage of slow-wave sleep each night were below those of normal young adults.¹⁰⁰ Thus, our subjects likely were not sleep deprived before study entry and the slow-wave sleep increase specifically in the stimulus session unlikely represents a rebound effect from the first night.

We found a first-night effect for a number of sleep measures. Our results corroborate those from other laboratory studies using nondepressed subjects that reported decreased sleep efficiency, increased wakefulness, reductions in rapid-eye movement and non-rapid-eye movement sleep, and longer latencies to rapid-eye movement and non-rapid-eye movement sleep stages during the first night.¹⁰¹⁻¹⁰⁶ However, they contrast other studies that failed to find such first-night disruptive sleep effects.^{107,108} Environmental sleeping conditions and increased arousal and vigilance may underlie such disruptions during the adaptation night.

Differential Effects of Lavender on Sleep in Men and Women

The significant session \times gender interactions for wake after sleep onset latency, stage 2 and rapid-eye movement sleep duration may reflect differences in odor abilities between men and women. Many studies have shown superior odor sensitivity, discrimination and identification in women (see reviews^{32,33}). Although the differences in stage 2 % sleep period time and rapid-eye movement duration and % sleep period time between the lavender and control sessions were significant in men, women also showed small to medium effect sizes for wake after sleep onset latency, stage 2 duration, rapid-eye movement duration and % sleep period time. Neural activation differences may explain why these sleep stages show opposite changes in men and women. For example, several studies have found gender differences in activation following odor exposure: odors activated different structures¹⁰⁹ or produced greater responses in women⁴⁶⁻⁴⁹ or in men.⁵¹ There also may be a chemical (systemic/non-perceptual) difference between genders for lavender.

In contrast to sleep measures, we did not find gender differences in mood change following lavender exposure, in concurrence with a previous study from our laboratory using the same odor and self-rated questionnaire.²⁴ Thus, collectively our results suggest that the gender differences in sleep are mediated by physiological and not psychological changes.

Gender Differences in Sleep

Significant gender differences in polysomnographic sleep, with large effect sizes, existed across all three nights. Longer sleep period time, shorter total wake time, greater sleep efficiency, and shorter latencies to sleep onset and the non-rapid-eye movement sleep stages demonstrate better sleep quality in women than men. Our data contrast with several studies that have failed to

detect gender differences in younger populations using polysomnography.⁶³⁻⁶⁸ They are, however, in agreement with data from middle-aged and elderly populations.⁵³⁻⁵⁸

Since the gender differences in sleep measures persisted across all three nights, they cannot be explained by sleep deprivation incurred from the adaptation night leading to better rebound sleep in women on the second and third nights. The gender differences, however, conceivably may be due to the effects of cyclical levels of reproductive hormones, including estrogen, progesterone and luteinizing hormone on sleep in women.¹¹⁰⁻¹¹³ However, since an equal number of women were in the luteal (N=6) or follicular (N=6) phases of their menstrual cycle, the reported gender differences cannot be due exclusively to hormones. In addition, only three women in our study used oral contraceptives; statistical analyses excluding these data did not alter the results. Thus, the gender differences cannot be attributed to oral contraceptive use.¹¹⁴ Overall, since our women were healthy and normally cycling, their sleep likely was not affected by menstrual phase.^{113,115,116}

Effects of Lavender Odor on Subjective Sleepiness

Lavender did not affect subjective sleepiness before bedtime, since Stanford Sleepiness Scale and Karolinska Sleepiness Scale scores assessed immediately after the stimulus or control exposure did not differ. Other mild sedatives such as melatonin also have produced changes in objective sleep without changes in Stanford Sleepiness Scale scores.¹¹⁷⁻¹¹⁹ These results may seem inconsistent with previous findings indicating immediate physiological changes with lavender exposure, including electroencephalographic changes.^{19,22} Such discrepant results may reflect the inaccuracy of subjects' self-rated evaluations or the possibility that physiological changes after lavender exposure can occur without subjects' awareness. Indeed, physiological

responses to odors, occurring independent of subject detection, have been reported in previous electroencephalographic studies when odors were below detection thresholds.²⁰ Similar to our study, Warm et al¹²⁰ found that muguet and peppermint did not alter Stanford Sleepiness Scale scores.

Subjective sleepiness ratings the following morning also did not differ between the two sessions. While this result is not surprising considering that lavender's sleep-promoting, sedative effects were found only in the early portion of the night, an overall restful sleep should influence subjects' sleepiness the following morning. Our study assessed sleepiness immediately upon awakening (within 1-2 minutes), a time when lingering sleepiness could be due to sleep inertia.¹²¹ As such, the study methodology may have made it difficult to detect overall differences in sleepiness in the morning, explaining why objective sleep differences on the first night were not reflected in the subjective sleepiness scales. Thus, assessing subjects' sleepiness later after awakening (i.e., 15 minutes) might be a more accurate morning indicator of this measure. In addition, other ratings of subjective sleep, such as estimations of sleep onset latency, number of awakenings, total time asleep and awake, and overall sleep quality may be more closely related to objective measures.

Karolinska Sleepiness Scale and Stanford Sleepiness Scale scores showed strong positive correlations with each other in the morning and evening in each session. In addition, slow-wave sleep % sleep period time was the only variable correlated with evening sleepiness scores solely in the stimulus session: greater sleepiness related to more slow-wave sleep. In the control sessions, morning sleepiness scores positively related to sleep onset latency, while in both sessions they positively related to stage 2 and stage 3 latency, indicating that the longer it took to reach these stages, the more sleepy subjects felt upon awakening. In the control session, evening

sleepiness scores negatively related to wake after sleep onset variables, indicating that greater sleepiness related to less disruptive sleep. Finally, in the control session, morning sleepiness scores negatively related to stage 2 and non-rapid-eye movement duration, and total sleep time and sleep period time, indicating that less sleepiness related to more sleep. Similarly, Akerstedt et al¹²² also reported that Karolinska Sleepiness Scale ratings in the morning strongly correlated with objective sleep efficiency.

Effects of Lavender Odor on Subjective Mood

Vigor scores were higher in the morning following lavender exposure compared to the control, a finding that corroborates the increase in restful deep sleep in the lavender session. Since no Profile of Mood States changes were seen immediately following exposure (at 2342h), lavender's effects on sleep are likely physiological and not psychological (i.e., not promoted by lavender's hedonics or intensity).

An absence of lavender's immediate effects on mood contrasts another study from our group that found that lavender oil increased depression, fatigue, anxiety, anger, confusion, and total mood disturbance, and decreased vigor on the Profile of Mood States compared to distilled water.²⁴ Our results also contrast with studies that found that lavender decreased tension/anxiety,^{25,123-126} improved mood^{88,125} and reduced total mood disturbance¹⁹ and stress.²⁶ Among other factors, differences across studies may be due to administration time or exposure duration.

In our study, vigor, confusion, fatigue and depression were higher in evening than in the morning, whereas total mood disturbance scores were higher in the morning. By contrast, tension and anger showed no changes across time or between sessions. Dollins et al¹¹⁹ found

that vigor decreased from 1900h to 0700h, but all other scales increased, whereas Hill and Hill¹²⁷ found that total mood disturbance, tension and depression scales were greater in the morning than afternoon. Higher evening confusion scores were expected and corroborate another study from our laboratory (Goel, unpublished), but differ from Florida-James et al.'s results.¹²⁸ Higher morning total mood disturbance scores, however, opposes data from another study from our laboratory (Goel, unpublished), but is in agreement with data from Hill and Hill¹²⁷ and Florida-James et al.¹²⁸ Vigor was higher at 2300h than at time points closer to bedtime or in the morning. This result differs from other studies that found higher vigor in the morning^{129,130} but confirms other findings.^{119,131} The low vigor scores in the morning may be due to a sleep inertia effect.

Depression was higher in the evening than morning, similar to some studies,^{129,132} but in opposition to other studies which found reverse diurnal variation.^{133,134} The lack of changes across time in tension and anger, both components of negative affect, are consistent with some studies^{130,135-137} but contrast others.^{119,127,138-140}

Relationship of Profile of Mood States to Polysomnographic and Sleepiness Measures

Very few sleep measures correlated with Profile of Mood States scores in the stimulus session. Correlations were found mostly for the fatigue scale, in the predicted directions: higher fatigue related to variables indicating better sleep, such as more sleep period time, shorter sleep onset latency and less wake after sleep onset. By contrast, the control session sleep measures showed many correlations with the vigor, anger, tension and confusion scales and only one correlation with fatigue, thus showing a much different pattern than the stimulus session. The only overlap between sessions was for fatigue and stage 1 latency: in the stimulus session, fatigue was negatively correlated with latency while in the control session it showed the opposite

relationship. These variant results suggest differential effects of the stimulus versus the control on evening fatigue scores and their relationship to sleep. Palinkas et al¹⁴¹ found that Profile of Mood States scores inversely predicted various sleep measures, including total sleep time, longest sleep event, sleep onset, sleep quality and number of sleep events, though another study did not find correlations between Profile of Mood States measures and total sleep time.¹⁴²

Profile of Mood States scores also related to sleepiness scores. Fatigue and vigor scores showed significant positive and negative correlations, respectively with Karolinska Sleepiness Scale and Stanford Sleepiness Scale scores. Higher vigor and less fatigue both significantly related to decreased sleepiness in the morning following the control and stimulus sessions, and in the evening of the stimulus session. However, these relationships were not seen in the evening of the control session. In addition, vigor in the morning following the stimulus session negatively related to evening stimulus Karolinska Sleepiness Scale and Stanford Sleepiness Scale scores. These relationships demonstrate consistency of self-rated measures relating to energy level.

Comparison to Other Stimuli

Our findings are consistent with previous experimental studies suggesting that sensory stimulation or salient behavioral experiences occurring during wakefulness affect subsequent sleep by modulating neural systems which regulate normal sleep/wake cycles.¹⁴³⁻¹⁴⁶ Daytime sensory experiences are thought to affect the recovery process during the subsequent night's sleep, supporting the homeostatic function of sleep--the view that sleep is a recovery period after an increase in physiological strain during wakefulness.¹⁴⁷

In support of this theory, various sensory stimuli or behavioral events, experienced before bedtime modify subsequent sleep by increasing slow-wave sleep. Slow-wave sleep increases

following exposure to auditory^{143,148,149} and visual stimuli.¹⁵⁰ Similarly, exercise consistently increases nighttime slow-wave sleep¹⁵¹⁻¹⁵⁵ as does body heating.¹⁵⁶⁻¹⁵⁹ Thus, olfactory cues may share a common mechanism with other sensory stimuli for modulating the release of specific sleep-inducing substances which promote deep sleep.¹⁴⁴

Neural Pathways for Odor Transduction

At present, the neuroanatomical pathways involved in mediating lavender's effects on sleep are unknown. A variety of different odors, including lavender, activate the primary olfactory cortex and its neural connections, including the amygdala, piriform cortex, entorhinal cortex, insular cortex and claustrum, the anterior cingulate and the orbitofrontal cortex.^{49-51,89,93,94,160-162} These olfactory targets may subsequently transduce information to the various brain centers implicated in the control of sleep and wake. Alternatively, lavender may exert its effects systemically through the blood after entry into the nasal passages.

Summary

This is the first study to examine the effects of an olfactory stimulus presented before sleep on objective measures of subsequent sleep. Previous studies were anecdotal, using uncontrolled conditions, small sample sizes and subjective sleep measurements. We found that lavender odor promotes deep sleep when presented before bedtime in healthy young men and women, thus substantiating its commonly claimed psychological and physiological sedative properties. In addition, lavender showed differential gender effects for wake after sleep onset latency, stage 2 and rapid-eye movement sleep: lavender increased stage 2 and decreased rapid-eye movement sleep and wake after sleep onset latency in women, but produced the opposite

effects in men. This study also revealed robust gender differences in sleep measures. Young women showed better sleep quality and efficiency overall: they fell asleep faster (shorter sleep onset latency), spent more time asleep and spent less time awake.

Our results yield important practical applications. Commercially available lavender oil may be used as a soporific agent, and therefore, a possible nonphotic alternative to other substances such as valerian, melatonin or benzodiazepines for relieving sleep disturbance. Lavender may be beneficial over these substances, since it does not produce “hangover” effects the next morning, as assessed by self-rated sleepiness scores. Moreover, lavender would likely produce larger changes in deep sleep and significant effects on other sleep measures in subjects who have difficulty sleeping, such as habitually poor or irregular sleepers, including the elderly. Other oils that are also natural sedatives, such as camomile may exert similar effects on sleep. In contrast, stimulating odors, such as peppermint may disrupt sleep. Aromatherapy, therefore, has promise for modifying sleep in various populations, including insomniacs, depressed patients, critically ill or hospitalized patients and the elderly. Therefore, it should be tested rigorously and systematically in these populations.

Acknowledgments

This research was supported by a grant from the Sense of Smell Institute. I am grateful to Dr. Albert Fry for his invaluable assistance in the gas chromatography analysis of the lavender oil.

FIGURE LEGENDS

Figure 1. Protocol timeline for the three consecutive night study.

Figure 2. SWS (stages 3+4) %SPT across the whole night for both sessions (mean \pm SD). * = significantly greater than the control night, $P < .05$.

Figure 3. (A) WASO latency for the whole control and experimental nights for men and women; (B) Stage 2 %SPT and (C) REM %SPT for men and women in the first half of the control and experimental nights (mean \pm SD). Each of these PSG variables showed significant session \times gender interactions. * = significantly different between the control and stimulus nights for men, $P < .05$.

Figure 4. Gender differences in sleep across all three nights (mean \pm SD): (A) total sleep time (TST, min); (B) sleep period time (SPT, %); (C) sleep efficiency (SE, %); (D) total wake time (TWT, min); (E) sleep onset latency (SOL, min). * = significantly different between men and women, $P < .05$.

Figure 5. Night and morning (A) SSS and (B) KSS scores for all three sessions (mean \pm SD). Subjective sleepiness ratings on both scales at night or the following morning did not differ among the three sessions. However, there were positive, strong correlations between KSS and SSS scores in the morning and evening for each corresponding session.

Figure 6. (A) Vigor POMS scores for all subjects in the stimulus and control sessions across all time points (mean \pm SD). *= significantly greater than the control session, $P < 0.05$; a=significantly greater than all other time points for the stimulus and control sessions, $P < 0.05$ - $P < 0.001$; b=significantly greater than 2342h for the stimulus session, $P < 0.001$; c=significantly greater than 2342h or 0800h for the control session, $P < 0.01$ - $P=0.001$. **(B)** TMD POMS scores for men and women pooled across the stimulus and control sessions for all time points (mean \pm SD). a=significantly less than 2342h and 0800h for men only, $P < 0.05$; b=significantly less than 2312h and 2342h for women only, $P < 0.01$ - $P < 0.001$; c=significantly less than 2342h and 0800h for men only, $P < 0.05$ - $P=0.001$; d=significantly less than 2342h for women only, $P=0.001$.

Figure 7. (A) Depression; **(B)** Fatigue; **(C)** Confusion POMS scores for all subjects pooled across the stimulus and control sessions for all time points (mean \pm SD). a=significantly different from all other time points, $P < 0.05$ - $P < 0.001$; b=significantly less than 2342h, $P < 0.05$.

REFERENCES

1. Buckle J. The role of aromatherapy in nursing care. *Nurs Clin North Am* 2001; 36:57-72.
2. Price S, Price L. *Aromatherapy for health professionals*. 2nd ed. London: Churchill Livingstone; 1999.
3. Tisserand R. Essential oils as psychotherapeutic agents. In: Van Toller S, Dodd GH, editors. *Perfumery: The psychology and biology of fragrance*. New York: Chapman Hall; 1988. p.167-181.
4. Rovesti P, Colombo E. Aromatherapy and Aerosols. *Soap, Perfumery Cosmetics* 1973; 46:475-478.
5. Wood K. The promise of aromatherapy. *Provider* 2003; 29:47-48.
6. Badia P, Wesensten N, Lammers W, Culpepper J, Harsh J. Responsiveness to olfactory stimuli presented in sleep. *Physiol Behav* 1990; 48:87-90.
7. Carskadon MA, Herz RS. Olfactory arousal threshold in stage 2, stage 4, and REM sleep in comparison to an auditory signal. *Sleep Res* 2003; 26:A445.
8. Hudson R. The value of lavender for rest and activity in the elderly patient. *Complement Ther Med* 1996; 4:52-57.
9. Hardy M. Sweet scented dreams. *Int J Aromatherapy* 1991; 3:12-13.
10. Henry J, Rusius CW, Davies M, Veazey-French T. Lavender for night sedation of people with dementia. *Int J Aromatherapy* 1994; 6:28-30.
11. Wolfe N, Herzberg J. Can aromatherapy oils promote sleep in severely demented patients? *Int J Geriatr Psychiatry* 1996; 11:926-927.
12. Connell FEA, Tan G, Gupta I, Gompertz P, Bennett GCJ, Herzberg JL. Can aromatherapy promote sleep in elderly hospitalized patients? *Geriatr Today: J Can Geriatr Soc* 2001; 4:191-195.
13. Miyake Y, Nakagawa M, Asakura Y. Effects of odors on humans (I). Effects on sleep latency. *Chem Senses* 1991; 16:183.
14. Svoboda KP, Karavia AN, McFarlane V. Case study: the effects of selected essential oils on mood, concentration and sleep in a group of 10 students monitored for 5 weeks. *Int J Aromatherapy* 2002; 12:157-161.

15. Sano A, Sei H, Seno H, Morita Y, Moritoki H. Influence of cedar essence on spontaneous activity and sleep of rats and human daytime nap. *Psychiatry Clin Neurosci* 1998; 52: 133-135.
16. Romine IJ, Bush AM, Geist CR. Lavender aromatherapy in recovery from exercise. *Percept Mot Skills* 1999; 88:756-758.
17. Nagai M, Wada M, Usui N, Tanaka A, Hasebe Y. Pleasant odors attenuate the blood pressure increase during rhythmic handgrip in humans. *Neurosci Lett* 2000; 289:227-229.
18. Torii S, Fukuda H, Kanemoto H, Miyanchi R, Hamauzu Y, Kawasaki M. Contingent negative variation and the psychological effects of odour. In: Van Toller S, Dodd G, editors. *Perfumery: The psychology and biology of fragrance*. New York: Chapman Hall; 1988. p.107-120.
19. Diego MA, Jones NA, Field T, et al. Aromatherapy positively affects mood, EEG patterns of alertness and math computations. *Int J Neurosci* 1998; 96:217-224.
20. Lorig TS, Herman KB, Schwartz GE, Cain WS. EEG activity during administration of low-concentration odors. *Bull Psychonomic Soc* 1990; 28:405-408.
21. Masago R, Matsuda T, Kikuchi Y, et al. Effects of inhalation of essential oils on EEG activity and sensory evaluation. *J Physiol Anthropol* 2000; 19:35-42.
22. Klemm WR, Lutes SD, Hendrix DV, Warrenburg S. Topographical EEG maps of human responses to odors. *Chem Senses* 1992; 17:347-361.
23. Lorig TS, Schwartz GE. Brain and odor: I. Alteration of human EEG by odor administration. *Psychobiology* 1988; 16:281-284.
24. Goel N, Grasso DJ. Olfactory discrimination and transient mood change in young men and women: Variation by clinical state, season, chronotype and time of day. *Chronobiol Int* (under review).
25. Lorig TS, Schwartz GE. EEG activity during relaxation and food imagery. *Psychophysiology* 1987; 24:599.
26. Motomura N, Sakurai A, Yotsuya Y. Reduction of mental stress with lavender odorant. *Percept Mot Skills* 2001; 93:713-718.
27. Schwartz GE. Subjective and respiratory differentiation of fragrance: interactions with hedonics. *Psychophysiology* 1986; 23:460.
28. Karamat E, Ilmberger J, Roblhuber K, Rupp C. Excitatory and sedative effects of essential oils on human reaction time performance. *Chem Senses* 1992; 16:847.

29. Yagyu T. Neurophysiological findings on the effects of fragrance: lavender and jasmine. *Integrative Psychiatry* 1994; 10:62-67.
30. Millot JL, Brand G, Morand N. Effects of ambient odors on reaction time in humans. *Neurosci Lett* 2002; 322:79-82.
31. Ludvigson HW, Rottman TR. Effects of ambient odors of lavender and cloves on cognition, memory, affect and mood. *Chem Senses* 1989; 14:525-536.
32. Brand G, Millot JL. Sex differences in human olfaction: Between evidence and enigma. *Q J Exp Psychol B* 2001; 54:259-270.
33. Velle W. Sex differences in sensory functions. *Perspect Biol Med* 1987; 30:490-522.
34. Dalton P, Doolittle N, Breslin PAS. Gender-specific induction of enhanced sensitivity to odors. *Nat Neurosci* 2002; 5:199-200.
35. Nawab SS, Miller CS, Dale JK, et al. Self-reported sensitivity to chemical exposures in five clinical populations and healthy controls. *Psychiatry Res.* 95:67-74; 2000.
36. Deems DA, Doty RL. Age-related changes in the phenyl ethyl alcohol odor detection threshold. *Trans Pa Acad Ophthalmol Otolaryngol* 1987; 39:646-650.
37. Kobal G, Kettenmann B. Olfactory functional imaging and physiology. *Int J Psychophysiol* 2000; 36:157-163.
38. Hulshoff Pol HE, Hijman R, Baare WF, van Eekelen S, van Ree JM. Odor discrimination and task duration in young and older adults. *Chem Senses* 2000; 25:461-464.
39. Segal NL, Topolski TD, Wilson SM, Brown KW, Araki L. Twin analysis of odor identification and perception. *Physiol Behav* 1995; 57:605-609.
40. Ship JA, Weiffenbach JM. Age, gender, medical treatment, and medication effects on smell identification. *J Gerontol* 1993; 48:M26-M32.
41. Öberg C, Larsson M, Bäckman L. Differential sex effects in olfactory functioning: the role of verbal processing. *J Int Neuropsychol Soc* 2002; 8:691-698.
42. Wysocki CJ, Gilbert AN. National Geographic Smell Survey: Effects of age are heterogenous. *Ann NY Acad Sci* 1989; 561:12-28.
43. Cain WS. Odor identification by males and females: predictions vs. performance. *Chem Senses* 1982; 7:129-142.
44. Doty RL, Shaman P, Applebaum SL, Giberson R, Siksorski L, Rosenberg L. Smell identification ability: Changes with age. *Science* 1984; 226:1441-1443.

45. Doty RL, Applebaum S, Zusho H, Settle RG. Sex differences in odor identification ability: a cross-cultural analysis. *Neuropsychologia* 1985; 23:667-672.
46. Becker E, Hummel T, Piel E, Pauli E, Kobal G, Hautzinger M. Olfactory event-related potentials in psychosis-prone subjects. *Int J Psychophysiol* 1993; 15:51-58.
47. Evans WJ, Cui L, Starr A. Olfactory event-related potentials in normal human subjects: effects of age and gender. *Electroencephalogr Clin Neurophysiol* 1995; 95:293-301.
48. Yousem DM, Maldjian JA, Siddiqi F; et al. Gender effects on odor-stimulated functional magnetic resonance imaging. *Brain Res* 1999; 818:480-487.
49. Levy LM, Henkin RI, Lin CS, Hutter A, Schellinger D. Odor memory induces brain activation as measured by functional MRI. *J Comput Assist Tomogr* 1999; 23:487-498.
50. Bengtsson S, Berglund H, Gulyas B, Cohen E, Savic I. Brain activation during odor perception in males and females. *Neuroreport* 2001; 12:2027-2033.
51. Levy LM, Henkin RI, Hutter A, Lin CS, Martins D, Schellinger D. Functional MRI of human olfaction. *J Comput Assist Tomogr* 1997; 21:849-856.
52. Armitage R, Hoffmann R. Sleep EEG, depression and gender. *Sleep Med Rev* 2001; 5:237-246.
53. Reynolds CF III, Kupfer DJ, Taska LS, Hoch CC, Sewitch DE, Spiker DG. Sleep of healthy seniors: a revisit. *Sleep* 1985; 8:20-29.
54. Kobayashi, R., Kohsaka, M., Fukuda, N., Honma, H., Sakakibara, S., Koyama, T. Gender differences in the sleep of middle-aged individuals. *Psychiatry Clin Neurosci* 1998; 52:186-187.
55. Fukuda N, Honma H, Kohsaka M, et al. Gender difference of slow wave sleep in middle aged and elderly subjects. *Psychiatry Clin Neurosci* 1999; 53:151-153.
56. Hume KI, Van F, Watson A. A field study of age and gender differences in habitual adult sleep. *J Sleep Res* 1998; 7:85-94.
57. Wauquier A, van Sweden B, Lagaay AM, Kemp B, Kamphuisen HA. Ambulatory monitoring of sleep-wakefulness patterns in healthy elderly males and females (greater than 88 years): the "Senieur" protocol. *J Am Geriatr Soc* 1992; 40:109-114.
58. Rediehs MH, Reis JS, Creason NS. Sleep in old age: focus on gender differences. *Sleep* 1990;13:410-424.
59. Hoch CC, Reynolds CF III, Kupfer DJ, Berman SR. Stability of EEG sleep and sleep quality in healthy seniors. *Sleep* 1988; 11:521-527.

60. Williams RL, Karacan I, Hirsch CJ. *Electroencephalography (EEG) of human sleep: Clinical implications*. New York: John Wiley and Sons Ltd; 1974.
61. Voderholzer U, Al-Shajlawi A, Weske G, Feige B, Riemann D. Are there gender differences in objective and subjective sleep measures? A study of insomniacs and healthy controls. *Depress Anxiety* 2003; 17:162-172.
62. Antonijevic IA, Murck H, Frieboes R, Holsboer F, Steiger A. On the gender differences in sleep-endocrine regulation in young normal humans. *Neuroendocrinology* 1999; 70:280-287.
63. Elsenbruch S, Harnish MJ, Orr WC. Heart rate variability during waking and sleep in healthy males and females. *Sleep* 1999; 22:1067-1071.
64. Dijk DJ, Beersma DGM, Bloem GM. Sex differences in the sleep EEG of young adults: visual scoring and spectral analysis. *Sleep* 1989; 12:500-507.
65. Armitage R. The distribution of EEG frequencies in REM and NREM sleep stages in healthy young adults. *Sleep* 1995; 18:334-341.
66. Carrier J, Land S, Buysse DJ, Kupfer DJ, Monk TH. The effects of age and gender on sleep EEG power spectral density in the middle years of life (ages 20-60 years old). *Psychophysiology* 2001; 38:232-242.
67. Armitage R, Hoffmann R, Trivedi M, Rush AJ. Slow-wave activity in NREM sleep: sex and age effects in depressed outpatients and healthy controls. *Psychiatry Res* 2000b;95:201-213.
68. Ehlers CL, Kupfer DJ. Slow-wave sleep: do young adult men and women age differently? *J Sleep Res* 1997; 6:211-215.
69. Armitage R, Hoffmann R, Fitch T, Trivedi M, Rush AJ. Temporal characteristics of delta activity during NREM sleep in depressed outpatients and healthy adults: group and sex effects. *Sleep*. 2000a; 23:607-617.
70. Mourtazaev MS, Kemp B, Zwinderman AH, Kamphuisen HAC. Age and gender affect different characteristics of slow waves in the sleep EEG. *Sleep* 1995; 18:557-564.
71. Huupponen E, Himanen S, Varri A, Hasan J, Lehtokangas M, Saarinen J. A study on gender and age differences in sleep spindles. *Neuropsychobiology* 2002; 45:99-105.
72. Rechtschaffen A, Kales A. *A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects*. Washington, DC: National Institute of Health; 1968.

73. Hoddes E, Zarcone V, Smythe H, Phillips R, Dement WC. Quantification of Sleepiness: a new approach. *Psychophysiology* 1973; 10:431-436.
74. Akerstedt T, Gillberg M. Subjective and objective sleepiness in the active individual. *Int J Neurosci* 1990; 52:29-37.
75. Horne JA, Östberg O. A self-assessment questionnaire to determine morningness-eveningness in human circadian rhythms. *Int J Chronobiol* 1976; 4:97-110.
76. Kerkhof GA. Inter-individual differences in the human circadian system: a review. *Biol Psychol* 1985; 20:83-112.
77. Duffy JF, Dijk DJ, Hall EF, Czeisler CA. Relationship of endogenous circadian melatonin and temperature rhythms to self-reported preference for morning or evening activity in young and older people. *J Investig Med* 1999; 47:141-150.
78. Bailey SL, Heitkemper MM. Circadian rhythmicity of cortisol and body temperature: morningness-eveningness effects. *Chronobiol Int* 2001; 18:249-261.
79. McNair DM, Lorr M, Droppleman LF. *Profile of Mood States (Manual)*. San Diego: Educational and Industrial Testing Service; 1992.
80. Jacob S, Hayreh DJ, McClintock MK. Context-dependent effects of steroid chemosignals on human physiology and mood. *Physiol Behav* 2001; 74:15-27.
81. Jacob S, McClintock MK. Psychological state and mood effects of steroidal chemosignals in women and men. *Horm Behav* 2000; 37:57-78.
82. Schiffman SS, Sattely Miller EA, Suggs MS, Graham BG. The effect of environmental odors emanating from commercial swine operations on the mood of nearby residents. *Brain Res Bull* 1995a; 37:369-375.
83. Schiffman SS, Sattely Miller EA, Suggs MS, Graham BG. The effect of pleasant odors and hormone status on mood of women at midlife. *Brain Res Bull* 1995b; 36:19-29.
84. Schiffman SS, Suggs MS, Sattely-Miller EA. Effect of pleasant odors on mood of males at midlife: comparison of African-American and European-American men. *Brain Res Bull* 1995c; 36:31-37.
85. Jockovich M, Cosentino D, Cosentino L, Wears RL, Seaberg DC. Effect of exogenous melatonin on mood and sleep efficiency in emergency medicine residents working night shifts. *Acad Emerg Med* 2000; 7:955-958.
86. Dollins AB, Zhdanova IV, Wurtman RJ, Lynch HJ, Deng MH. Effect of inducing nocturnal serum melatonin concentrations in daytime on sleep, mood, body temperature, and performance. *Proc Natl Acad Sci* 1994; 91:1824-1828.

87. Wright SW, Lawrence LM, Wrenn KD, Haynes ML, Welch LW, Schlack HM. Randomized clinical trial of melatonin after night-shift work: efficacy and neuropsychologic effects. *Ann Emerg Med* 1998; 32:334-340.
88. Knasko SC. Ambient odor's effect on creativity, mood, and perceived health. *Chem Senses* 1992; 17:27-35.
89. Zatorre RJ, Jones-Gotman M, Evans AC, Meyer E. Functional localization and lateralization of human olfactory cortex. *Nature* 1992; 360:339-340.
90. Levick SE, Lorig T, Wexler BE, Gur RE, Gur RC, Schwartz GE. Asymmetrical visual deprivation: a technique to differentially influence lateral hemispheric function. *Percept Mot Skills* 1993; 76:1363-1382.
91. Millot JL, Brand G. Effects of pleasant and unpleasant ambient odors on human voice pitch. *Neurosci Lett* 2001; 297:61-63.
92. Savic I, Berglund H. Right-nostril dominance in discrimination of unfamiliar, but not familiar, odours. *Chem Senses* 2000; 25:517-523.
93. Savic I, Gulyas B. PET shows that odors are processed both ipsilaterally and contralaterally to the stimulated nostril. *Neuroreport* 2000; 11:2861-2866.
94. Royet JP, Zald D, Versace R, et al. Emotional responses to pleasant and unpleasant olfactory, visual, and auditory stimuli: a positron emission tomography study. *J Neurosci* 2000; 20:7752-7759.
95. Leathwood PD, Chauffard F. Quantifying the effects of mild sedatives. *J Psychiatr Res* 1982; 17:115-122.
96. Benca RM, Obermeyer WH, Thisted RA, Gillin JC. Sleep and psychiatric disorders. A meta-analysis. *Arch Gen Psychiatry* 1992; 49:651-668.
97. Waldman CS, Tseng P, Meulman, P, Whittet HB. Aromatherapy in the intensive care unit. *Care Critically Ill* 1993; 9:170-174.
98. Richards K, Nagel C, Markie M, Elwell J, Barone C. Use of complementary and alternative therapies to promote sleep in critically ill patients. *Crit Care Nurs Clin North Am* 2003; 15:329-340.
99. Cannard G. On the scent of a good night's sleep. *Nursing Standard* 1995; 9:21.
100. Carskadon MA, Dement WC. Normal human sleep: An overview. In: Kryger MH, Roth T, Dement WC, editors. *Principles and Practice of Sleep Medicine*. 3rd ed. New York: W.B. Saunders Company; 2000. p.15-25.

101. Agnew HW Jr, Webb WB, Williams RL. The first night effect: an EEG study of sleep. *Psychophysiology* 1966; 2:263-266.
102. Schmidt HS, Kaelbling R. The differential laboratory adaptation of sleep parameters. *Biol Psychiatry* 1971; 3:33-45.
103. Webb WB, Campbell SS. The first night effect revisited with age as a variable. *Waking Sleeping* 1979; 3:319-324.
104. Mendels J, Hawkins DR. Sleep laboratory adaptation in normal subjects and depressed patients ("first night effect"). *Electroencephalogr Clin Neurophysiol* 1967; 22:556-558.
105. Lorenzo JL, Barbanj MJ. Variability of sleep parameters across multiple laboratory sessions in healthy young subjects: the "very first night effect". *Psychophysiology* 2002; 39:409-413.
106. Toussaint M, Luthringer R, Schaltenbrand N, et al. First-night effect in normal subjects and psychiatric inpatients. *Sleep* 1995; 18:463-469.
107. Browman CP, Cartwright RD. The first-night effect on sleep and dreams. *Biol Psychiatry* 1980; 15:809-812.
108. Kader GA, Griffin PT. Reevaluation of the phenomena of the first night effect. *Sleep* 1983; 6:67-71.
109. Savic I, Berglund H, Gulyas B, Roland P. Smelling of odorous sex hormone-like compounds causes sex-differentiated hypothalamic activations in humans. *Neuron* 2001; 31:661-668.
110. Lee KA, Shaver JF, Giblin EC, Woods NF. Sleep patterns related to menstrual cycle phase and premenstrual affective symptoms. *Sleep* 1990; 13:403-409.
111. Montplaisir J, Lorrain J, Denesle R, Petit D. Sleep in menopause: differential effects of two forms of hormone replacement therapy. *Menopause* 2001; 8:10-16.
112. Manber R, Armitage R. Sex, steroids, and sleep: a review. *Sleep* 1999; 22:540-555.
113. Driver HS, Baker FC. Menstrual factors in sleep. *Sleep Med Rev* 1998; 2:213-229.
114. Burdick RS, Hoffmann R, Armitage R. Short note: oral contraceptives and sleep in depressed and healthy women. *Sleep* 2002; 25:347-349.
115. Baker FC, Waner JI, Vieira EF, Taylor SR, Driver HS, Mitchell D. Sleep and 24 hour body temperatures: a comparison in young men, naturally cycling women and women taking hormonal contraceptives. *J Physiol* 2001; 530:565-574.

116. Driver HS, Dijk DJ, Werth E, Biedermann K, Borbely AA. Sleep and the sleep electroencephalogram across the menstrual cycle in young healthy women. *J Clin Endocrinol Metab* 1996; 81:728-735.
117. Pires ML, Benedito-Silva AA, Pinto L, Souza L, Vismari L, Calil HM. Acute effects of low doses of melatonin on the sleep of young healthy subjects. *J Pineal Res* 2001; 31:326-332.
118. Zhdanova IV, Wurtman RJ, Lynch HJ, et al. Sleep-inducing effects of low doses of melatonin ingested in the evening. *Clin Pharmacol Ther* 1995; 57:552-558.
119. Dollins AB, Lynch HJ, Wurtman RJ, Deng MH, Lieberman HR. Effects of illumination on human nocturnal serum melatonin levels and performance. *Physiol Behav* 1993; 53:153-160.
120. Warm JS, Dember, WN, Parasuraman R. Effects of olfactory stimulation on performance and stress in a visual sustained attention task. *J Soc Cosmet Chem* 1991; 42:199-210.
121. Tassi P, Muzet A. Sleep inertia. *Sleep Med Rev* 2000; 4:341-353.
122. Akerstedt T, Hume K, Minors D, Waterhouse J. The meaning of good sleep: a longitudinal study of polysomnography and subjective sleep quality. *J Sleep Res* 1994; 3:152-158.
123. Louis M, Kowalski SD. Use of aromatherapy with hospice patients to decrease pain, anxiety, and depression and to promote an increased sense of well-being. *Am J Hosp Palliat Care* 2002; 19:381-386.
124. Itai T, Amayasu H, Kuribayashi M, et al. Psychological effects of aromatherapy on chronic hemodialysis patients. *Psychiatry Clin Neurosci* 2000; 54:393-397.
125. Dunn C, Sleep J, Collett D. Sensing an improvement: an experimental study to evaluate the use of aromatherapy, massage and periods of rest in an intensive care unit. *J Adv Nurs* 1995; 21:34-40.
126. Kawai T, Noro K. Psychological effect of stereoscopic 3-D images with fragrances. *Ergonomics* 1996; 39:1364-1369.
127. Hill CM, Hill DW. Influence of time of day on responses to the profile of mood states. *Percept Mot Skills* 1991; 72:434.
128. Florida-James G, Wallymahmed A, Reilly T. Effects of nocturnal shiftwork on mood states of student nurses. *Chronobiol Int* 1996; 13:59-69.
129. Adan A, Guardia J. Circadian variations of self-reported activation: a multidimensional approach. *Chronobiologia* 1993; 20:233-244.

130. Wood C, Magnello ME. Diurnal changes in perceptions of energy and mood. *J R Soc Med* 1992; 85:191-194.
131. Brendel DH, Reynolds CF III, Jennings JR, et al. Sleep stage physiology, mood, and vigilance responses to total sleep deprivation in healthy 80-year-olds and 20-year-olds. *Psychophysiology* 1990; 27:677-685.
132. Robbins PR, Tanck RH. A study of diurnal patterns of depressed mood. *Motiv Emot* 1987; 11:37-49.
133. Monk TH, Fookson JE, Moline ML, Pollak CP. Diurnal variation in mood and performance in a time-isolated environment. *Chronobiol Int* 1985; 2:185-193.
134. Boivin DB, Czeisler CA, Dijk DJ, et al. Complex interaction of the sleep-wake cycle and circadian phase modulates mood in healthy subjects. *Arch Gen Psychiatry* 1997; 54:145-152.
135. Taub JM, Berger RJ. Diurnal variations in mood as asserted by self-report and verbal content analysis. *J Psychiat Res* 1974; 10:83-88.
136. Monk TH, Leng VC, Folkard S, Weitzman ED. Circadian rhythms in subjective alertness and core body temperature. *Chronobiologia* 1983; 10:49-55.
137. Bohle P, Tilley AJ. Predicting mood change on night shift. *Ergonomics* 1993; 36:125-33.
138. de Castro JM. Circadian rhythms of the spontaneous meal pattern, macronutrient intake, and mood of humans. *Physiol Behav* 1987; 40:437-446.
139. Thayer RE. Problem perception, optimism, and related states as a function of time of day (diurnal rhythm) and moderate exercise: Two arousal systems in interaction. *Motiv Emot* 1987; 11:19-36.
140. Adan A, Sanchez-Turet M. Gender differences in diurnal variations of subjective activation and mood. *Chronobiol Int* 2001; 18:491-502.
141. Palinkas LA, Houseal M, Miller C. Sleep and mood during a winter in Antarctica. *Int J Circumpolar Health* 2000; 59:63-73.
142. Bell IR, Bootzin RR, Ritenbaugh C, et al. A polysomnographic study of sleep disturbance in community elderly with self-reported environmental chemical odor intolerance. *Biol Psychiatry* 1996;40:123-133.
143. Cantero JL, Atienza M, Salas RM. Effects of waking-auditory stimulation on human sleep architecture. *Behav Brain Res* 2002; 128:53-59.

144. Garcia-Garcia F, Beltran-Parrazal L, Jimenez-Anguiano A, Vega-Gonzalez A, Drucker-Colin R. Manipulations during forced wakefulness have differential impact on sleep architecture, EEG power spectrum, and Fos induction. *Brain Res Bull* 1998; 47:317-324.
145. Velluti RA. Interactions between sleep and sensory physiology. *J Sleep Res* 1997; 6:61-77.
146. Drucker-Colin R. The function of sleep is to regulate brain excitability in order to satisfy the requirements imposed by waking. *Behav Brain Res* 1995; 69:117-124.
147. Borbely AA. Circadian and sleep-dependent processes in sleep regulation. In: Aschoff J, Daan S, Groos G, editors. *Vertebrate Circadian Systems*. Berlin: Springer-Verlag; 1982. p. 237-242.
148. Fruhstorfer B, Fruhstorfer H, Grass P. Daytime noise and subsequent night sleep in man. *Eur J Appl Physiol Occup Physiol* 1984; 53:159-163.
149. Fruhstorfer B, Pritsch MG, Fruhstorfer H. Effects of daytime noise load on the sleep-wake cycle and endocrine patterns in man: I. 24 hours neurophysiological data. *Int J Neurosci* 1988; 39:197-209.
150. Horne JA, Walmsley B. Daytime visual load and the effects upon human sleep. *Psychophysiology* 1976; 13:115-120.
151. Youngstedt SD, O'Connor PJ, Crabbe JB, Dishman RK. The influence of acute exercise on sleep following high caffeine intake. *Physiol Behav* 2000; 68:563-570.
152. Bunnell DE, Bevier W, Horvath SM. Effects of exhaustive exercise on the sleep of men and women. *Psychophysiology* 1983; 20:50-58.
153. Horne JA, Staff LH. Exercise and sleep: body-heating effects. *Sleep* 1983; 6:36-46.
154. Horne JA, Moore VJ. Sleep EEG effects of exercise with and without additional body cooling. *Electroencephalogr Clin Neurophysiol* 1985; 60:33-38.
155. Youngstedt SD, O'Connor PJ, Dishman RK. The effects of acute exercise on sleep: a quantitative synthesis. *Sleep* 1997; 20:203-214.
156. Horne JA, Reid AJ. Night-time sleep EEG changes following body heating in a warm bath. *Electroencephalogr Clin Neurophysiol* 1985; 60:154-157.
157. Sung EJ, Tochihara Y. Effects of Bathing and Hot Footbath on Sleep in Winter. *J Physiol Anthropol* 2000; 19:21-27.
158. Bunnell DE, Agnew JA, Horvath SM, Jopson L, Wills M. Passive body heating and sleep: influence of proximity to sleep. *Sleep* 1988; 11:210-219.

159. Dorsey CM, Teicher MH, Cohen-Zion M, et al. Core body temperature and sleep of older female insomniacs before and after passive body heating. *Sleep* 1999; 22:891-898.
160. Savic I, Gulyas B, Larsson M, Roland P. Olfactory functions are mediated by parallel and hierarchical processing. *Neuron* 2000; 26:735-745.
161. Savic I, Gulyas B, Berglund H. Odorant differentiated pattern of cerebral activation: comparison of acetone and vanillin. *Human Brain Mapping* 2002; 17:17-27.
162. Savic I. Brain imaging studies of the functional organization of human olfaction. *Neuroscientist* 2002; 8:204-211.

Table 1. Mean \pm SD whole night sleep parameters for the adaptation, stimulus and control nights.

PSG MEASURE	ADAPTATION NIGHT	STIMULUS NIGHT	CONTROL NIGHT
TST ^{a,c}	440.53 \pm 36.18	460.58 \pm 23.66	457.02 \pm 24.59
SPT ^c	455.18 \pm 29.00	465.98 \pm 17.84	462.71 \pm 20.62
TWT ^{b,c}	36.55 \pm 33.52	18.23 \pm 23.85	21.60 \pm 23.66
SE ^{a,c}	92.32 \pm 7.05	96.13 \pm 4.96	95.48 \pm 4.98
SME ^a	96.70 \pm 3.39	98.82 \pm 2.72	98.75 \pm 2.23
SOL ^c	21.07 \pm 24.52	12.44 \pm 18.21	16.03 \pm 19.59
WASO, min ^b	14.63 \pm 14.15	5.58 \pm 12.38	5.55 \pm 10.07
WASO, %SPT ^b	3.30 \pm 3.39	1.22 \pm 2.71	1.22 \pm 2.24
WASO, latency* ^a	98.47 \pm 101.23	191.61 \pm 125.44	223.57 \pm 129.20
Stage 1, min ^b	25.83 \pm 18.42	10.34 \pm 10.01	13.66 \pm 14.03
Stage 1, %SPT ^b	5.73 \pm 4.12	2.21 \pm 2.13	2.99 \pm 3.16
Stage 1, latency ^c	21.07 \pm 24.52	12.37 \pm 18.24	27.05 \pm 60.65
Stage 2, min* ^a	304.27 \pm 38.61	308.40 \pm 29.05	307.10 \pm 34.83
Stage 2, %SPT* ^a	66.83 \pm 7.08	66.22 \pm 5.99	66.34 \pm 6.80
Stage 2, latency ^c	26.55 \pm 24.31	16.92 \pm 19.50	19.82 \pm 20.42
Stage 3, min ^a	15.97 \pm 12.15	25.42 \pm 10.47	21.31 \pm 12.63
Stage 3, %SPT ^a	3.51 \pm 2.70	5.44 \pm 2.20	4.57 \pm 2.67
Stage 3, latency ^c	59.60 \pm 36.16	56.08 \pm 43.02	62.74 \pm 41.56
Stage 4, min ^a	3.47 \pm 7.57	7.05 \pm 12.62	5.39 \pm 10.46
Stage 4, %SPT ^a	.75 \pm 1.66	1.50 \pm 2.70	1.14 \pm 2.22
Stage 4, latency	58.54 \pm 37.22	50.70 \pm 21.83	67.25 \pm 44.40
Stages 3+4, min ^a	19.43 \pm 18.55	32.47 \pm 19.43	26.69 \pm 20.04
Stages 3+4, %SPT ^a	4.27 \pm 4.08	6.94 \pm 4.11	5.72 \pm 4.24
Stages 3+4, latency ^a	36.75 \pm 17.05	50.35 \pm 25.79	57.21 \pm 35.90
NREM, min	338.60 \pm 72.24	351.21 \pm 28.86	347.45 \pm 31.55
NREM, %SPT	76.57 \pm 6.32	75.36 \pm 5.48	75.06 \pm 5.62
REM, min* ^a	87.68 \pm 33.64	109.19 \pm 25.40	109.71 \pm 23.65
REM, %SPT* ^a	19.89 \pm 6.38	23.41 \pm 5.36	23.71 \pm 5.10
REM, latency	124.62 \pm 76.86	108.29 \pm 49.63	112.37 \pm 61.39

*Significant session night \times gender interaction for the stimulus and control nights ($P < .05$). See text for details.

^aSignificant session night effect across all nights ($P < .05$). Adaptation night significantly less than the stimulus and control nights (all P 's $< .05$).

^bSignificant session night effect across all nights ($P < .05$). Adaptation night significantly greater than the stimulus and control nights (all P 's $< .05$).

^cSignificant gender effect across all nights ($P < .05$). See text for details.

Table 2. Pearson's correlation coefficients (r) relating morning and evening SSS and KSS scores across the three sessions.

SLEEPINESS SCALE	SSS ADAPTATION NIGHT		SSS STIMULUS NIGHT		SSS CONTROL NIGHT	
	Morning	Evening	Morning	Evening	Morning	Evening
KSS						
Adaptation Evening	-.37	.83**	-.18	.27	-.12	.37
Control Evening	-.31	.38*	-.14	-.01	-.07	.82**
Stimulus Evening	-.10	.32	.02	.79**	-.08	.001
Adaptation Morning	.57**	-.34	-.02	.10	.07	-.49**
Control Morning	.32	-.07	.25	-.19	.83**	-.27
Stimulus Morning	-.03	-.27	.85**	-.05	.42*	-.38*

*P < .05.

**P < .01.

Table 3. Pearson's significant correlation coefficients (*r*) relating morning and evening POMS scores and PSG variables in the control and stimulus sessions.

PSG MEASURE	POMS CONTROL		POMS STIMULUS	
	Morning	Evening	Morning	Evening
TST	-.45*C, -.57**A	-.46**T, -.43*V, .38*C		
SPT	-.61**A	-.37*V, -.45*T, -.38*A		.38*F
TWT	.44*C, .57**A	.44*T, .46*V, .36*A, -.40*C		
SE	-.44*C, -.57**A	-.44*T, -.46*V, -.36*A, .40*C		
SME	-.38*C			
SOL	.60**A	.43*T, .40*V, .41*A, -.37*C		-.36*F
WASO, min	.38*C			
WASO, latency	.38*C	-.43*V		-.57*F, -.59*C
Stage 1, min	.39*C			.49**F
Stage 1, %SPT	.40*C			.48**F
Stage 1, latency	.41*T, .41*D	.36*F		-.36*F
Stage 2, min	-.51**C	-.42*V		
Stage 2, %SPT	-.43*C			
Stage 2, latency	.60*A	.43*T, .41*V, .38*A		
Stage 3, min		-.37*V		
Stage 3, latency	.58*A	.40*T, .45*V		
Stage 4, latency	.73**A			
Stages 3+4, latency	.59**A		-.37*V	
NREM, min	-.38*F, -.48**C	-.47*V		
NREM, %SPT	-.41*C	-.37*V, .41*A		
REM, min				.38*C
REM, %SPT		-.42*T, -.47**A		
REM, latency		.41*A		-.36*T

*P < .05; **P < .01.

C, Confusion; A, Anger; T, Tension; D, Depression; F, Fatigue; V, Vigor.

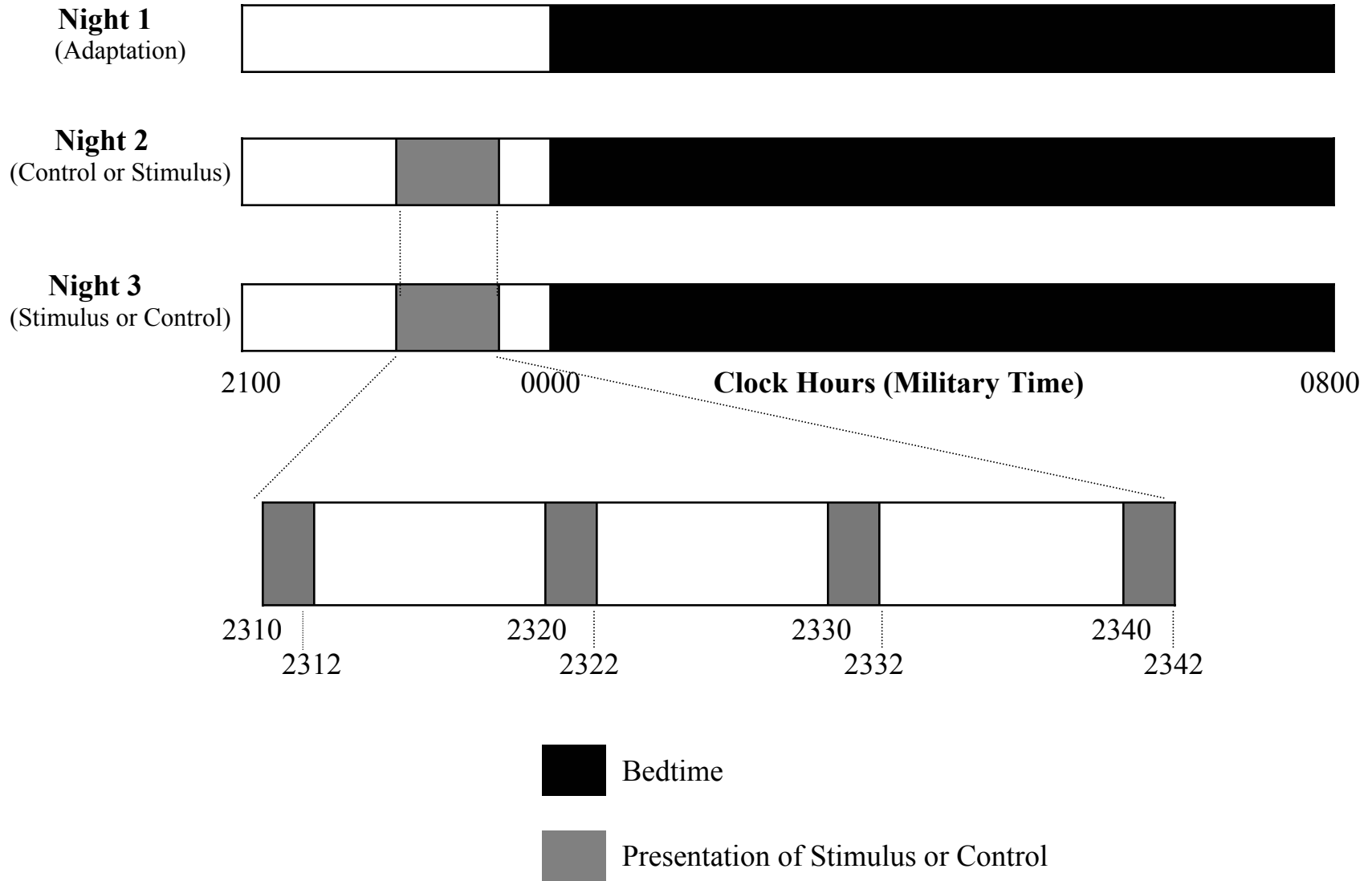
Table 4. Pearson's significant correlation coefficients (*r*) relating morning and evening SSS and KSS scores and PSG measures in the control and stimulus (S) sessions.

PSG MEASURE	KSS		SSS	
	Morning	Evening	Morning	Evening
TST			-.40*	
SPT			-.52**	
SME				.42*
SOL	.38*		.47**	
WASO, min				-.43*
WASO, %SPT				-.43*
Stage 2, min	-.39*			
Stage 2, latency	.41*		.48**, .40*(S)	
Stage 3, min		.39*		
Stage 3, %SPT		.38*		
Stage 3, latency	.43*, .37*(S)		.46*, .43*(S)	
Stages 3+4, %SPT				.38*(S)
NREM, min	-.47**			

*P < .05.

**P < .01.

Figure 1



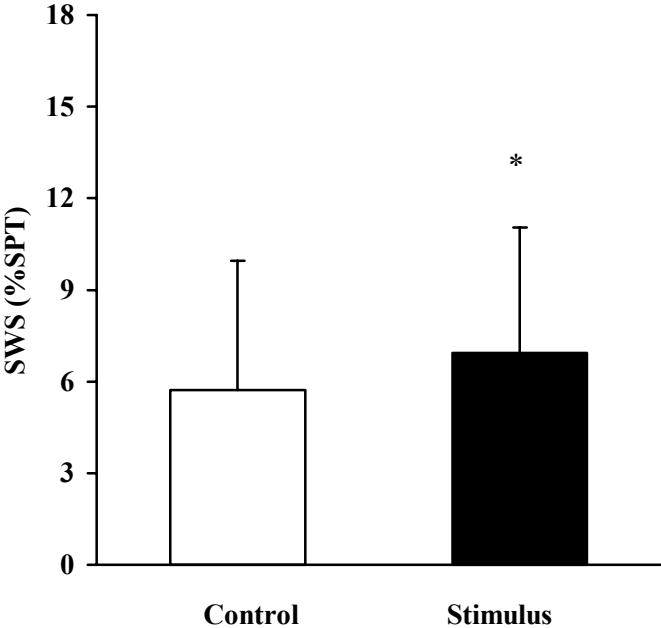


Figure 2

Figure 3

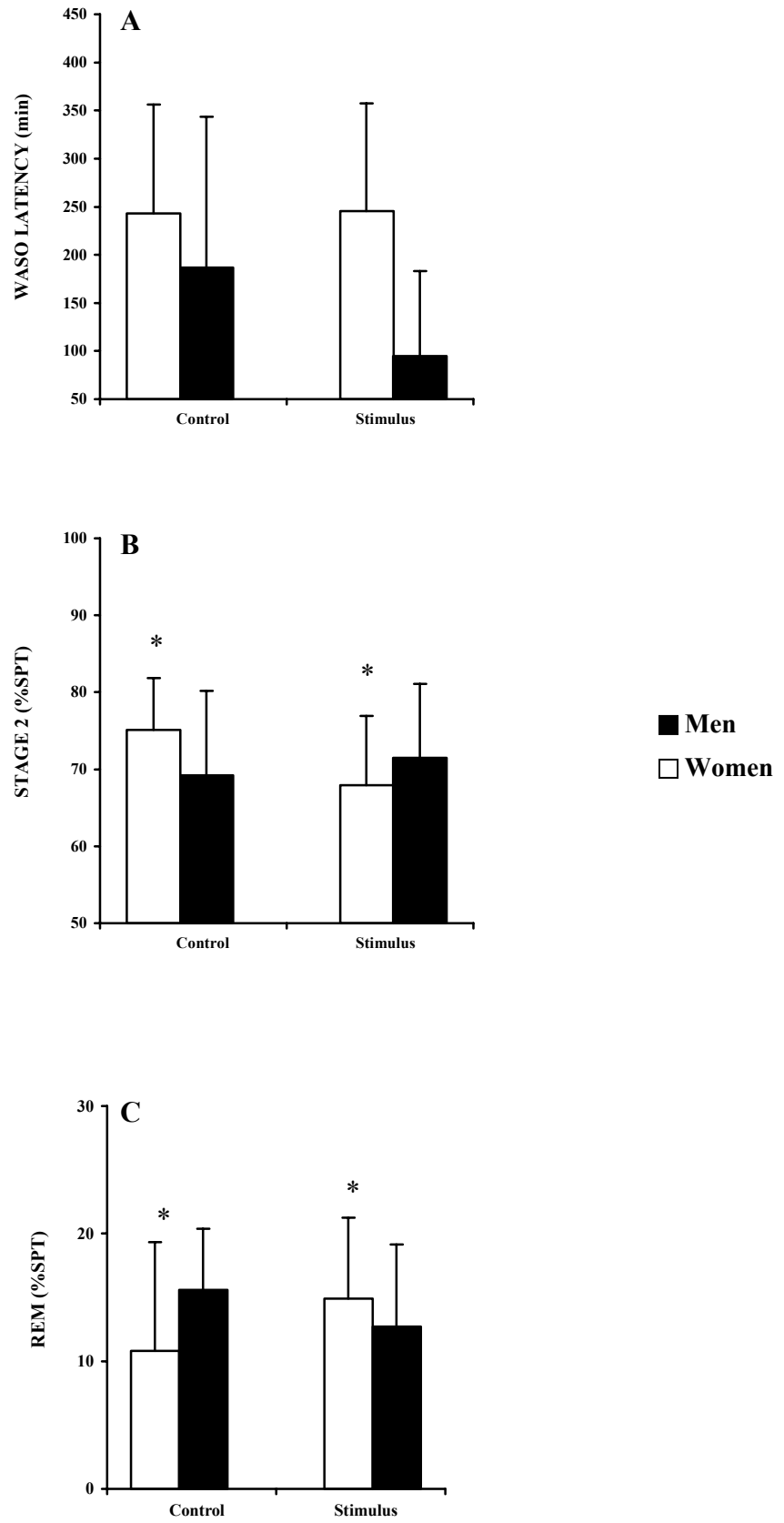


Figure 4

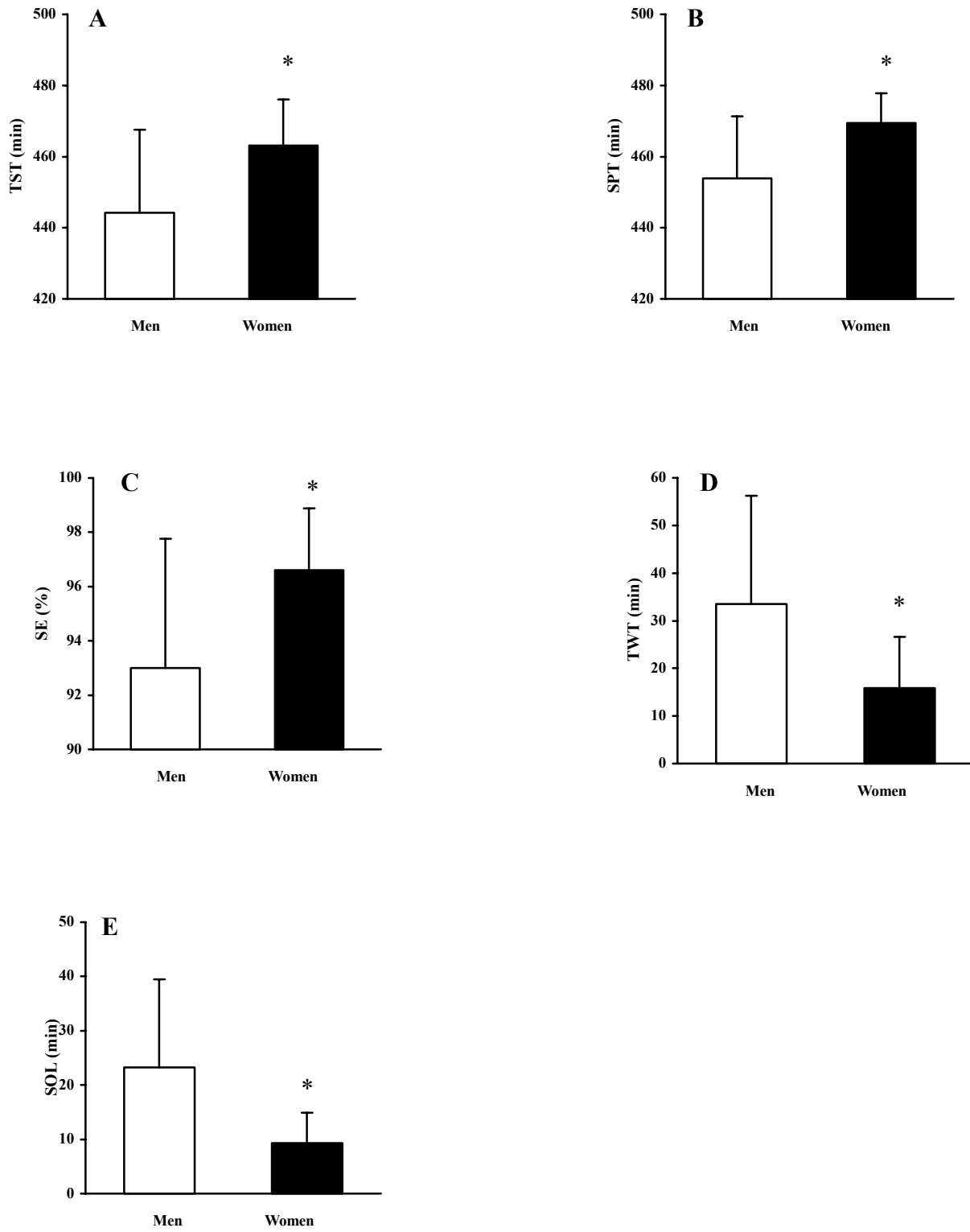


Figure 5

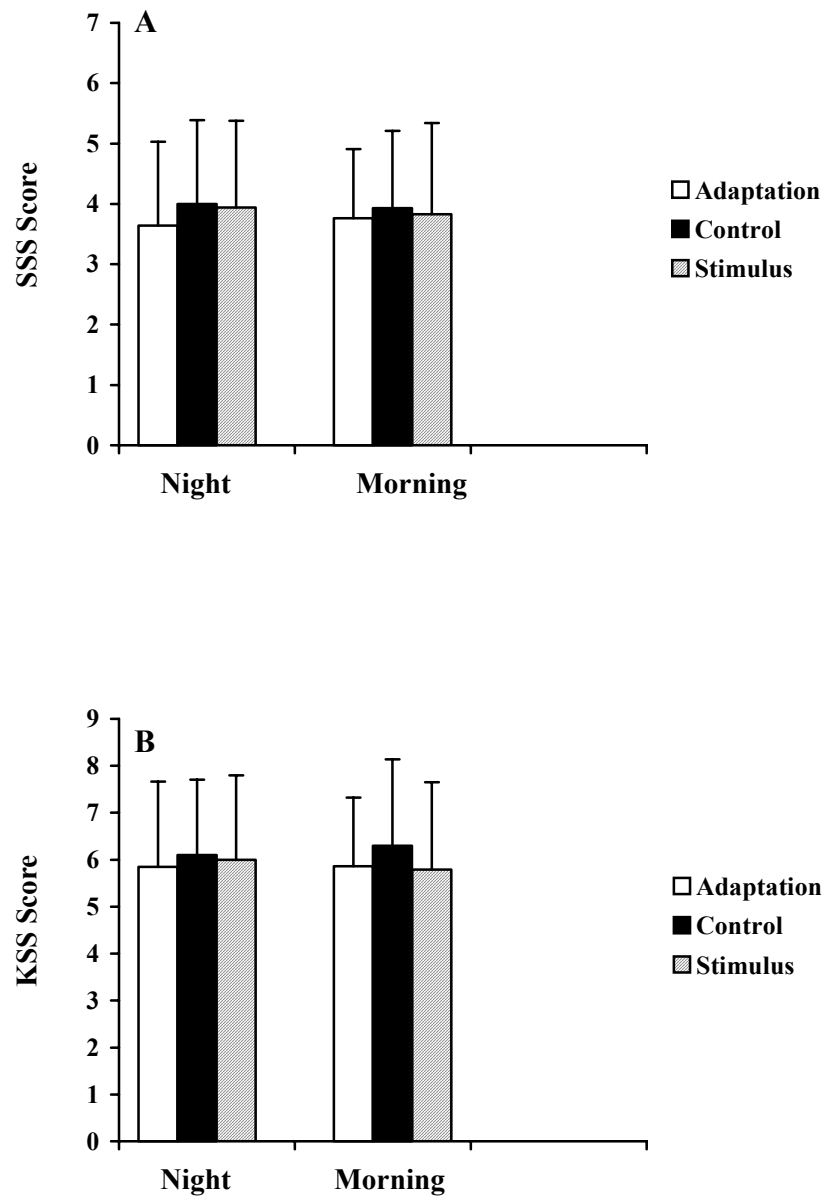
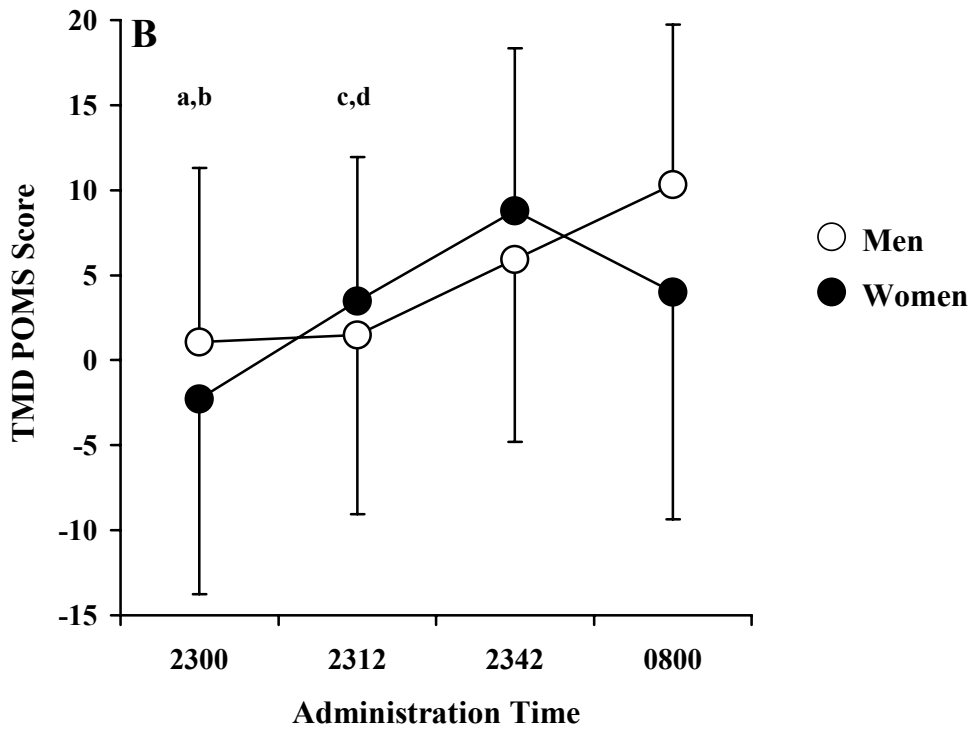
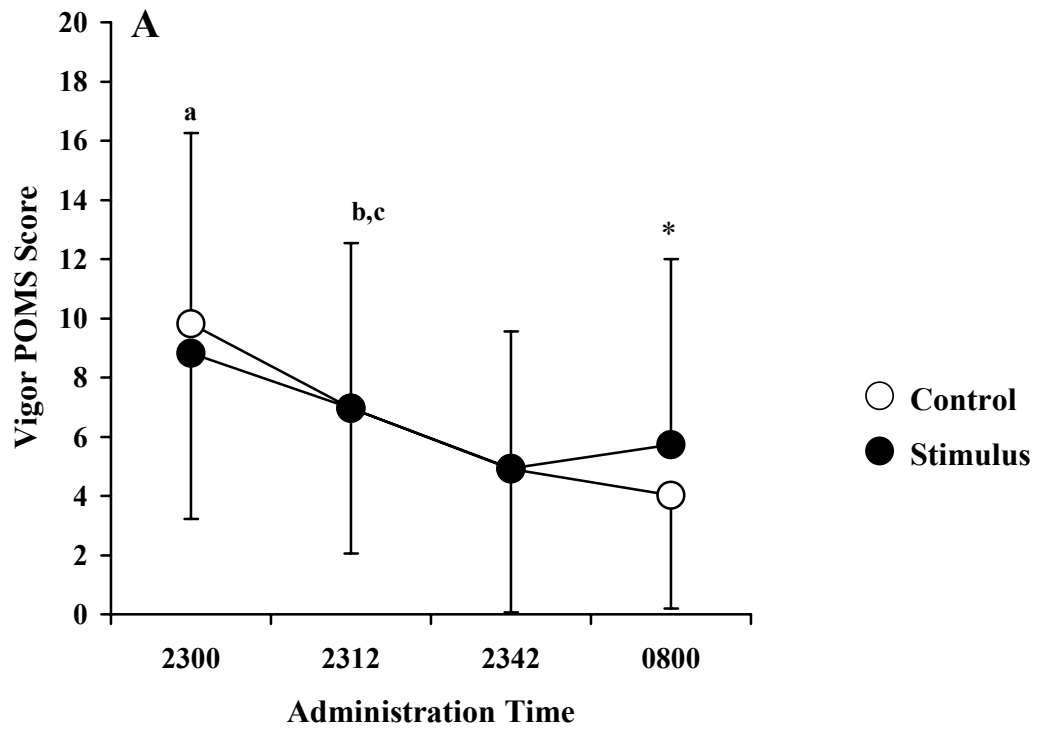


Figure 6



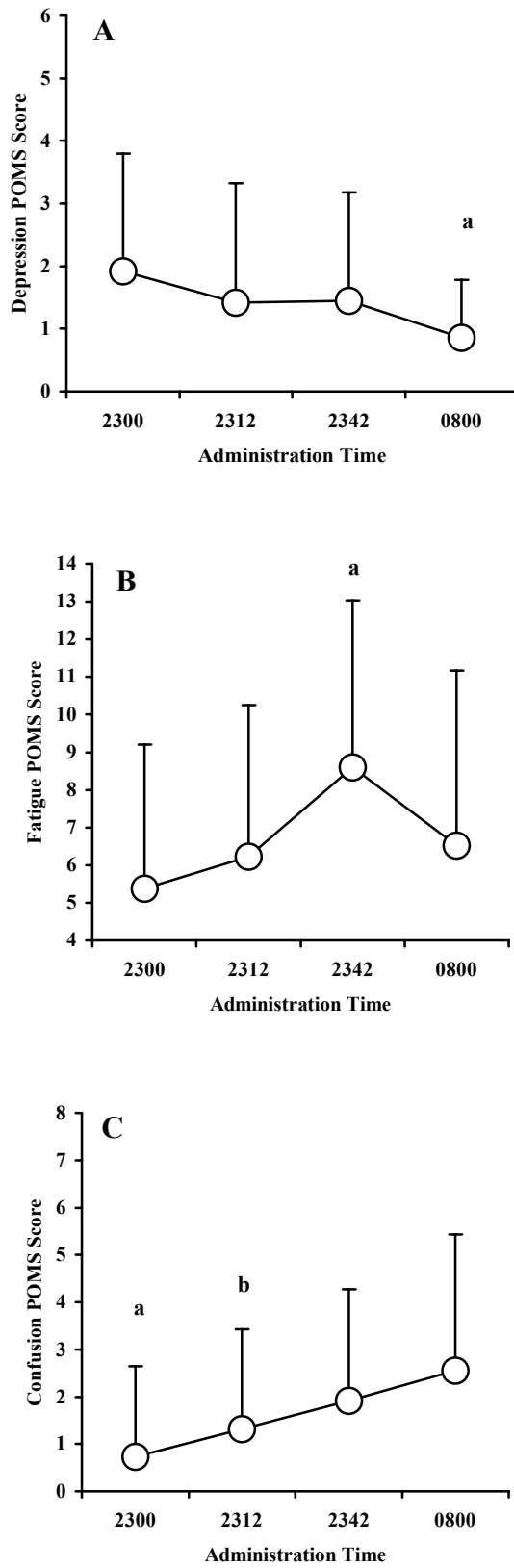


Figure 7