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Research article

24-h sleep deprivation impairs early attentional modulation of neural processing: An event-related brain potential study

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ABSTRACT

Prior research indicates sleep deprivation negatively impacts selective attention, although less is known about the neural bases of these effects. The present study used event-related brain potentials (ERPs) to examine whether the effects of total sleep deprivation could be traced to the earliest stages of sensory processing influenced by selective attention. Participants were randomly assigned either to a regular sleep or 24-h total sleep deprivation condition. Following either sleep deprivation or regular sleep, participants completed a dichotic listening selective attention task while ERPs were acquired. Well-rested participants showed typical attentional modulation of the N1 between 150 and 250 msec, with larger amplitude responses to attended relative to unattended auditory probes. In contrast, these effects were significantly reduced in sleep-deprived participants, who did not show significant effects of selective attention on early neural processing. Similar group differences were observed in the later processing negativity, from 300 to 450 msec. Taken together, these results indicate that 24-h total sleep deprivation can significantly reduce, or eliminate, early effects of selective attention on neural processing.

1. Introduction

Sleep deprivation is a pervasive problem linked to cognitive deficits, increased levels of irritability, and adverse health outcomes [for reviews, see 2,9,24]. For those working or driving while sleep deprived, error-related accidents also become more likely, at great cost both to individuals and society [9,12]. Despite these detrimental consequences, over 20% of adults are estimated to get insufficient sleep on a regular basis [12], and sleep deprivation is especially common among college student populations [26,31].

Experimental studies of sleep deprivation typically examine the effects of total sleep deprivation, defined as 24-h or more without sleep, though repeated nights of partial sleep deprivation have similarly detrimental effects [33]. Across a number of experimental studies, sleep deprivation is causally related to a range of cognitive deficits, though aspects of attention appear particularly vulnerable [1,12,15,24]. Here, we focus specifically on selective attention, or the ability to preferentially process relevant information from the environment while excluding irrelevant distractors. In behavioral studies, sleep deprivation is associated with deficits on a range of tasks requiring selective attention, including filtering irrelevant visual stimuli from a memory array [10], finding embedded figures in complex images [1], and performance on Stroop-like tasks [24]. However, as behavior represents the sum of multiple stages of processing, behavioral studies have not yet been able to specify which stages of processing are affected by sleep deprivation, nor the neural systems most affected.

Using functional magnetic resonance imaging (fMRI), recent studies have begun to address the neural mechanisms underlying the effects of sleep deprivation on selective attention. These studies suggest that such deficits result in part from disruption of fronto-parietal top-down control networks, which following sleep deprivation exhibit reduced task-related neural activity and/or aberrant functional connectivity [3,25]. fMRI studies have also begun to characterize the effects of such disruptions on sensory processing. For example, the visual system parahippocampal place area (PPA), which in well-rested participants responds preferentially to house over face stimuli, fails to show attentional modulation following total sleep deprivation [25] unless the stimuli appear in predictably cued patterns [3]. However, the relatively poor temporal resolution of fMRI renders it difficult to interpret whether the differences in PPA activity reflect feed-forward versus feedback differences in attentional modulation and, more specifically, whether earlier stages of sensory processing are impaired by sleep deprivation.

Despite the temporal limitations of fMRI, Kong and colleagues [23] used fMRI to assess the effects of total sleep deprivation on two at least partially dissociable components of selective attention: distractor

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suppression (reduced processing of task-irrelevant stimuli) and signal enhancement (increased processing of task-relevant stimuli). The authors used chimeric house-face stimuli, which superimposed face and house stimuli into a single visual image. Participants attended either to the face or house stimuli in the chimeric images in separate blocks. Kong and colleagues found that sleep deprivation selectively impaired distractor suppression, with sleep-deprived participants failing to show the same levels of reduced PPA activity to chimeric house-face stimuli when faces were attended (i.e., houses as irrelevant distractor) relative to PPA activity in a baseline passive viewing condition. In contrast, no differences were observed between the sleep-deprived and well-rested participants in the enhancement of PPA activity to the chimeric stimuli when houses were attended relative to the passive viewing baseline. Together, these fMRI studies suggest that sleep deprivation impacts attentional modulation and may be specific to impairing distractor suppression, though it is unclear whether such effects occur during early stages of sensory processing.

In contrast to fMRI, event-related brain potentials (ERPs) have exquisite temporal resolution, making the technique particularly valuable to studies of selective attention [18–20]. In a typical ERP selective attention paradigm, competing streams of visual or auditory stimuli are presented simultaneously, with participants attending selectively to one of the streams. Comparing ERPs to probe stimuli in the attended versus unattended stream provides a relatively pure index of the effects of selective attention on neural processing, while keeping the physical stimuli, task demands, and overall arousal levels constant. Using variations of this paradigm, the effects of selective attention have been documented during the first few hundred milliseconds of neural processing [18–20,34]. For auditory stimuli, these enhancements are most consistently observed on the N1, the first large negative deflection in the ERP waveform emerging approximately 100 msec after stimulus onset. As a relatively early sensory component, early N1 attention modulation is believed to reflect sensory gain control as a largely feed-forward modulation of neural activity [16]. As well, in studies of change-over-time or between-group comparisons, the ERP technique can separately assess differences in distractor suppression versus signal enhancement by comparing neural responses to probe stimuli embedded in either the unattended or attended stream [13,27,30]. Such studies have shown change-over-time or group differences specific to both distractor suppression [13,30] and signal enhancement [27], indicating the technique is sensitive to both types of group difference. Most importantly, the temporal resolution of the ERP technique allows claims of overall differences in the effects of selective attention on neural processing to be isolated in time and traced to early stages of sensory processing.

To date, only a few studies have used ERPs to examine the effects of sleep deprivation on aspects of attention, and none have used a manipulation of selective attention as described above. For example, both Zerouali and colleagues [35] and Cote and colleagues [7] evaluated the N1 elicited by auditory sounds among participants exposed to partial sleep deprivation. While neither study found significant overall effects of partial sleep deprivation on N1 amplitude, these studies did not use a manipulation of selective attention that maintained constant task demands and arousal level across conditions. Instead, the studies compared the N1 across different task conditions, or only in a single condition. Thus, it remains unclear, both from the available fMRI and ERP data, whether sleep deprivation affects early stages of sensory processing that, in well-rested individuals, are the first stages of processing modulated by selective attention.

The goal of the present study was to address these limitations by examining the effects of total sleep deprivation on early indices of selective attention using a well-established ERP measure of selective auditory attention. Adult volunteers completed an ERP dichotic listening selective attention task following random assignment either to regular sleep or 24-h of monitored sleep deprivation. It was predicted that sleep deprivation would result in reduced or absent effects of selective attention on the N1, indicating disruptions in early, feed-forward effects of selective attention on neural processing. We also examined the later processing negativity, believed to index further endogenous processing of attended stimuli, particularly when these stimuli are more difficult to discriminate [14,28]. It was further predicted, based on previous fMRI literature [23], that if deficits could be localized they would be specific to, or larger for, distractor suppression versus signal enhancement.

2. Material and methods

2.1. Participants

The final sample included 35 participants, aged 18–22, including 20 in the regular sleep condition (5 male, mean age 19.8 years) and 15 in the sleep deprivation condition (4 male, mean age 19.7 years), drawn from an original sample of 46 participants. Reasons for exclusion included equipment malfunction (one regular sleep participant) or poor ERP data quality following standard artifact rejection procedures detailed below (7 sleep deprived participants; 3 regular sleep participants). All participants were fluent in English with normal or corrected-to-normal vision. Participants received $20 for participation, regardless of condition. All participants provided informed consent. Procedures were approved by Willamette University's Institutional Review Board.

2.2. Procedure

One to three days prior to testing, participants came to the laboratory for a tour and study overview. During this orientation, participants completed demographic questionnaires and signed a consent form. The day before testing, participants were informed via email if randomly assigned to the sleep deprivation (n = 22) or regular sleep (n = 24) condition. Participants in both conditions were requested to abstain from napping, and also caffeine, alcohol, and psychoactive substances, for the 24-h prior to testing. Participants in the regular sleep condition were instructed to go bed at their usual time and report to the lab at 8 am for testing. Participants in the sleep deprivation condition were told to report to the lab at 10 pm for monitoring, with testing to commence at 8 am the following day. Following previous research [4,5,32], adherence to the sleep deprivation condition was monitored by trained research assistants who ensured participants remained awake throughout the sleep deprivation period. During the sleep deprivation period, participants could engage in non-strenuous activities of their choosing, such as reading, watching TV, or doing homework.

2.3. ERP assessment of selective auditory attention

Participants completed an ERP selective auditory attention task based on a modified version of a classic auditory attention paradigm [17]. Variations of this task have been used to assess auditory selective attention in young children and adults [11,28–30]. Briefly, pairs of 2.5-3.5 min children's stories (one male-narrated, one female-narrated) were recorded and pasted into separate channels of a stereo audio channel. Stories were presented from speakers ~21 inches on either side of the participant, who was instructed to attend to one of the two stories, while ignoring the story in the unattended channel.

In total, participants attended to eight stories (attended narrator counterbalanced within a participant). Attended side, right (R) or left (L), was pseudorandomized with order RLLRRLLR. After each story, participants were asked three basic comprehension questions about the attended story. Due to experimenter error, comprehension questions were not asked of three participants (one regular sleep; two sleep deprived participants).[1] Of participants with comprehension question

[1] All of the ERP results reported in the main text remained if analyses were restricted to participants with comprehension question data.

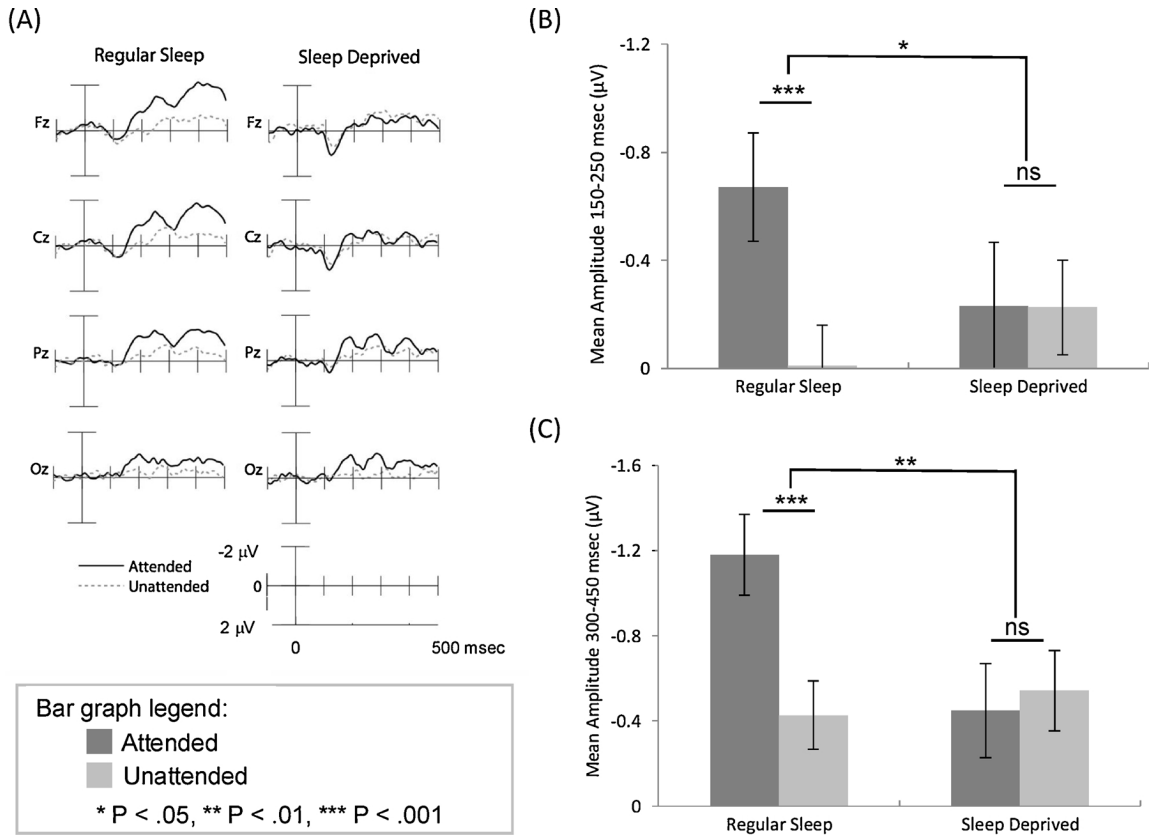

Fig. 1. For each condition and group, panels show (A) grand average evoked potentials at representative midline electrodes, (B) mean amplitude of the N1 from 150 to 250 msec, and (C) mean amplitude of the later processing negativity from 300 to 450 msec.

data available, all answered at least 50% correctly, which in our previous studies was a criterion for study inclusion [13,21,27]. On average, participants in the regular sleep conditions answered 97% of questions correctly (SD = 5%) and in the sleep deprived condition 91% correctly (SD = 13%), which approached a trend-level difference, t(30) = 1.68, $p = 0.10$.

ERPs were time-locked to 100 msec linguistic and nonlinguistic auditory probe sounds superimposed on each narrative. Linguistic probes were 100 msec recordings of a female voice, different from the female narrators, saying the syllable/ba/. Nonlinguistic probes were also 100 msec in length and created by scrambling 4–6 msec of the/ba/ to produce a 'bzz' sound retaining the main acoustic properties of the linguistic probe. Interstimulus interval (ISI) between successive probes was 200 msec, 500 msec, or 1000 msec, with the different ISIs and probe types randomized throughout the session. Analysis focused on the early N1 response to linguistic probe stimuli as this probe type demonstrates the most consistent effects of selective attention in well-rested adults tested with this paradigm [11,22,28], as well as the later processing negativity. Across the eight stories, approximately 430/ba/ probes were presented per condition (Attend/Unattend).

EEG was recorded at 1024 Hz with the Active-Two system, using 32 Ag/AgCl-tipped electrodes embedded in an elastic cap (FP1/2, F7/8, F3/4, FT7/8, Fz, FC5/6, T7/8, C5/6, C3/4, Cz, CP5/6, CP1/2, P3/4, Pz, PO3/4, P7/8, O1/2, Oz) (Biosemi, Amsterdam, Netherlands). Free electrodes were placed on the left and right mastoids, the outer canthus of each eye (to record horizontal eye movement), and below the left eye (to detect blinks). Electrode offsets were maintained at or below 25 μV throughout each session. In cases of single bad electrodes, observed in 1% of the recorded data, the bad channel was excluded from analysis.

Analyses were completed using the EEGLAB toolbox for Matlab [8]. EEG data were down-sampled to 256 Hz, high-pass filtered at 0.1 Hz and low-pass filtered at 40 Hz, then re-referenced to the average of the left and right mastoids. Continuous EEG data for each electrode were divided into epochs beginning 100 msec before each probe event and ending 500 msec after each event. Artifact rejection was done manually, using visual inspection to remove any epochs containing large voltage deviations, blinks, or other muscle movement. All participants included in the final analysis had at least 150 trials/condition available for analysis. The mean number of trials for the regular sleep group following artifact correction was 340 (SD = 56) and 342 (SD = 55) for the attended and unattended conditions, respectively, and for the sleep deprived group, 291 (SD = 61) and 288 (SD = 59) for the attended and unattended conditions, respectively. This difference between groups was statistically significant for both attended trials, $t(33) = 2.45$, $p < .05$, and unattended trials, $t(33) = 2.77$, $p < .01$. To ensure the different number of trials did not drive between-group ERP differences, all analyses reported below were also conducted using a data set in which the number of trials between groups was artificially matched by removing a random subset of clean ERP trials from each regular sleep participant to equate the number of trials in each group, with no change in the pattern of results reported below. As well, given there were more participants in the regular sleep group, supplemental analyses were conducted restricted to a random subset of 15 control participants, also with no change in the pattern of results reported below.

2.4. Data analysis

ERP analyses focused on the N1 and the later processing negativity, with mean amplitudes calculated from 150 to 250 and 300–450 msec post-stimulus onset, respectively. These time windows were based on visual inspection of the data and prior studies of adults with this paradigm [6,11,22,28]. Mean amplitudes were assessed over a 10-electrode fronto-central region of interest (F3/4, FC5/6, C5/6, C3/4, Fz, Cz) and subjected to a 2 × 2 mixed design ANOVA including factors

Sleep Condition (regular/deprived, between subjects) and Attention (attended/unattended, within subjects), with follow-up step down *t*-tests as appropriate.

3. Results

Fig. 1 presents the grand average ERPs in each group at midline electrodes, as well as mean amplitude for each condition and group for the N1 and processing negativities. As shown in Fig. 1, whereas the regular sleep group appeared to show robust effects of selective attention in the N1 and processing negativity time windows, these effects were not apparent in the sleep deprived group. Statistical analyses confirmed these visual observations.

Analysis of the N1 indicated a significant main effect of Attention, $F(1,33) = 4.91$, $p < .05$, $\eta_p^2 = 0.13$ (Attended > Unattended). The main effect of Sleep Condition was not significant, $F(1,33) = 0.24$, $p = .63$, $\eta_p^2 = 0.01$. Critical to the main hypothesis of the study, a significant Attention x Sleep Condition interaction indicated the effect of attention on early neural processing differed between the regular and sleep-deprived groups, $F(1,33) = 4.83$, $p < .05$, $\eta_p^2 = 0.13$. Step-down analyses examined the attention effect separately in each of the participant groups. Participants in the regular sleep condition showed a robust effect of selective attention on neural processing, $t(19) = 4.29$, $p < .001$, Cohen's $d = +0.96$. In contrast, participants randomly assigned to the sleep deprivation condition did not show significant effects of attention on neural processing, $t(14) = -0.01$, $p = .99$, Cohen's $d = +0.00$. To examine whether group differences could be isolated to signal enhancement versus distractor suppression, the two groups were directly compared in the attended and unattended condition. While the effect size for each comparison was moderate, the analysis failed to localize group differences between the sleep deprived and regular sleep groups to either process: unattended probes, $t(33) = 0.91$, $p = .36$, Cohen's $d = -0.32$, attended probes, $t(33) = -1.42$, $p = .17$, Cohen's $d = +0.48$.

Analysis of the later processing negativity, from 300 to 450 msec, also indicated a significant main effect of Attention, $F(1,33) = 8.36$, $p < 0.01$, $\eta_p^2 = 0.20$ (Attended > Unattended) but not of Sleep Condition, $F(1,33) = 1.59$, $p = 0.21$, $\eta_p^2 = 0.05$. A significant Attention x Sleep Condition interaction indicated the effect of attention differed between the regular and sleep-deprived groups, $F(1,33) = 13.87$, $p = .001$, $\eta_p^2 = 0.30$. Step-down analyses indicated a significant effect of attention in the regular sleep group, $t(19) = -4.71$, $p < .001$, Cohen's $d = -1.04$ but not in the deprived sleep group, $t(14) = 0.62$, $p = .62$, Cohen's $d = +0.15$. Follow-up analyses indicated these group differences were specific to reduced signal enhancement in the sleep deprived group relative to the regular sleep group: attended probes, $t(33) = -2.52$, $p < .05$, Cohen's $d = +0.86$; unattended probes, $t(33) = 0.46$, $p = .64$, Cohen's $d = -0.15$.

4. Discussion

The present study used event-related brain potentials to examine the effects of 24-h sleep deprivation on early neural indices of auditory selective attention. Findings indicated that sleep deprivation resulted in significantly reduced effects of selective attention on the auditory N1, from 150 to 250 msec after stimulus onset, though these effects could not be localized to differences specific to either signal enhancement or distractor suppression. Sleep deprivation also led to significant reductions in the later processing negativity, from 300 to 450 msec, which were specific to reduced signal enhancement.

These findings extend prior neuroimaging research by showing that sleep deprivation can significantly reduce, or eliminate, the effects of selective attention on the earliest stages of neural processing known to be affected by selective attention. Whereas previous fMRI studies indicated disruptions in attentional modulation of the parahippocampal place area [23,25], the temporal resolution of fMRI did not allow conclusions to be drawn about whether this difference reflected differences in feed-forward versus feed-back attentional modulation. Here, we took advantage of the exquisite temporal resolution of ERPs to examine whether sleep deprivation impaired attentional modulation during the first few hundred milliseconds of processing. The impairments observed in N1 attentional modulation, believed to index feed-forward sensory gain [16], indicate that total sleep deprivation impacts very early stages of sensory processing and continue to be observed during the later processing negativity. As well, the present study's focus on auditory selective attention indicates that impairments following sleep deprivation extend beyond the visual modality to auditory attention.

In contrast to a prior fMRI study [23], which suggested the sleep deprivation specifically impairs distractor suppression, the present study could not localize group differences in the early N1 to either distractor suppression or signal enhancement mechanisms, whereas differences in the later processing negativity were specific to reduced signal enhancment. As the effect sizes for between-group comparisons of the early N1 for both distractor suppression and signal enhancement were both moderate in magnitude, it would appear unlikely that this difference across studies can be accounted for by statistical power. As well, previous studies using this paradigm have demonstrated sensitivity in differences specific to both distractor suppression [13,30] and signal enhancement [27]. One possibility is that the effects of sleep deprivation differ for earlier versus later neural processing. At early stages of processing, such as those indexed by the early N1, effects may be broadly distributed across both signal enhancement and distractor suppression, and for the processing negativity, believed to index further endogenous processing of selected stimuli [14,28], to signal enhancement. In contrast, during later stages of processing and/or those that might include feed-back attention modulation, such as those indexed by PPA modulation, differences may become larger for or specific to distractor suppression. However, given the range of differences across studies, including stage of processing (early N1/processing negativity versus later PPA activity), stimulus modality (auditory versus visual), and temporal resolution of methodology (milliseconds for ERPs versus seconds for fMRI), future research is needed to evaluate these differences further.

The present findings help clarify the neural underpinnings of performance decrements following sleep deprivation. Previous behavioral studies indicate that sleep deprivation negatively affects a range of cognitive tasks [2,9,24], with those requiring selective attention particularly vulnerable [1,10,12,15,24]. The present findings suggest that for tasks requiring selective attention, sleep deprivation impairs neural modulation indicating successful early selection of the attended stream, likely emerging from the combined influence of diminished distractor suppression and signal enhancement, with later endogenous processing differences emerging from reduced signal enhancement. This finding has an interesting parallel in a study of visual working memory, in which sleep deprivation only affected performance on the task when there were visual distractors present that needed to be actively filtered [10]. In contrast, sleep deprivation did not affect working memory when there were no co-present, competing stimuli. This suggests that performance decrements for tasks that require selective attention emerge from differences not only in later stages of processing (e.g., response selection or execution), but also affects aspects of selecting the relevant input stream, and processing it preferentially while reducing the processing of competing information. From a human factors standpoint, these findings have implications, where modifications to the environment are possible that could increase discriminability of signals. More broadly, this research suggests that under conditions of sleep deprivation, it may be more difficult to discriminate relevant from irrelevant streams, increasing the need for aids or methods to increase signal salience and discriminability. Future studies can identify further differences in other stages of processing and/or how manipulating aspects of task or stimulus could mitigate the negative impacts of sleep

deprivation.

The present study included several limitations that suggest directions for future research. First, the study used a between-subjects design, limiting statistical power. A within-subjects design would increase power and also allow examination of intra-subject variability in the effects of sleep deprivation. Second, the study focused exclusively on auditory selective attention. Future studies could include both visual and auditory paradigms to examine the degree to which impairments following sleep deprivation differ, either in magnitude or in stage of processing, across the two modalities. Finally, future research could include sensitive behavioral assays of selective attention, permitting potential links between neural differences in attentional modulation and performance on specific tasks.

Taken together, the present study indicates that even very early stages of neural processing fail to show attention modulation following total sleep deprivation. At the earliest stages of processing, these differences cannot be localized to deficits in signal enhancement versus distractor suppression, though reduced attentional modulation of later endogenous processing indexed by the processing negativity reflects reduced signal enhancement of attended information. Combined with the robust literature on the behavioral impacts of sleep deprivation on a range of cognitive tasks, these findings suggest that efforts to mitigate the effects of sleep deprivation on human performance must account not only for later stages of decision making, but also for differences in early sensory processing that are affected by sleep deprivation.

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