An ERP Examination of the Different Effects of Sleep Deprivation on Exogenously Cued and Endogenously Cued Attention

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Background: Behavior and neuroimaging studies have shown selective attention to be negatively impacted by sleep deprivation. Two unresolved questions are (1) whether sleep deprivation impairs attention modulation of early visual processing or of a later stage of cognition and (2) how sleep deprivation affects exogenously versus endogenously driven selective attention.

Study Objectives: To investigate the time course and different effects of sleep deprivation on exogenously and endogenously cued selective attention.

Design: Participants performed modified Attention Network Tests (ANTs) using exogenously and endogenously cued targets to index brain networks underlying selective attention. Target-locked event-related potentials (ERPs) were recorded as participants performed the Attention Network Tests on 2 days separated by 24 hours of total sleeplessness.

Participants: Fourteen US Military Academy cadets and 12 US Army soldiers from the Ironhorse Brigade, Ft. Hood, Texas.

Measurement and Results: For both Attention Network Tests, sleep deprivation led to slowed response times, decreased accuracy rates, a diminished positive P3 (450- to 550-ms) ERP component, and an enhanced P2 (312- to 434-ms) ERP component. In contrast, the parietal N1 (157- to 227-ms) ERP response was reduced with sleep deprivation for endogenously, but not exogenously, cued targets. These sleep deprivation-related effects occurred in the context of typical behavior and ERP patterns expected in a cued spatial-attention task.

Conclusions: These findings suggest that as little as 24 hours of sleep deprivation affects both early and late stages of attention selection but affects endogenously driven selective attention to a greater degree than it does exogenously driven selective attention.

Keywords: Sleep deprivation; selective attention; ERPs; Attention Network Test

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SLEEP DEPRIVATION IS A COMMON CONDITION AMONG THE GENERAL POPULATION[1] THAT HAS A NEGATIVE IMPACT ON COGNITION AND BEHAVIOR performance.[2] Understanding the factors underlying the behavior and neural consequences of sleep deprivation is important, given that many professionals in our society (medical, emergency, military) are often required to perform critical services under sleep-deprived conditions.[3-6] Accumulating evidence suggests that behavior impairments associated with sleep deprivation result from the influence of lowered physiologic arousal levels on cognitive performance.[7-13] Different cognitive operations may be more or less affected by lowered arousal levels, as it appears that sleep deprivation impacts some cognitive processes more than others.[14-15] Indeed, some high-level cognitive processes may be indirectly impacted by sleep deprivation via the influence of sleep deprivation on key low-level processes upon which the high-level processes rely.

One example of such a critical cognitive process is attention. Sustained vigilant attention, an ability required for many behavior tasks, is strongly affected by sleep deprivation.[16] Sleep deprivation also impairs behavior indices of selective attention—the ability to attend to one information source while excluding irrelevant items.[17-19] Researchers have examined psychophysiologic and brain metabolism correlates of attention changes in sleep deprivation. Electrodermal indices of attention-orienting responses to auditory stimuli are delayed, reduced in amplitude, and faster in habituation following sleep deprivation.[19] Three functional magnetic resonance imaging (fMRI) studies have shown that overall thalamic activity increases during selective[11,13] and sustained[12] visuospatial attention tasks under sleep deprivation. Because the thalamus is thought to form part of an alerting attention network,[20] such increases in thalamic activity may reflect a compensatory mechanism during a state of low arousal.[11,13] However, when task trials during which lapses of attention occur are analyzed separately from nonlapse trials, then thalamic activity is found to be reduced under sleep deprivation,[13] suggesting that such attention lapses may reflect a failure or reduced efficiency of compensatory mechanisms, such as feedback from cognitive control systems that monitor performance. Decreases in parietal cortex activation and reduced levels of deactivation in visual and insular cortex, as well as the cingulate gyrus, have also been found during sustained attention under sleep deprivation.[12] An additional fMRI study[21] found selective spatial-attention impairment during sleep deprivation to be accompanied by decreases in activation of the posterior cingulate cortex. The parietal cortex and posterior cingulate cortex are areas that are also known to be critical components of the brain attention networks underlying sustained and selective attention.[21-24]

One question not addressed by the above-cited studies is whether sleep deprivation-related decrements in selective attention reflect impairment of attention modulation of early

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visual processing or of a later stage of cognition, such as decision making or response selection. Behavior impairments may reflect any combination of sleep deprivation-related changes in early or late-stage attention, so behavior techniques alone cannot unambiguously differentiate between early- and late-stage attention selection.[25] Electrodermal and fMRI methods are also insufficient to resolve this issue due to the poor temporal resolution of these techniques. Instead, event-related potentials (ERPs), with their ability to resolve the timing of brain events on the order of milliseconds, are ideally suited to address this question. Several ERP studies have examined the impact of sleep deprivation on vigilant attention during target detection[26,27] and selective attention as it interacts with working memory[17,28-30] and visuomotor memory.[31] In general, these studies have found sleep deprivation to reduce early (~ 160-200 ms) or late (> 250 ms) ERP component amplitudes, or to delay the latencies of these components. Although these findings are consistent with the hypothesis that sleep deprivation affects early and late stages of both vigilant and selective attention, none of the studies investigating sleep-deprivation influences on selective attention used tasks designed to tap this cognitive process independently of working or visuomotor memory processes. Hence, a major aim of the present study was to investigate the time course of sleep-deprivation influences on selective attention using a task that was relatively independent of other cognitive processes.

An additional question considered by the present study was how sleep deprivation affects the neural consequences of voluntary shifts of selective attention driven by factors endogenous to individuals versus attention shifts driven primarily by exogenous factors. Neuroimaging and electrophysiologic studies carried out so far have only examined the effects of sleep deprivation on either exogenous or endogenous selective attention.[11,17,21] No study to date has investigated sleep-deprivation influences on selective attention in a manner allowing direct comparison between the outcomes of the 2 types of attention processes. It is possible that sleep deprivation might have a different effect on the neural concomitants of endogenous and exogenous selective attention, since the latter appears to rely primarily upon automatic bottom-up processes[32] that may be less susceptible to sleep deprivation.

Here we used ERPs to investigate (1) if sleep deprivation affects selective-attention modulation of early, late, or both early and late stages of cognitive information processing and (2) if sleep deprivation affects endogenous and exogenous selective attention differently. Using exogenous and endogenous versions of the Attention Network Test (ANT)—tasks designed to directly engage alerting, orienting, and executive attention functions[20,33,34]—we tested individuals while they were well rested and after they were totally sleep deprived for 24 hours. We predicted that sleep deprivation would affect the influence of both types of selective attention on late stages of cognitive processing at the least, but that the influence of exogenous selective attention would be affected by sleep deprivation to a lesser degree than the influence of endogenous attention due to the reliance of exogenous attention on bottom-up processes that are less susceptible to sleep deprivation.

METHODS

Participants

Fifteen cadets from the United States Military Academy (referred to herein as the West Point, or WP, group), and 14 US Army soldiers from the Ironhorse Brigade, Ft. Hood, Texas (referred to herein as the Fort Hood, or FH, group) participated in this study, in which they were tested in 2 sessions (Day 1, Day 2) separated by 24 hours, during which time they were not allowed to sleep at all. Data from 1 West Point cadet and 2 Fort Hood soldiers were excluded from the final analysis due to an insufficient number of acceptable trials on Day 1 and/or Day 2, after excluding trials contaminated with electroencephalographic (EEG) artifact and eye blinks or movements (see EEG analysis section below). Thus, the final participant sample consisted of 14 West Point cadets (8 women and 6 men; 21.29 ± 0.55 years or age, 11 right-handed) and 12 Fort Hood soldiers (2 women and 10 men; 24.0 ± 0.84 years of age, all right-handed). All participants were fully informed of the experiment methods and proceedings before they consented to participate. This study was approved by the appropriate institutional review boards (the University of Texas at Austin and the United States Military Academy).

Stimuli and Procedure

Day 1

Participants completed 2 visits (Day 1, Day 2) at the University of Texas at Austin Imaging Research Center. Before the Day 1 visit, participants were instructed to obtain approximately 8 hours of sleep; a human monitor enforced this instruction. Participants underwent EEG recording in the morning, approximately 1 to 5 hours after awakening.

After the setup for EEG recording, subjects performed exogenous and endogenous versions of the ANT. A schematic of the basic ANT protocol is shown in Figure 1A. At the beginning of each trial, participants fixated on a centrally displayed cross. Next, depending on condition, a double-arrowhead cue stimulus was (Neutral-Cue or Spatial-Cue conditions) or was not (No-Cue condition) displayed for 200 milliseconds. After a variable interval (300 to 1450 ms; mean interval = 842 ms), a letter target stimulus (*X* or *H*; Figure 1A, upper inset) was presented for 300 milliseconds, flanked on either side by matching letters (congruent condition) or mismatching letters (incongruent condition). At a viewing distance of 100 cm, the cue stimuli subtended approximately 0.85° of vertical visual angle, with a gap separation of approximately 0.17°; individual target letters subtended approximately 0.43° horizontally (~ 2.17° across all 5 letters) and 0.40° vertically. All stimuli were white in color, were displayed against a black background, and were presented to the participants on an 18-inch computer cathode ray tube screen with a 60-Hz refresh rate. DMDX stimulus presentation software[35] was used to record subjects' categorization responses and to time the stimulus presentations.

The participants' task was to categorize the center letter of the targets as being the same as (*congruent*) or different from (*incongruent*) the flanking letters by pressing 1 of 2 computer mouse

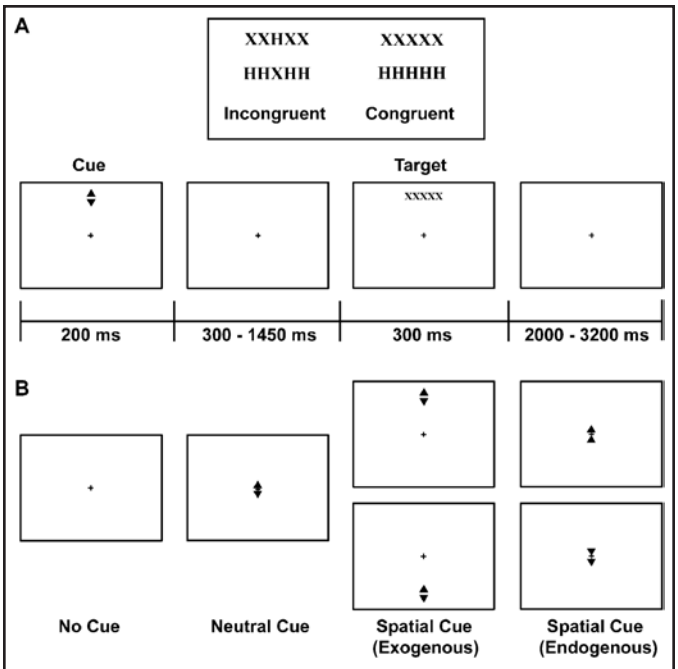

Figure 1—(A) Basic Attention Network Test (ANT) protocol. A cue stimulus was presented for 200 ms. After a variable interval (300-1450 ms), a target stimulus was displayed for 300 ms. Target stimuli (upper right inset) consisted of X or H letters flanked by incongruent or congruent flankers. Intertrial intervals ranged from 2000-3200 ms. (B) The 3 cueing conditions used in each task: No Cue, Neutral, and Spatial Cue. The Spatial-Cue condition differed according to Exogenous or Endogenous ANT (see text for details).

buttons held in the right hand. Participants pressed the left button for congruent responses and the right button for incongruent responses. Target stimuli were presented with equal probability at 1 of 2 locations 1.5° above or below fixation. Participants were instructed to view peripherally displayed stimuli by shifting their visuospatial attention toward the target location while keeping their gaze directed toward the central fixation cross. They were also encouraged to use the cues to guide their attention toward the locations of subsequently presented targets.

Target trials were categorized according to whether the targets were preceded by a cueing stimulus that was predictive of the spatial location of the subsequently presented target. For No-Cue trials (Figure 1B, 1st column), no cueing stimulus preceded the targets. For Neutral-Cue trials (Figure 1B, 2nd column), targets were preceded by a double-triangle symbol that was ambiguous as to the target's spatial location. For Spatial-Cue trials (Figure 1B, 3rd and 4th columns), targets were preceded by a double-triangle symbol that predicted the subsequent spatial location of the targets with 100% accuracy.

Subjects were administered 2 versions of this test that assessed either exogenous or endogenous allocation of selective attention. The Exogenous and Endogenous versions of the ANT were exactly the same except for the Spatial-Cue condition. In the Exogenous ANT (Figure 1, lower inset, 3rd column), the Spatial-Cueing triangles pointed in opposite directions but appeared in the spatial locations of the subsequently presented targets. Thus, for the Exogenous ANT, attention shifts were driven primarily by a factor external to the subjects, in that the onset of the cueing stimuli captured attention toward the location of the subsequently presented target. In the Endogenous ANT (Figure 1, lower inset, 4th column), the Spatial-Cueing triangles all appeared at central fixation with the triangles pointing in the direction of the spatial locations of the subsequently presented targets. Here, attention shifts were driven primarily by a factor internal to the participants, in that they voluntarily shifted their attention after interpreting the triangle directions.

Following a training block of 10 trials, participants received 2 to 3 blocks of each ANT. Each block consisted of 40 Spatial-Cue trials, 40 Neutral-Cue trials, and 40 No-Cue trials (120 trials total per block). The interval between the offset of a target and the next trial was 2000 to 3200 milliseconds (mean interval = 2562 ms); the time limit to make a response was 2000 milliseconds. Exogenous and Endogenous ANTs were performed in counterbalanced order across subjects.

Day 2

Twenty-four hours after initial testing, participants completed an additional two to three 120-trial blocks of Exogenous and Endogenous ANT. Participants performed the 2 ANTs in the same order as on Day 1. Importantly, over the 24-hour period between the first and second test, participants were monitored around the clock, engaged in group activities, and were not allowed to sleep. Participant fatigue was such that they struggled to stay awake during Day 2 task performance; thus, careful monitoring and intervention by the experimenter were required to keep participants awake and on task.

ERP Acquisition

Sixty-seven channels of scalp EEG signals were recorded while each subject performed the ANTs, using active Ag/AgCl electrodes mounted in a BioSemi electrode cap (BioSemi B. V., Amsterdam, The Netherlands). Recording sites in the cap included standard and extended 10-20 system locations (Figure 2). Four additional electrodes were affixed to the outer canthi and inferior orbits of both eyes to monitor vertical and horizontal electrooculographic (EOG) activity (eg, eye movements and blinks). All channels were amplified by a Biosemi Active II amplifier system in 24-bit DC mode at an initial sampling rate of 2048 Hz (400-Hz bandwidth) downsampled online to 256 Hz, with EEG signals recorded with respect to a common mode sense-active electrode placed between sites PO3 and POZ. Because active electrodes make skin preparation redundant, electrode impedances were not measured; however, half-cell potentials of the electrode/gel/skin interface were kept between ± 40 mV, following standard recommendations for the Active II system.

Behavior Data Analysis

Analysis of behavior data was restricted to trials with response times (RTs) longer than 300 milliseconds and less than 2000 milliseconds that contained no eye movements within the cue or target interval or significant EEG artifacts (see ERP data analysis section, below); in this manner, the behavior trials represent the same set of trials that was entered into the ERP analysis. For each participant, mean hit rates (percentage of correct trials within the total number of correct and incorrect responses

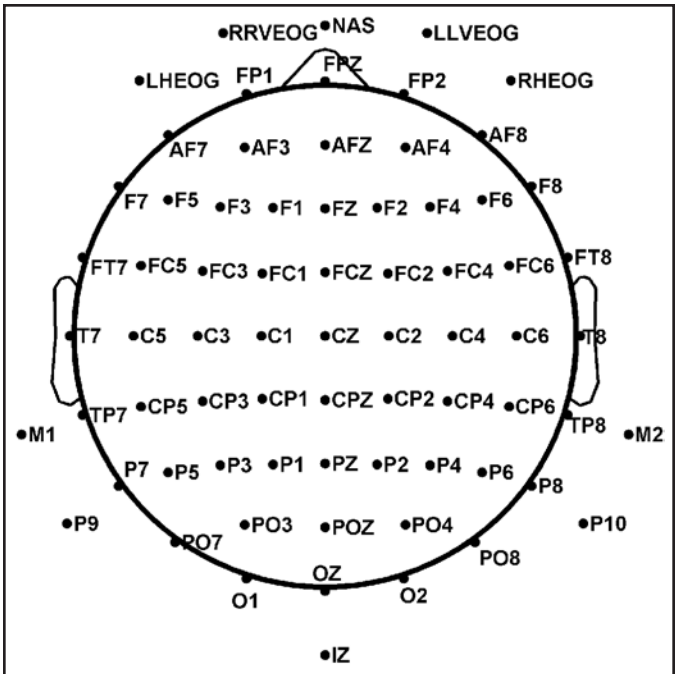

Figure 2—Extended 10-20 scalp locations of electroencephalographic (EEG) recording electrodes. Note that sites outside the radius of the head represent locations that are below the equatorial plane (FPZ-T7-T8-OZ plane) of the (assumed spherical) head model.

after removal of timeout and artifact trials) and RTs were computed for each combination of cue type and congruency conditions (6 combinations total). All behavior data were analyzed via repeated-measures analysis of variance (ANOVA) with appropriate corrections for nonsphericity and multiple comparisons during posthoc testing (see Results section).

ERP Data Analysis

Continuous data were imported offline into the MATLAB computing software environment (The Math Works, Inc., Natick, MA) using the EEGLAB toolbox[36] for MATLAB, in which all subsequent analysis was performed via in-house scripts that utilized EEGLAB functions. Single 2000-millisecond EEG epochs were extracted from -750 milliseconds to 1250 milliseconds with respect to the onset of the target stimuli; trials with incorrect responses or trials with RTs outside the acceptable time range (300 - 2000 ms) were excluded from further analysis. Next, the trials were transformed to a linked-mastoids reference for the purposes of removing muscle and signal artifacts from the EEG record by visual inspection. Faulty EEG channels were replaced using an EEGLAB-based spherical spline interpolation algorithm[37] (m = 5; 50-term expansion) applied to the remaining channels. The mean number of interpolated channels for Day 1 and Day 2 was 1.04 ± 0.19 and 1.38 ± 0.22, respectively. Typically, 1 to 2 channels were interpolated for those subjects requiring channel interpolation, with the majority of interpolated channels located outside the scalp regions of interest under statistical analysis (see below).

In a cued-attention task of this nature, it is necessary to reject trials containing blink or saccade-related EOG activity in the cue-target interval, thus removing trials in which participants may have viewed targets via saccades to the target location rather than by covert shifts of visuospatial attention. Additional horizontal and vertical EOG channels were computed offline for this purpose. The horizontal EOG channel was computed as the bipolar montage of the outer canthi EOG signals; the vertical EOG channel was computed from the bipolar montage of an electrode located at the nasion and the average of the electrodes placed at the inferior orbits. EEG trials containing EOG amplitudes higher than 50 μV or lower than -50 μV (after removal of the constant DC offset from the EOG signal) within the cue-target interval were rejected from the analysis in MATLAB via automatic algorithm. The rejection interval for target trials spanned the onset of the preceding cue stimulus (ranging from -500 ms to -1650 ms pretarget onset) to 300 milliseconds after the target onset. A second stage of visual inspection was performed to remove any low-amplitude EOG contamination that was missed by the automatic rejection algorithm. In addition, ERP responses in the time range greater than 300 milliseconds after the target onset were also of interest. Therefore, EOG activity in this interval was removed from the scalp EEG signals via a recursive least-mean squares regression procedure[38] implemented within the Automatic Artifact Removal toolbox v1.3 for MATLAB and applied to the entire 2 seconds of each trial.

After rejection or correction of the EOG artifacts, the derived horizontal and vertical EOG channels were removed from the data, and the single EEG trials were then transformed to an average reference. To improve the estimate of the average reference, the 2 outer canthi and 2 inferior orbit EOG channels were included in the calculation (71 channels total entered into the average reference estimate). Next, the average-reference transformed EEG trials were band-pass filtered between 0.45 Hz and 32 Hz (166-point 0-phase-shift, finite-impulse response filter with half amplitudes at the stated frequencies) and epochs were truncated to -200 milliseconds to 600 milliseconds. The relatively large high-pass cutoff value of 0.45 Hz reduced low-frequency drift arising from minor fatigue-related head motion and residual cue-induced activity[39] that may have contaminated target epochs. EEG epochs were then separated according to each of the 3 possible preceding cue types (Spatial, Neutral, No Cue), collapsed across congruency condition. We did not further subdivide epochs by congruency condition due to the lower number of available trials on Day 2. The average numbers of trials entering into the ERPs are given in Table 1.[a] Target-locked ERPs were computed by averaging trials separately for each condition and subject and were baseline corrected to the -200 millisecond to 0 millisecond prestimulus interval.

Target-locked ERP amplitudes were measured for 3 early posterior components: the P1 (90-150 ms), the N1 (150-250 ms), the P2 (250-450 ms) and for the probability insensitive P3 (450-550 ms). The time windows used to analyze each of these 3 components were visually estimated from the across-subject grand-average ERPs on Days 1 and 2 and are typical of these components when evoked in response to visual stimuli.[40] Each ERP component was quantified as the mean signal amplitude over those contiguous time points in which a component was greater than or equal to 50% of its across-participant grand-average peak amplitude within the interval for which that component was defined; mean amplitudes were extracted over these time points for each participant and condition of interest. This method of quantifying ERP components

Table 1—ERP Trial Numbers per Task, Condition, and Day

	Exogenous		Endogenous	
	Day 1	Day 2	Day 1	Day 2
Spatial	65 (4)	45 (3)	63 (4)	45 (3)
Neutral	65 (3)	45 (3)	63 (4)	46 (3)
No Cue	57 (4)	41 (3)	58 (3)	40 (3)

Values represent mean trial numbers across participants entering into each grand-average event-related potential (ERP). SE values in parentheses. Day 2 included sleep deprivation.

has the advantage of mitigating any artifact effects arising from between-condition imbalances in trial numbers,[41] as observed here between the Day 1 and Day 2 conditions (Table 1).

All ERP results were analyzed by examining component mean amplitudes in repeated-measures ANOVAs with appropriate corrections for nonsphericity and multiple comparisons during post-hoc testing (see Results section). For graphic display, individual ERPs were averaged across participants within each condition, generating representative grand-average ERPs for each condition. ERP-component scalp topographies were visualized by first computing the average 50% peak cutoff time limits across electrodes and condition. The mean-amplitude values at each electrode within that time interval were displayed as scalp topographies using algorithms from the EEGLAB MATLAB toolbox.

RESULTS

Behavior

The RT and hit-rate data were statistically analyzed via an omnibus ANOVA with within-participants factors of Task Type (Exogenous, Endogenous), Day (Day 1, Day 2), and Cue Type (Spatial, Neutral, No Cue), with Group as a between-subjects factor. The P values of all within-subject tests involving more than 2 conditions were adjusted using the Greenhouse–Geisser correction for nonsphericity. For ease of interpretation, reports of all significant behavior F tests subject to Greenhouse-Geisser correction include uncorrected degrees of freedom, corrected P values, and the Greenhouse-Geisser epsilon value ε. All post-hoc comparisons were Bonferroni corrected.

Target RTs

For the RT data, a main effect of Cue Type ($F_{2,48} = 63.19$, $P < 0.001$, $\varepsilon = 0.92$) indicated that participants were faster to correctly categorize targets for Spatial-Cue trials than for Neutral-or No-Cue trials ($P < 0.003$) and were also faster for Neutral- than No-Cue trials ($P < 0.003$); see Table 2, right columns. Furthermore, RTs to correctly categorize targets were longer on Day 2 when participants were fatigued, as compared with Day 1 when they were well rested (main effect of Day: $F_{1,24} = 22.61$, $P < 0.001$); see Table 2, right columns.

Target Hit Rates

These RT differences were observed in the context of high hit rates across both days (Table 2, left columns) that did not significantly differ between cue conditions (P values > 0.09). Thus, the RT advantages of spatial cueing and target-flanker congruency that were observed on both days were not due to a tradeoff between speed and hit rate. Nevertheless, when examining the hit-rate data in the same repeated-measures ANOVA as above, a significant main effect of Day ($F_{1,24} = 12.57$, $P < 0.002$) indicated that hit rates were slightly lower overall on Day 2, as compared with Day 1; see Table 2, left columns.

Table 2—Behavior Results

	Hit Rate, %		RT, ms	
	Day 1	Day 2	Day 1	Day 2
Exogenous				
Spatial	96 (1)	92 (1)	694 (21)	759 (27)
Neutral	96 (1)	90 (2)	740 (23)	819 (26)
No Cue	95 (1)	91 (2)	775 (23)	837 (27)
Endogenous				
Spatial	97 (1)	93 (1)	687 (22)	771 (30)
Neutral	94 (1)	92 (1)	724 (22)	800 (29)
No Cue	94 (1)	91 (1)	746 (22)	824 (30)

Mean hit rates and reaction times (RTs) per task, condition, and day. SE values in parentheses. Day 2 included sleep deprivation.

Target Misses

We also compared the proportions of trials on Days 1 and 2 in which no behavior response was given within the specified time limits (ie, 2000 ms "timeouts") to index the preponderance of attention lapses during this task. This comparison was performed after collapsing across all conditions because insufficient trials were available to include cueing or congruency as factors. This overall proportion of timeouts increased on Day 2, compared with Day 1, for both ANTs (17% ± 2% SE on Day 2 vs 4% ± 1% SE on Day 1, $F_{1,24} = 23.91$, $P < 0.001$). This finding supports the observation that subjects struggled to stay awake and on task during Day 2 task performance and, thus, required careful monitoring and intervention by the experimenter to keep the subjects awake and on task.

Event-related Potentials

Figure 3 shows the scalp topographies of the grand-average target-locked P1, N1, P2, and P3 ERP components elicited in response to Spatially, Neutrally, and Non-Cued targets during the Exogenous and Endogenous ANT. For brevity, ERP-component topographies shown are collapsed across Day because similar topographies were present across both Days 1 and 2. The P1, N1, and P3 ERP components displayed a dipolar scalp topography typically seen in response to visual stimuli, consisting of positive (P1, P2) or negative (N1) potential changes over posterior scalp locations. These responses were accompanied by opposite polarity changes over anterior scalp locations (see Figure 3). The similarity in anterior and posterior topography suggests that the 2 responses are likely not completely independent, due in part to volume conduction of electric signals emanating from the same, likely posterior, source or sources. The one exception to this pattern was the P3 ERP response,

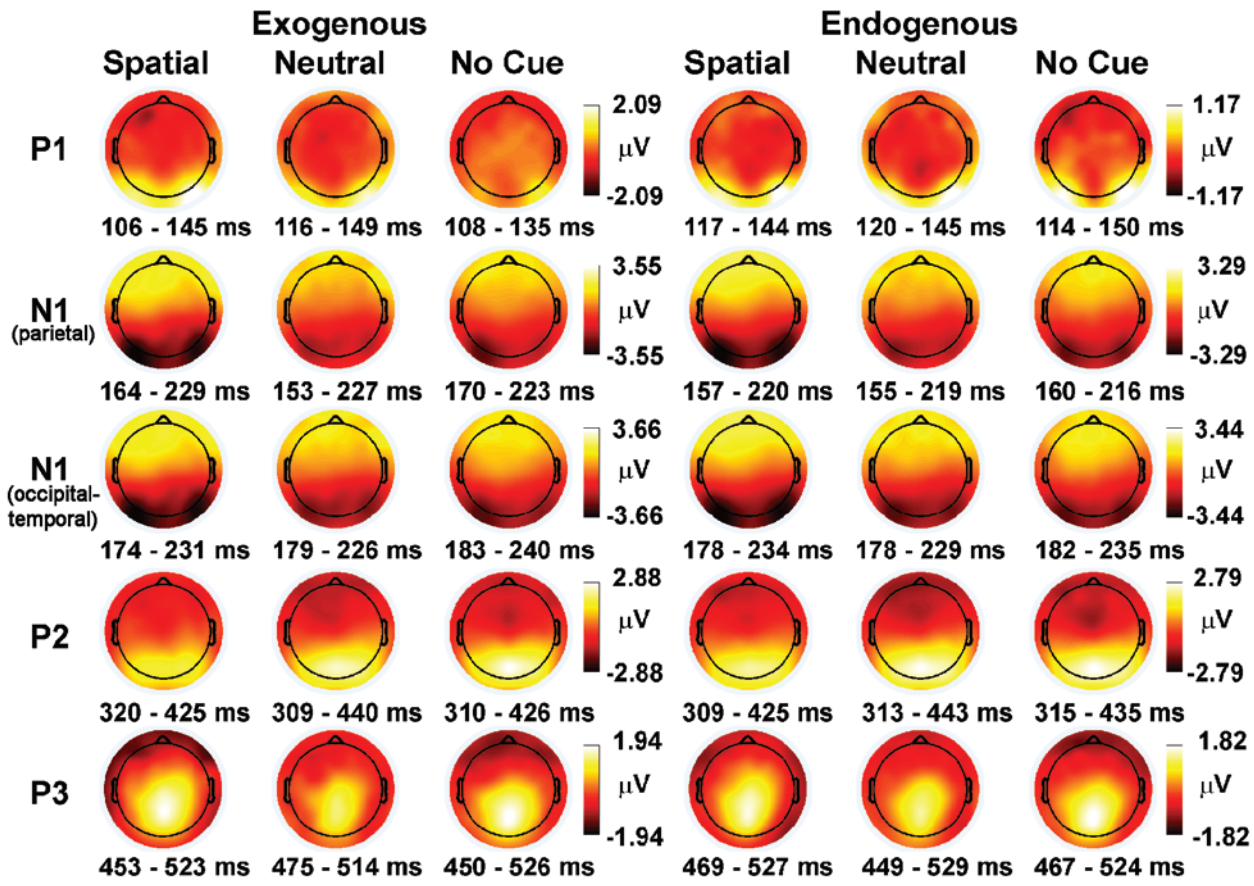

Figure 3—Scalp topographies of P1, N1, P2, and P3 event-related potential (ERP) components (averaged over stated intervals) elicited during the 2 Attention Network Tests (ANT). Light colors indicate positive values, dark colors indicate negative values. Intervals listed are those contiguous time points in which ERP component amplitudes were greater than or equal to 50% of the across-participant grand-average peak amplitude (see Methods).

Table 3—ERP Cueing Effects

Cue Condition	Exogenous ANT				Endogenous ANT			
	P1	N1	P2	P3	P1	N1	P2	P3
Spatial Cue	1.22 (0.25)	-2.74 (0.30)	1.11 (0.13)	1.48 (0.24)	0.75 (0.23)	-2.78 (0.25)	1.39 (0.13)	1.30 (0.23)
Neutral Cue	0.87 (0.24)	-1.94 (0.30)	1.51 (0.13)	1.05 (0.22)	0.66 (0.24)	-2.01 (0.30)	1.61 (0.17)	1.28 (0.17)
No Cue	0.56 (0.18)	-2.04 (0.32)	1.45 (0.18)	1.39 (0.19)	0.77 (0.16)	-2.01 (0.31)	1.71 (0.17)	1.27 (0.21)

Mean P1, ventral N1, P2, and P3 event-related potential (ERP) amplitudes by Cue condition collapsed across Day (all values in μV; SE in parentheses). ANT refers to Attention Network Test.

which was positive in polarity over most of the scalp except the anterior ventral regions. Figures 4 and 5 show grand-average ERPs for Exogenous and Endogenous ANTs collapsed across day; Figures 6 to 8 show the effects of sleep deprivation on the grand-average ERPs. Table 3 summarizes the mean ERP amplitudes for each task, collapsed across day; Tables 4 and 5 summarize the sleep deprivation-related ERP effects.

The mean ERP amplitudes extracted at each electrode site were averaged across separate posterior regions of interest for each component before statistical analysis. This procedure has the advantage of simplifying data interpretation and reducing the problem of spurious interactions involving scalp location.[42] P1 and P2 amplitudes were collapsed across ventral occipital regions (IZ, OZ, O1,O2, POZ, PO3, PO4, PO7, PO8, P7, P8, P9, P10). N1 amplitudes were analyzed over 2 separate regions, ventral lateral occipital-temporal sites (O1,O2, PO3, PO4, PO7, PO8, P7, P8, P9, P10) and dorsal parietal/centro-parietal locations (PZ, P1, P2, P3, P4, P5, P6, CPZ, CP1, CP2, CP3, CP4, CP5, CP6). The P3 was analyzed over midline parietal, centro-parietal, and central sites (PZ, P1, P2, CPZ, CP1, CP2, CZ, C1, C2); see Figure 2 for a schematic of EEG recording locations. Posterior regions of interest were chosen because these locations demonstrate the maximum loci of visual ERP components,[40] as well as maximal ERP effects related to attention biasing of visual target processing.[39,43] Furthermore, the analysis of the N1 component over separate ventral lateral occipital-temporal and dorsal parietal/central-parietal scalp regions is supported by

prior observations of dissociable attention-related N1 responses.[44] The regionally averaged ERP amplitudes for each component were statistically analyzed via an omnibus repeated-measures ANOVA with Task Type (Exogenous, Endogenous), Day (Day 1, Day 2), and Cue Type (Spatial, Neutral, No Cue) as within-participants factors and with Group as a between-subjects factor. P values of within-subject tests were adjusted via Greenhouse–Geisser corrections for nonsphericity when appropriate (reports of all significant Greenhouse–Geisser corrected F tests include uncorrected degrees of freedom, corrected P values, and the Greenhouse-Geisser epsilon value ε), and all posthoc comparisons were Bonferroni corrected.

P1 Results

A Task × Cue-Type interaction was significant for this component ($F_{2,48}$ = 4.24, P < 0.02, ε = 0.98). Decomposition of this interaction indicated that the P1 was larger during Spatial-Cue versus No-Cue trials for the Exogenous ANT (P < 0.006); see Figure 4 and Table 3. No other main or interaction effects were significant for the P1 (P values > 0.08). Most importantly, there was no effect of Day (P = 0.99).

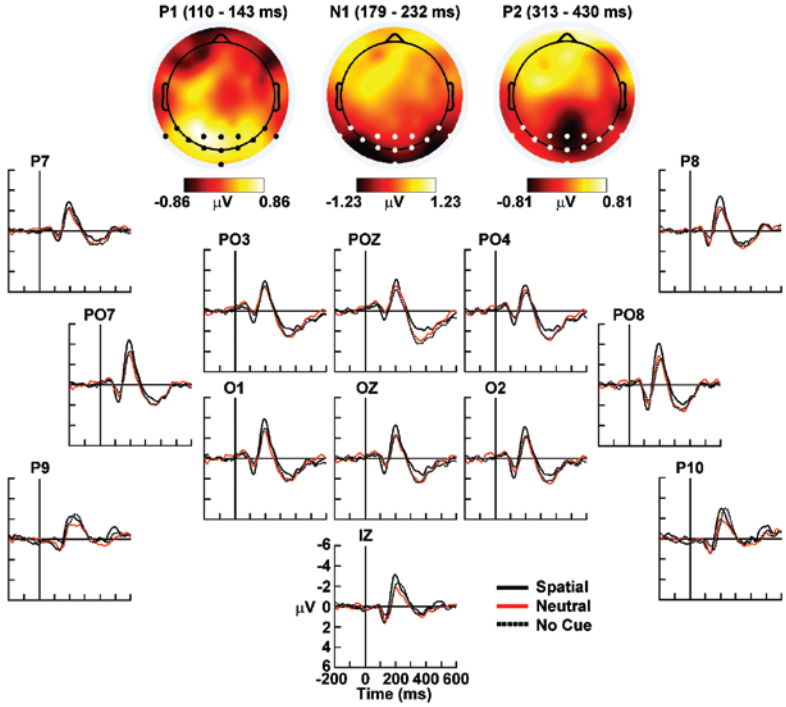

Figure 4—Representative event-related potential (ERP) effects of exogenous selective attention. Waveforms depict grand-average Exogenous Attention Network Test target-locked ERPs for Spatial-Cue (solid black line), Neutral-Cue (red line), and No-Cue (dashed black line) conditions averaged across fresh and fatigue conditions. Negative polarity is oriented upward. Waveform scalp locations are shown on topographic maps (top row) displaying Spatial-Cue—Neutral-Cue differences for mean P1, ventral N1, and P2 ERPs. Spatial-Cue—No-Cue contrasts (not shown) exhibited similar topographies. Light colors indicate positive values; dark colors indicate negative values.

Occipital-Temporal N1 Results

A main effect of Cue Type was significant for occipital-temporal N1 amplitudes ($F_{2,48}$ = 19.51, P < 0.001, ε = 0.89), indicating a larger N1 response for Spatial versus Neutral and Spatial versus No-Cue trials (P values < 0.003); see Figures 4 and 5 and Table 3. No other main effects or interactions were significant for the occipital-temporal N1. Again, there was no effect of Day (P = 1).

Parietal N1 Results

Main effects of Day ($F_{1,24}$ = 17.55, P < 0.001) and Cue Type ($F_{2,48}$ = 5.45, P < 0.007, ε = 1.00) were significant for the parietal N1 amplitudes. These effects were qualified by a significant Task × Day × Cue-Type interaction ($F_{2,48}$ = 3.59, P < 0.035, ε = 0.95). Decomposition of this interaction indicated that parietal N1 responses in response to Spatially Cued targets were reduced in amplitude from Day 1 to Day 2 for the Endogenous ANT (P < 0.002) but not for the Exogenous ANT (P > 0.27); see Figure 6 and Table 4. In contrast, Neutral and No-Cue trials displayed smaller parietal N1 amplitudes for Day 2 versus Day 1 for both ANTs (P values < 0.021); see Figure 6 and Table 4.

Finally, a significant Task × Group crossover interaction was present for the parietal N1 ($F_{1,24}$ = 5.30, P < 0.03). Mean N1 amplitudes were smaller for the Exogenous versus Endogenous ANT for the WP group (-1.06 µV ± 0.16 µV vs -1.14 µV ± 0.17 µV, respectively) and larger for the Exogenous versus Endogenous ANT for the FH group (-1.14 µV ± 0.18 µV vs. -0.91 µV ± 0.18 µV, respectively). However, none of these differences were significant on follow-up posthoc testing (P values > 0.14). No other main effects or interactions were significant for the parietal N1 (P values > 0.08).

P2 Results

A main effect of Cue Type was significant for the P2 ($F_{2,48}$ = 8.68, P < 0.002, ε = 0.74), indicating larger P2 amplitudes for Spatial versus Neutral and Spatial versus No-Cue trials (P values > 0.021); see Figures 4 and 5, and Table 3. A significant main effect of Day ($F_{1,24}$ = 9.27, P < 0.006) indicated an increase in P2 amplitude from Day 1 to Day 2. Finally, a main effect of Task ($F_{1,24}$ = 8.43, P < 0.008) indicated larger P2 amplitudes for Endogenous versus Exogenous ANT; see Figure 7 and Table 5. No other main effects or interactions were significant (P values > 0.09).

P3 Results

A significant main effect of Day ($F_{1,24}$ = 7.35, P < 0.012) indicated an overall decrease in P3 amplitude from Day 1 to Day 2; see Figure 8 and Table 5. No other main effects or interactions were significant (P values > 0.08).

DISCUSSION

The present study used ERPs to investigate whether sleep deprivation impairs early-stage or late-stage attention selection and whether such impairment differs according to whether attention is allocated in an exogenous or endogenous manner. To begin, it should be noted that we observed typical effects of

Table 4—Effects of Sleep Deprivation on the Dorsal N1 ERP Component

Day	Exogenous ANT			Endogenous ANT		
	Spatial Cue	Neutral Cue	No Cue	Spatial Cue	Neutral Cue	No Cue
1	-1.33 (0.17)	1.25 (0.17)	-1.09 (0.14)	-1.48 (0.17)	-1.34 (0.22)	-0.99 (0.15)
2	-1.16 (0.13)	-1.01 (0.20)	-0.76 (0.12)	-0.79 (0.11)	-0.76 (0.13)	-0.78 (0.17)

Mean dorsal N1 ERP event-related potential (ERP) amplitudes by Day and Cue Type (all values in μV; SE in parentheses). ANT refers to Attention Network Test.

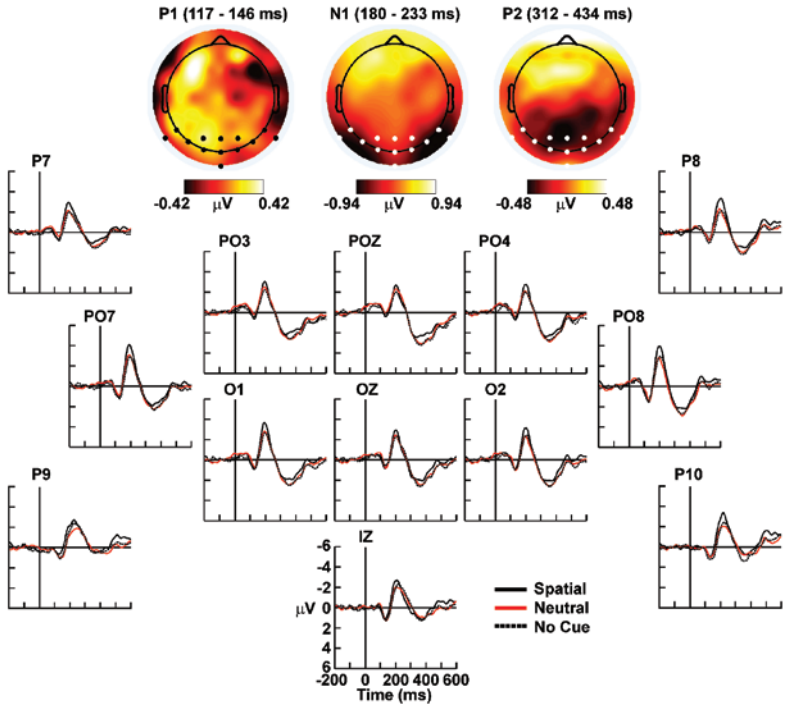

Figure 5—Representative event-related potential (ERP) effects of Endogenous selective attention. Waveforms depict grand-average Endogenous Attention Network Test (ANT) target-locked ERPs for Spatial-Cue (solid black line), Neutral-Cue (red line), and No-Cue (dashed black line) conditions averaged across fresh and fatigue conditions. Negative polarity is oriented upward. Waveform scalp locations are shown on topographic maps (top row) displaying Spatial-Cue – Neutral-Cue differences for mean P1, ventral N1, and P2 ERPs components. Spatial-Cue – No-Cue contrasts (not shown) exhibited similar topographies. Light colors indicate positive values; dark colors indicate negative values.

attention modulation of target processing, which has been previously reported for Spatial-Cueing tasks of this type.[25,33,34] Participants exhibited faster RTs, larger-amplitude early ERPs (P1 and/or N1 components), and smaller-amplitude late ERPs (P2 component) when selective spatial attention was exogenously and endogenously allocated to the targets. The clear presence of these effects demonstrates that this experimental paradigm successfully tapped selective-attention processes, despite the fact that participants were highly fatigued on Day 2.

The Effects of Sleep Deprivation on Visual Attention

With respect to the main purpose of this study, sleep deprivation decreased the parietal N1 response to spatially cued targets only during the Endogenous ANT and not the Exogenous ANT; that is, smaller parietal N1 amplitudes were observed with fatigue only for those trials requiring endogenously cued shifts in attention to target locations (Figure 6). Since the parietal N1 component is known to be highly sensitive to manipulations of attention,[44] this observation supports the conclusion that exogenous shifts of attention depend more on low-level automatic cognitive processes[32] that may be less influenced by a general decrease in arousal, as would be seen in sleep deprivation. This conclusion is consistent with other evidence that automatic processes are relatively insensitive to alcohol intoxication,[45] vigilance decrements,[46] and high levels of mental load[47]. Indeed, previous research suggests that exogenous shifts of attention are insensitive to high levels of mental load.[32] Nonetheless, given that there were some qualitative N1 decreases during Spatial-Cue trials for the Exogenous ANT, it is likely that exogenous attention processes will ultimately be more affected by more extended periods of sleep deprivation, ie, longer than 24 hours. Furthermore, in the present study, the parietal N1 component was also reduced with fatigue during Neutral- and No-Cue trials for both ANTs, suggesting that sleep deprivation also decreased general vigilance as well. Thus, the early stages of selective-attention processing can be affected by as little as 24 hours of sleep deprivation. This is consistent with earlier observations that ERP components in this latency range decrease with sleep deprivation during target-discrimination tasks requiring sustained vigilant attention[26,27] and working-memory tasks that engage selective attention by requiring participants to compare stimuli to target locations designated on previous trials.[30]

The fatigue-related N1 ERP reductions observed here occurred over parietal and centro-parietal scalp regions but not over occipital-temporal scalp regions. This observation is consistent with previous evidence of dissociable attention-sensitive parietal and occipital-temporal N1 components, the latter of which has been found to arise from activity in the lateral occipital cortex.[44] The source of the parietal N1 has not yet been determined, but, if it is located in parietal cortex, then it is likely to be a deep source, since a previously published current-source density analysis that is sensitive to superficial cortical ERP sources found minimal parietal-scalp current associated with this component.[44] Indeed, the parietal N1 ERP component may reflect the summed volume-conducted activity of multiple brain regions (parietal, occipital, temporal) involved in visual target processing; hence, we cannot definitively conclude that the present findings reflect a modulation of ERP source activity located solely in parietal cortex (the establishment of the cortical locus of the present N1 ERP effects is beyond the scope of the present paper and awaits further research). Nonetheless, a parietal generator for the present fatigue-related ERP effects would be consistent with previous fMRI studies of fatigue-attention relationships that showed decreases in dorsal parietal and posterior cingulate corti-

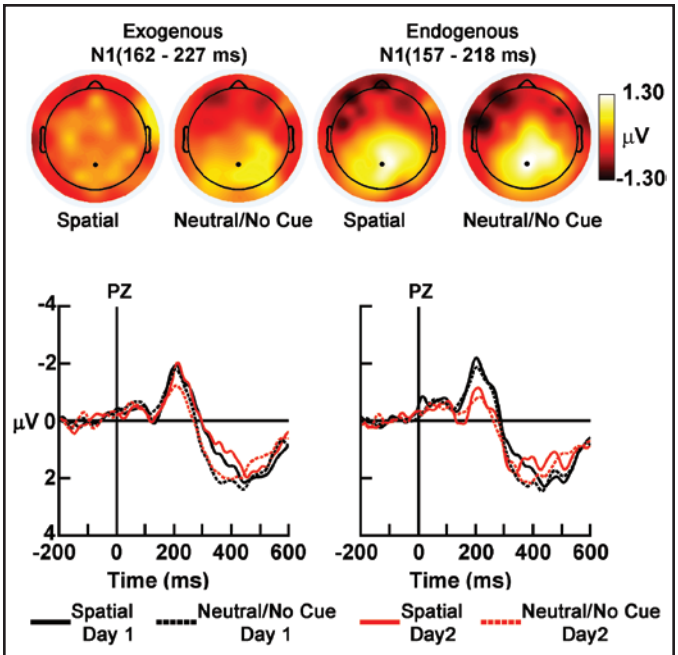

Figure 6—Representative effects of fatigue on the dorsal N1 event-related potential (ERP) component. Waveforms depict representative posterior dorsal grand-average Exogenous (left column) and Endogenous (right column) target-locked ERPs for Day 1 (black lines) and Day 2 (red lines) during the Spatial-Cue condition (solid lines) and collapsed across the Neutral-Cue and No-Cue conditions (dashed lines). Negative polarity is oriented upward. Waveform scalp locations are shown on topographic maps (top row) displaying mean Day 2 – Day 1 differences for the dorsal N1 ERP component averaged over the indicated time intervals (mean component interval across cue condition and day). Light colors indicate positive values; dark colors indicate negative values. Exogenous and Endogenous topographic maps are set to the same scale for ease of comparison.

cal activity associated with sleep deprivation.[12,21] Parietal brain regions are known to be involved in sustained and selective attention[22-24] and form part of a larger frontoparietal attention-control network.[20,48] This frontoparietal network is activated by exogenously and endogenously directed attention to target attributes, but dorsal frontoparietal networks have been shown to be preferentially engaged during endogenous attention shifts, whereas exogenously directed shifts of attention appear to primarily engage right-hemisphere ventral frontoparietal regions.[48] The effects of sleep deprivation on parietal activity may generalize beyond attention deficits, however, because fMRI and positron emission tomography studies have found decreases in parietal cortex activity to be associated with sleep deprivation-related arithmetical performance decrements.[49,50]

The second main finding of the present study was that sleep deprivation decreased the amplitude of the P3 response (450-550 ms) from Day 1 to Day 2. One influential theory[51] models P3 amplitude as depending on 3 factors: stimulus probability, stimulus meaning, and the amount of information transmitted by a stimulus. Since stimulus probability was not manipulated in the present task, these P3 findings suggest an influence of sleep deprivation on processing related to stimulus meaning, transmitted information, or both. Further research is needed to determine the relative impact of sleep deprivation on this processing.

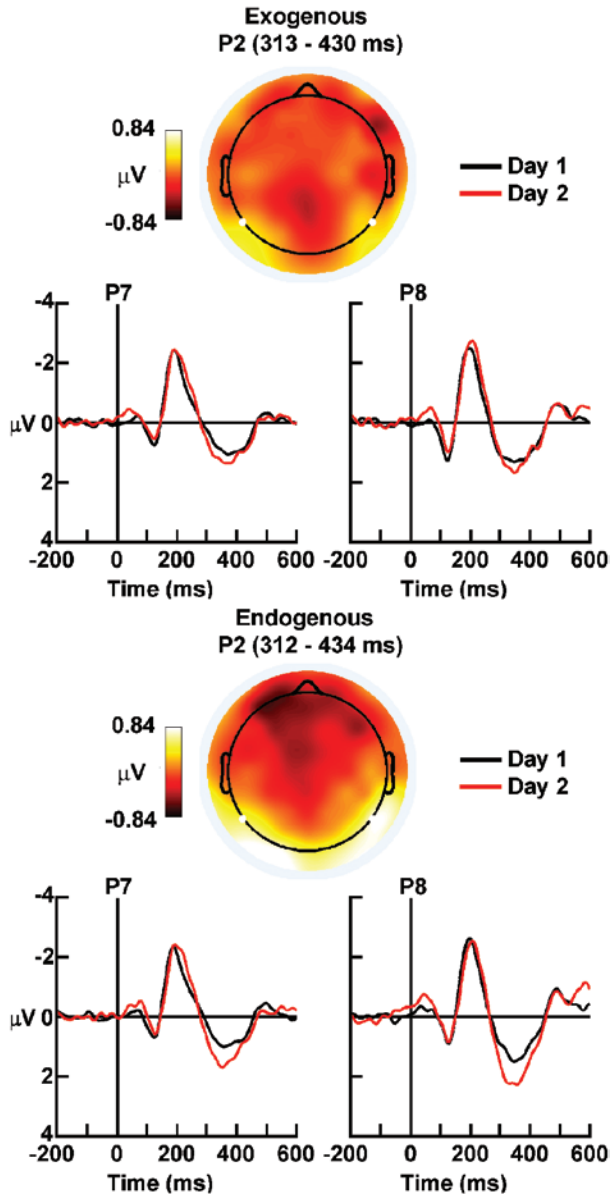

Figure 7—Representative effects of fatigue on the Exogenous (top panel) and Endogenous (bottom panel) Attention Network Test (ANT) P2 ERP components. Waveforms depict grand-average target-locked ERPs for Day 1 (black line) and Day 2 (red line) collapsed across all 3 cue conditions. Negative polarity is oriented upward. Waveform scalp locations are shown on topographic maps (top row) displaying mean Day 2 – Day 1 differences over indicated time intervals (mean component interval across cue condition and day). Light colors indicate positive values; dark colors indicate negative values. Exogenous and Endogenous topographic maps are set to the same scale for ease of comparison.

The finding of fatigue-related decreases in N1 and P3 amplitudes is consistent with the results of previous electrophysiologic studies of the effects of sleep deprivation on attention, cognition, and perception. ERP indexes of vigilant attention are reduced, delayed, or both reduced and delayed,[27,28] as are ERPs elicited during working memory and visuomotor memory tasks.[17,28-31] Auditory evoked potential amplitudes have been shown to be reduced and latencies increased with sleep deprivation.[52,53] The amplitude of the contingent negative variation, a slow potential

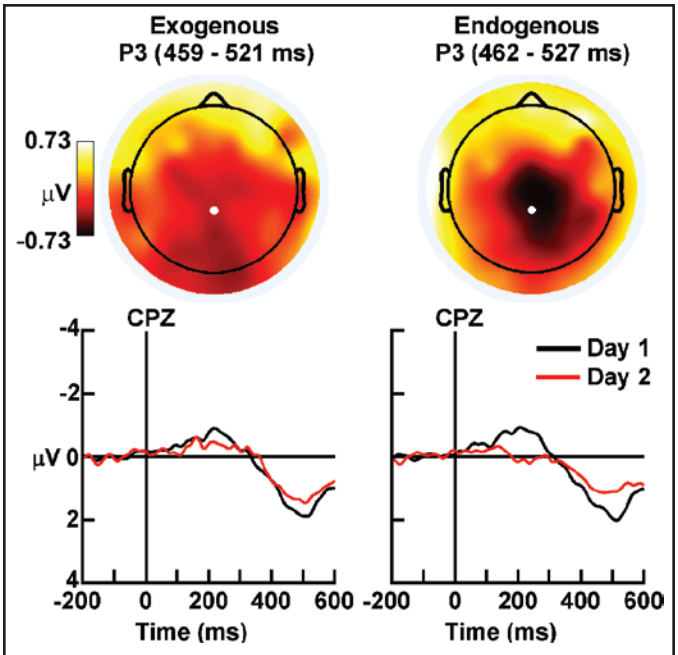

Figure 8—Representative effects of fatigue on the Exogenous and Endogenous Attention Network Test (ANT) P3 component. Waveforms depict grand-average target-locked event-related potentials (ERP) for Day 1 (black line) and Day 2 (red line) collapsed across all 3 cue conditions. Negative polarity is oriented upward. Waveform scalp locations are shown on topographic maps (top row) displaying mean Day 2 – Day 1 differences over indicated time intervals (mean component interval across cue condition and day). Light colors indicate positive values; dark colors indicate negative values. Exogenous and Endogenous topographic maps are set to the same scale for ease of comparison.

indicative of response preparation, is also reduced with sleep deprivation,[52] as are electrodermal indexes of auditory attention-orienting responses.[19] P300 ERP component responses have been found to be reduced and delayed with sleep deprivation,[28,54] whereas the error-related negativity, a response-locked ERP occurring 80 to 100 ms milliseconds following response errors, also exhibits reduced amplitudes with sleep deprivation.[55] ERP reductions have been thought to occur from an increase in across-trial latency variability of the stimulus-evoked neural response, a decrease in the average magnitude of the neural response produced across trials, or both.[30] We should note, however, that certain cognitive tasks may actually lead to increases in cerebral activity as a result of compensatory responses to sleep deprivation[13,56] or a nonspecific increase in cortical activation.[57]

The amplitude of the occipital P2 ERP component increased from Day 1 to Day 2 for both ANTs. This is in contrast with the N1/P3 findings and with the general behavior of ERPs under fatigue observed in previous studies of sleep deprivation. One possible explanation for this apparent discordance is that the occipital P2 observed here reflects activity of neural sources that are affected differently by sleep deprivation than either the N1 or P3. This possibility is supported by the fact that the Day 1 versus Day 2 P2 difference was primarily located over posterior ventral scalp regions rather than dorsal regions, thus indicating a different source distribution for this component than for the other 2 components. An alternative explanation for the present P2 findings is that participants increased the allocation of volitional attention

Table 5—Effects of Sleep Deprivation on the P2 and P3 ERP Components

Day	Exogenous ANT		Endogenous ANT	
	P2	P3	P2	P3
1	1.25 (0.12)	1.46 (0.24)	1.32 (0.13)	1.57 (0.25)
2	1.46 (0.17)	1.16 (0.20)	1.82 (0.20)	1.00 (0.15)

Mean P2 and P3 ERP amplitudes for each Cue condition (all values in μV; SE in parentheses). ERP refers to event-related potential; ANT, Attention Network Test.

resources as a compensatory response[13,56] to sleep deprivation-induced deficits in early-stage attention processing. For example, if participants were engaged in additional cognitive processing, such as response monitoring, to compensate for the detrimental effects of sleep deprivation, then this could account for the observation that sleep deprivation led to slowed RTs and decreased accuracy rates for both ANT. A third alternative explanation is that the present P2 findings were due to practice effects rather than fatigue. In the present study, fresh and fatigue conditions were not counterbalanced across days, and the fresh condition always preceded the fatigue condition. Previous studies have found the P2 component to increase with task repetition.[58,59] Thus, it is possible that the present increase in P2 amplitude from Day 1 to Day 2 was not due to fatigue at all but, instead, was due to practice effects. Further research is required to distinguish between these 3 explanatory possibilities for the present P2 findings.

An additional observation of this study was of small parietal N1 differences between the WP and FH participants. Mean N1d amplitudes were smaller for the Exogenous versus Endogenous ANT for the WP group and larger for the Exogenous versus Endogenous ANT for the FH group. Since we did not assess individual neuropsychological abilities, we can only speculate that these observations may reflect between-group differences in cognitive ability and the effects these differences might have on sustained attention during the cue-target interval. For example, admission to WP is highly selective, so these individuals are likely to be of higher general IQ than the FH participants. Elucidation of this issue is beyond the scope of the present study, but we should point out that there were no between-day group differences, and, thus, the present parietal N1 group differences do not affect our main conclusions regarding the different effects of sleep deprivation on Exogenous and Endogenous attention, particularly with respect to the N1 findings.

Limitations of the Present Study

It is important to note several important limitations of the present study. First, the average number of trials was lower for Day 2 than Day 1, due to the significant presence of fatigue-related decreases in task performance and increases in motion and EOG artifacts on Day 2, factors unavoidable in sleep-deprivation research. Low trial numbers could affect the signal-to-noise ratio of the Day 2 ERPs and potentially result in spurious effects when comparing across Days 1 and 2. Significant attempts were made to reduce the potential problems associated with different signal-to-noise ratios by artifact scoring the data in a conservative manner to eliminate trials laden

with high-frequency noise. In addition, very low-frequency noise induced via fatigue-related head and body movement was reduced via a relatively high-cutoff (0.45-Hz) high-pass filtering of the EEG data (see Methods). The efficacy of our artifact-removal procedures is supported by the fact that we found attention to significantly modulate the target-locked N1 and P2 components, as is typically observed in cued-attention tasks of this kind. Finally, the method used to quantify ERP components, being an area measure (see Methods), tends to mitigate any artifact effects arising from between-condition imbalances in trial numbers.[41]

A second limitation of this study was the use of a sequential design (fresh then fatigued recording sessions) rather than a cross-over design (fresh and fatigued session order counterbalanced among participants), which would better control for practice effects or differences in arousal and motivation that were not related to sleep deprivation across days. Use of a sequential design was necessary for this study due to the schedule restrictions placed upon our subjects, who were all members of the US Army. Participants were monitored continuously during task performance, and every effort was made by the experimenters to keep these highly motivated participants engaged and on task while under the fatigue condition during the Day 2 session. The resulting data are also inconsistent with practice effects because (1) short-term repetition typically leads to faster RTs, improved accuracy rates, or both for a variety of tasks,[58,59] which are behavior patterns opposite to those observed here, and (2) the amplitudes of the N1 and P3 are either unaffected or increase with repetition,[28,54,60] in contrast with the present observations for these ERP components.

A third limitation of this study arises from the choice in the present study to investigate the effect of sleep deprivation on the attention modulation of stimulus processing, a phenomenon that has been at the center of attention research for several decades.[25] Therefore, the present results, centering on behavior and ERP responses to target stimuli, index the effect of sleep deprivation on the consequences of selective attention shifting, ie, the attention modulation of stimulus processing, an indirect index of the effects of sleep deprivation on attention shifting. Investigation of the direct effects of sleep deprivation on attention shifting requires analysis of ERPs evoked in response to the cue stimuli; such an analysis would not be optimal in the present study due to the relatively short cue-target intervals employed (see Methods). Despite this problem, we performed a preliminary analysis of the cue-locked ERPs (see Supplemental Materials available on the *SLEEP* website at www.journalsleep.org). This analysis suggests that decrements in exogenous attention shifts may be driven by changes in the initial processing of sensory transients that capture attention in a bottom-up fashion, whereas sleep deprivation-related decrements in endogenous attention shifts may be driven by changes at later stages of cognitive processing in response to the cues, consistent with the notion that voluntary attention shifts are primarily driven by top-down processes. These findings add to our target ERP findings by also suggesting a greater effect of sleep deprivation on endogenous, relative to exogenous, cuing. It should be stated that, because of the less than optimal methodology, these results are tentative and await further tests in an experiment optimized to examine the cue period.

CONCLUSION

In conclusion, the present study has provided evidence that as little as 24 hours of sleep deprivation affects the earliest stages (N1 ERP component) of endogenously driven attention selection, whereas early stages of exogenously driven attention-selection processes are less affected by sleep deprivation. In addition, later stages of attention-modulated information processing seem to be affected equally for both types of attention selection. These findings have important implications for work operations in which fatigued individuals are required to monitor for and respond to rapid changes in their environment. They suggest that the monitoring and response performance of such sleep-deprived individuals may be most susceptible to the ill-effects of sleep deprivation when voluntary shifts of attention are required to implement and sustain job performance.

FOOTNOTE

Trial numbers were substantially lower on Day 2, as compared with Day 1, as corroborated by a Task × Day × Cue-Type analysis of variance (ANOVA) performed on the trial numbers (main effect of Day, $P < 0.001$). This decrease in trial numbers was due to the significant presence of fatigue-related decreases in task performance and increases in motion and electrooculographic artifacts on Day 2. The question of whether the removal of these trials affected the distribution of cue-target intervals (CTI) differently across conditions was examined with an ANOVA. A Task × Day × Cue-Type × CTI (5 levels ranging from 500 to 1650 ms) ANOVA indicated no significant main effects or interactions involving Task, Day, or Cue Type (P values > 0.13). A main effect of CTI indicated a slightly greater percentage of short- versus long-duration CTI trials (~2% difference, $P < 0.015$), corresponding to a negligible 1-trial difference, on average.

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DISCLOSURE STATEMENT

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