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Performance monitoring following total sleep deprivation: Effects of task type and error rate

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ABSTRACT

There is a need to understand the neural basis of performance deficits that result from sleep deprivation. Performance monitoring tasks generate response-locked event-related potentials (ERPs), generated from the anterior cingulate cortex (ACC) located in the medial surface of the frontal lobe that reflect error processing. The outcome of previous research on performance monitoring during sleepiness has been mixed. The purpose of this study was to evaluate performance monitoring in a controlled study of experimental sleep deprivation using a traditional Flanker task, and to broaden this examination using a response inhibition task. Forty-nine young adults (24 male) were randomly assigned to a total sleep deprivation or rested control group. The sleep deprivation group was slower on the Flanker task and less accurate on a Go/NoGo task compared to controls. General attentional impairments were evident in stimulus-locked ERPs for the sleep deprived group: P300 was delayed on Flanker trials and smaller to Go-stimuli. Further, N2 was smaller to NoGo stimuli, and the response-locked ERN was smaller on both tasks, reflecting neurocognitive impairment during performance monitoring. In the Flanker task, higher error rate was associated with smaller ERN amplitudes for both groups. Examination of ERN amplitude over time showed that it attenuated in the rested control group as error rate increased, but such habituation was not apparent in the sleep deprived group. Poor performing sleep deprived individuals had a larger Pe response than controls, possibly indicating perseveration of errors. These data provide insight into the neural underpinnings of performance failure during sleepiness and have implications for workplace and driving safety.

1. Introduction

There is a need to understand the neurophysiolgical mechanisms of performance deficits that result from sleep loss. Previous research shows that frontal brain regions and attentional networks are disrupted during sleep deprivation (Portas et al., 1998; Wu et al., 1991; Thomas et al., 2000; Drummond and Brown, 2001; Harrison and Horne (2000). Specifically, lapses in attention and mood disruptions are some of the most robust effects of sleep loss (See Durmer and Dinges, 2005 for review). Failure in top-down prefrontal control during sleep loss has also been suggested to result in inappropriate emotional responses due to a disconnect between the medial prefrontal cortex (PFC) and amygdala (Yoo et al., 2007). Performance monitoring and error processing are known to involve the anterior cingulate cortex (ACC) region of the PFC (van Veen and Carter, 2002). Thus, investigating the effects of sleep deprivation on performance monitoring allows examination of the effects of sleep loss on localized brain regions. A small number of studies have been carried out to investigate the effects of sleep deprivation on performance monitoring; the approaches have been varied and the results equivocal. We therefore sought to examine the effects of total sleep deprivation on performance monitoring using two different tasks under controlled experimental conditions. We examined behavior and brain responses during a traditional error monitoring Flanker task where errors involved selecting the wrong response option, and a Go–NoGo task where errors involved failure to withhold or inhibit a response.

In performance monitoring tasks that involve high conflict and error detection, the ACC located on the medial surface of the frontal lobe (van Veen and Carter, 2002) becomes activated. The ACC is divided into two subdivisions: the dorsal cognitive (dACC) and ventral affective (vACC) subdivision (Kanske and Kotz, 2011). The error-related negativity (ERN) is thought to be generated in the dACC, whereas the source of the error positivity (Pe) is likely a composite of earlier components from the ACC and later ones from parietal regions which reflect signal detection processes (van Veen and Carter, 2002; Ridderinkhof et al, 2009). The ERN is largest over frontal and central midline electrodes and is represented by a sharp negative deflection in the EEG with a magnitude of approximately 10 μV usually peaking 100 ms after erroneous responses (Falkenstein et al., 1991; Gehring et al., 1993). For the Pe component, an earlier frontal peak, as well as a parietal peak around 300 ms has been observed (van Veen and Carter, 2002; Overbeek et al, 2005). The functional role of the ERN has been suggested as serving an error detection and compensation process (Gehring et al., 1993) or a conflict

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detection process (Botvinick et al., 1999). Others have suggested a role in a dopaminergic negative reinforcement learning system as a dopamine signal is able to modify and shape future performance based on endogenous feedback (Holroyd and Coles, 2002). In general, the ERN represents an attentional control process that is engaged whenever one needs to shift response strategy (e.g., following an error detection). The Pe is thought to reflect an independent process of error processing distinct from the ERN (Overbeek et al, 2005). It has been hypothesized to index conscious error recognition particularly for events of motivational significance, response strategy, and emotional evaluation of error (Falkenstein, 2004; Ridderinkhof et al, 2009).

The ERN and Pe components are typically investigated using a Flanker task; the participant is asked to respond to the center letter (H or S) in a string of five letters where the flanking letters are either congruent (e.g., SSSSS) or incongruent (e.g., HHSHH) with the target (Erikson and Erikson, 1974). To date, only a small number of studies have investigated the impact of sleep loss on error monitoring. The results of these studies have been mixed, possibly due to small sample sizes, and varying levels of sleep loss investigated and type of tasks employed. A few studies have found reduced accuracy and response time (RT) (Tsai et al., 2005; Drummond et al., 2006; Hsieh et al., 2007, 2010; Anderson and Platten, 2011; Cain et al., 2011) on error monitoring and response inhibition tasks, whereas others have not (Murphy et al., 2006; Schapkin et al., 2006; Asaoka et al., 2010; Cain et al., 2011). Scheffers et al. (1999), Murphy et al. (2006) and Tsai et al. (2005) reported evidence for impaired remedial behavior after sleep deprivation (i.e., failure to adjust accuracy or RT on the trial immediately following an error). Most of these studies have observed reduced ERN amplitudes (Scheffers et al., 1999; Tsai et al., 2005; Hsieh et al., 2007, 2010), but some studies have failed to find support for an effect of sleepiness (Asaoka et al., 2010; Murphy et al., 2006). The Pe appears attenuated when individuals are both sleepy (Asaoka et al., 2010; Murphy et al., 2006), and more extremely sleep deprived (Tsai et al., 2005; Hsieh et al., 2010), but has not always been reported in previous studies.

Sleep deprivation has been shown to increase impulsive behaviors on response inhibition tasks (Drummond et al., 2006). Response inhibition is usually measured with a Go/NoGo paradigm; the participant is instructed to make a motor execution to a target stimulus (Go) and to inhibit motor responses to an equiprobable or rarely occurring NoGo stimulus. Go stimuli elicit a classic parietal P300 (Donchin and Coles, 1988), while NoGo stimuli elicit large anterior driven N2 and P300 components generated from the ACC that reflect conflict detection and response inhibition respectively (Fallgatter et al., 2002). Although Flanker tasks are more frequently employed to measure performance monitoring, Go/NoGo tasks produce similar error rates and are thus ideal to examine response-locked processes such as the ERN and Pe. Individuals subjected to noise-induced sleep disturbance have shown reduced N2 and P300 components on response inhibition tasks (Schapkin et al., 2006; Breimhorst et al., 2008); however, to date no studies have used response-locked ERPs to investigate the underlying neural correlates of response inhibition following total sleep deprivation.

The purpose of the current study was to investigate the effects of sleep deprivation on performance monitoring. Based on previous research and evidence that sleep deprivation impairs frontal lobe function, it was expected that sleep deprived participants would show deficits in behavioral and electrophysiological measures on two different performance monitoring tasks.

It was expected that all participants would make more errors to incongruent stimuli and NoGo inhibitions consistent with the goals of the Flanker and Go–no-Go tasks respectively. Behaviorally, it was expected that sleep deprived participants would be less accurate and slower overall, consistent with previous research. Group differences were also expected for the stimulus-locked N2 and P300 components reflecting deficits in inhibitory and attentional resources respectively. The major hypothesis investigated in the present study was with respect to error processing in particular. The response-locked ERN and Pe ERP components following task error trials were expected to be attenuated in sleep deprived individuals compared to rested controls, showing frontally-mediated deficits in the performance monitoring system.

2. Method

2.1. Participants

Participants were recruited through advertisements in the Psychology Department and classroom presentations. Eligible candidates must have been between the ages of 18 and 30, healthy (free from medications), good sleepers, right-handed, and free of any history of psychiatric conditions and traumatic brain injury. Sixty-eight individuals initially met inclusion criteria. Four were removed after polysomnography (PSG) screening for having either poor sleep efficiency (2) or periodic limb movements (2). Eight participants were withdrawn during the experimental protocol due to: lack of interest (2), technical malfunctions (1), tolerance to sleep deprivation (2), personal scheduling conflicts (1) and, poor electrophysiological signal quality (2). Seven individuals were not able to be scheduled to participate. Thus, the final sample included 49 participants (Control Group (Men: n = 13; M_{age} = 19.23, SD = 1.48; Women: n = 12; M_{age} = 19.25, SD = 1.29) and Sleep Deprivation (Men: n = 11; M_{age} = 20.55, SD = 2.21; Women: n = 13; M_{age} = 19.15, SD = 1.57)).

2.2. Procedures

All study procedures were cleared by the local Research Ethics Board. Study completion entailed a $110 honorarium or $90 plus course credit. Volunteers initially participated in a telephone interview to screen for inclusion criteria. Suitable candidates then completed on-line questionnaires and were scheduled for PSG screening. PSG records were scored according to standard procedures (Rechtschaffen and Kales, 1968) and evaluated for sleep disordered breathing and periodic limb movement.

Eligible participants were enrolled in the main study which included two consecutive nights and one day in the sleep laboratory. All participants arrived at 21:00 on a Thursday for a Baseline night of sleep (23:00–07:00). They returned at 21:00 on Friday for the Experimental night when they were randomly assigned to either a control group who were permitted an 8-h sleep opportunity (23:00–07:00) or a sleep deprivation group who remained awake for approximately 34 h. Sleep deprived participants were continuously supervised by research assistants; they passed the time watching movies and playing games. On both nights, participants practiced performance tasks and completed pre-sleep questionnaires. Participants who slept were awoken at 07:00, asked to complete a post-sleep questionnaire and then provided breakfast. The Positive and Negative Affect Scale (PANAS; Watson et al., 1988), Stanford Sleepiness Scale (SSS; Hoddes et al., 1973) and mood visual analog scales (VAS) were administered every hour while awake.

Electrode caps were applied at 09:30 on Saturday and Performance Assessment Batteries (PABs) were administered at 10:30–12:00 (to assess frontal lobe function) and at 14:00–15:30 (to assess emotion processing; data not reported here). The morning PAB began with subjective measures of sleepiness and mood, a resting EEG recording, and a reaction time task. Tasks were then administered in the following fixed order: Flanker (15 min), Novelty Processing (15 min), Response Inhibition (15 min), and 2-back working memory (10 min).

In the Flanker task, stimuli were presented using letters "H" and "S" in gray font on a black backdrop centered on the computer monitor. Participants were instructed to respond to the central target letter which was flanked by an array of four other letters. Speed and accuracy were stressed equally to participants. Congruent trials had flanking congruent letters that matched the central target whereas incongruent trials had a target letter flanked by incongruent letters. The Flanker task contained

600 trials evenly divided into 4 blocks with breaks. One third of trials were congruent (HHHHH or SSSSS) and two thirds were incongruent (HHSHH, SSHSS). The stimulus duration time was 200 ms; inter-trial interval varied randomly between 1200 and 1700 ms. Participants used a response pad labeled with the two target letter options (H and S); they were instructed to use both hands to respond, using the index fingers only. Response options were counterbalanced between bedrooms (S and H).

In Go/NoGo task, "X" and "+"stimuli were presented in gray on a black backdrop centered on the computer monitor. Participants were instructed to respond to the target "X" and were to inhibit responses to the "+". Participants were encouraged to respond as quickly as possible but remain as accurate as possible. The task contained 600 trials which were divided evenly into 4 blocks with breaks. Eighty percent of the trials were the target Go "X" whereas twenty percent were NoGo "+" inhibition trials. Stimulus duration was 50 ms and inter-trial interval varied at random between 1000 and 2000 ms. Participants used the keyboard and were instructed to respond with the zero key on the keypad and using the right index finger only.

All electrophysiological signals during PSG screening, Baseline and Experimental nights, and waking EEG were recorded using Neuroscan Synamps II amplifiers and v4.5 software (Neuroscan, Inc., El Paso). Electrodes recorded electrocardiography (EKG), electromyography (EMG; submental), electrooculography (EOG; outer canthus of each eye), and electroencephalography (EEG; O1, O2, C3 and C4 for sleep recordings; a Neuroscan 64-channel Ag/AgCl Quikcap with a central site reference between Cz and CPz was used for waking recordings). Impedances for PSG and waking data were maintained at 10 kΩ or less. Prior to analysis of ERPs, all EEG recordings were re-referenced offline to an average of mastoid sites, A1 and A2. Hardware filters used to record EEG were DC to 100 Hz. Stimuli were delivered using STIM software (Neuroscan, Inc., El Paso) and presented on a computer screen in the participant's private bedroom.

2.3. Data analysis

Flanker and Go/NoGo ERP individual averages were bandpass filtered at 1–30 Hz (6 dB/octave) for stimulus-locked, and 1–20 Hz (6 dB/octave) for response-locked ERPs. Response-locked ERPs were epoched from −800 ms to 600 ms. The baseline used for the response-locked ERPs utilized a sweep correction range of −800 ms to −600 ms to avoid stimulus effects which have been shown to influence the response-locked ERP (Verleger et al., 2005; Murphy et al., 2006). In the Flanker task, individual averages for error responses were calculated by combining all error trials regardless of congruency.

A mean amplitude peak detection method was used to measure the Pe component generated to correct and error trials in Flanker and Go/NoGo tasks as clearly defined peaks are not often observed. The Pe was measured between 200 and 400 ms for the Flanker task (as per Nieuwenhuis et al., 2001), and between 50 to 400 ms for the Go/NoGo task (where it was apparent in the grand average). Although the entire 64-channel montage was explored, only electrode sites FCz and Pz are reported because these were the sites where both stimulus-locked and response-locked ERPs were largest.

One of the hallmarks of sleep deprivation is periodic lapses in responses (i.e., omissions due to micro sleeps or reduced attention). If a lapse in attention were to occur surrounding a NoGo trial, the failure to respond could mistakenly be classified as a correct inhibition. Therefore, due to the confounding nature of lapses in NoGo accuracy in the Go/NoGo task after sleep deprivation, criteria were set for determining the validity of a correct NoGo inhibition. For a NoGo trial to be considered valid, participants must have responded correctly to the preceding and following Go trials surrounding a NoGo trial.

Table 1
Reaction time data from simple RT task.

	N	Control		N	Sleep deprivation		*df*	*t*	*p*
		M	*SD*		*M*	*SD*			
Mean RT	24	309.72	35.43	24	404.04	89.30	46	−4.81	<.001
SD RT	24	55.86	19.96	24	108.49	39.65	46	−5.81	<.001
COV RT	24	0.18	0.05	24	0.26	0.08	46	−4.38	<.001
Mean 10% fast	24	238.31	28.42	24	289.36	68.47	46	−3.37	.002
Mean 10% slow	24	426.79	76.16	24	647.85	161.63	46	5.12	<.001
Missed trials	24	0.67	1.01	24	8.46	12.68	46	−3.00	.006
No. of lapses	24	1.00	1.50	24	8.29	8.56	46	−4.11	<.001

Note: Lapses are RTs > 500 ms; COV RT = coefficient of RT variation; statistics on 10% slowest RTs were calculated on the inverse data to account for violations to normality.

3. Results

3.1. Validation of experimental sleep deprivation

In order to verify that groups had comparable sleep on the baseline night, sleep architecture was compared between groups. On the Baseline night, groups did not differ on total sleep time, sleep efficiency or percent time in each stage of sleep. Sleep efficiency was 93.07% ($SD = 5.12$) for the control group and 93.40% ($SD = 4.20$) for sleep deprivation group on the Baseline night. Further, control participants obtained an overall sleep efficiency of 95.00% ($SD = 3.00$) on the Experimental night.

Subjective sleepiness, fatigue and visual analog mood scales were surveyed in pre-and post-sleep questionnaires. Group (Control, Sleep Deprivation) by Time (Thursday evening, Friday morning, Friday evening, Saturday morning) mixed-model ANOVAs were run to investigate the effects of sleep deprivation. Significant Group by Time interactions were found for subjective sleepiness, $F(3, 129) = 8.74$, $p < .001$, $\eta^2 = .17$, fatigue, $F(3, 141) = 10.7$, $p < .001$, $\eta^2 = .19$, and for the mood scales of calm/irritable, $F(3, 129) = 5.36$, $p = .004$, $\eta^2 = .11$, energetic/sluggish, $F(3, 129) = 2.94$, $p = .036$, $\eta^2 = .06$, and relaxed/tense, $F(3, 129) = 3.99$, $p = .018$, $\eta^2 = .09$. In comparison to controls, the sleep deprived group reported more subjective sleepiness, $t(46) = -3.94$, $p < .001$, fatigue, $t(47) = -3.71$, $p = .001$, irritability, $t(33) = -3.47$, $p = .001$, sluggishness, $t(47) = -2.87$, $p = .006$, and tenseness, $t(31) = -2.79$, $p = .009$, on Saturday at 07:00 (i.e., 24 h awake for sleep deprived group), but not at other times. Also to verify the expected effects of the sleep deprivation manipulation, groups were compared on reaction time from a simple RT task administered during the morning PAB. As expected, the sleep deprivation group was significantly slower and more variable in RT performance. See Table 1 for descriptive statistics and t-tests.

3.2. Error monitoring: behavioral performance on the Flanker task

Group (Control, Sleep Deprivation) by Stimulus Type (congruent, incongruent) mixed-model ANOVAs were conducted to assess behavioral accuracy, coefficient of RT variation and omission rate across groups. A Stimulus Type main effect was found for response accuracy, $F(1, 48) = 141.8$, $p < .001$, $\eta^2 = .75$; all participants made more errors to incongruent trials ($M = 15.14\%$, $SD = 7.92$) than congruent trials ($M = 6.61\%$, $SD = 5.16$). A Stimulus Type main effect was also found for omission rate, $F(1, 48) = 5.69$, $p = .021$; all participants missed more incongruent trials ($M = 6.23\%$, $SD = 6.50$) than congruent trials ($M = 5.51$, $SD = 5.81$). A Group main effect was found for coefficient of RT variation, $F(1, 48) = 7.69$, $p = .008$, $\eta^2 = .14$; the sleep deprived group ($M = 0.24$, $SD = .04$) was more variable than controls ($M = 0.21$, $SD = .04$), collapsed across congruent and incongruent trials.

To assess behavioral RT during the Flanker task, a Group (Control, Sleep Deprivation) by Stimulus Type (congruent, incongruent) by Accuracy (correct, error) mixed-model ANOVA was conducted. There was a significant Stimulus Type by Accuracy interaction, $F(1, 48) = 31.8$, $p < .001$, $\eta^2 = .40$. When participants responded correctly, they were significantly slower to incongruent trials ($M = 404.51$ ms, $SD = 44.46$) compared to congruent trials ($M = 372.24$ ms, $SD = 42.34$); $t(49) = -14.0$, $p < .001$.

Table 2
Effects sleep deprivation on accuracy, omission rate and reaction time in the Flanker task.

	Control				Sleep deprivation			
	Congruent		Incongruent		Congruent		Incongruent	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Accuracy (%)	94.55	5.01	86.17	6.97	92.33	5.01	83.65	8.67
Omissions (%)	4.17	4.90	4.95	6.13	6.75	6.38	7.41	6.71
RT correct (ms)	355.71	35.93	385.20	36.86	387.50	42.68	347.08	63.33
RT error (ms)	325.59	63.74	317.29	36.40	422.34	44.00	344.02	46.40
COV RT	.21	.03	.21	.04	.24	.04	.24	.06

Note: *M* = means; *SD* = standard deviation; COV RT = coefficient of RT variation; N = 24, for control and N = 26 for sleep deprived group.

There was a main effect of Group for RT, $F(1, 48) = 6.16$, $p = .017$, $\eta^2 = .11$; sleep deprived participants ($M = 375.24$ ms, $SD = 41.68$) responded significantly slower than controls ($M = 345.95$ ms, $SD = 41.68$), collapsed across Stimulus Type and Accuracy. See Table 2 for Flanker behavioral performance data.

3.3. Error monitoring: post-error behavioral adjustments

Reaction times were taken on correct trials following a correct response as well as trials following error responses and submitted to a Group (Control, Sleep Deprivation) by Response Type (Correct, Error) mixed model ANOVA. A main effect of Group was found, $F(1, 48) = 5.98$, $p = .018$, $\eta^2 = .11$. The sleep deprived group ($M = 408.92$ ms, $SD = 41.21$) was significantly slower than controls ($M = 380.39$ ms, $SD = 41.21$) for all response types. There was no main effect of Response Type or an interaction to suggest post-error slowing in either group. A trend was found suggesting that the sleep deprived group ($M = 86.58\%$, $SD = 8.95$) was less accurate than controls ($M = 90.87\%$, $SD = 8.57$) on trials following an incorrect response, $t(48) = 1.73$, $p = .09$.

3.4. Error monitoring: stimulus-locked N2 and P300 on the Flanker task

Group (Control, Sleep Deprivation) by Stimulus type (Congruent, Incongruent) mixed-model ANOVAs were run to investigate the differences in N2 amplitude and latency at site FCz, and P300 amplitude and latency at electrode sites FCz and Pz. A Stimulus Type main effect of N2 amplitude, $F(1, 47) = 8.87$, $p = .005$, $\eta^2 = .16$, and N2 latency, $F(1, 46) = 10.45$, $p = .002$, $\eta^2 = .19$, was observed such that all participants elicited a larger but delayed N2 to incongruent stimuli compared to congruent stimuli. A Stimulus Type main effect was observed for P300 latency at both FCz, $F(1, 46) = 23.7$, $p < .001$, $\eta^2 = .34$, and Pz, $F(1, 47) = 5.42$, $p = .024$, $\eta^2 = .10$, such that incongruent trials (FCz: $M = 382.98$ ms, $SD = 31.73$, Pz: $M = 373.86$ ms, $SD = 54.92$) were delayed compared to congruent trials (FCz: $M = 362.75$ ms, $SD = 28.28$, Pz: $M = 355.86$ ms, $SD = 44.81$). There was also a Group main effect: sleep deprived participants ($M = 383.92$ ms, $SD = 37.48$) had a delayed P300 compared to controls ($M = 344.29$ ms, $SD = 38.26$) at electrode site Pz for both trial types combined, $F(1, 47) = 13.14$, $p = .001$, $\eta^2 = .22$. See Table 3 for Flanker stimulus-locked ERPs.

3.5. Error monitoring: response-locked ERN and Pe on the Flanker task

In a preliminary *t*-test to compare ERN amplitude, there was no support for the expected group difference between controls ($M = -7.07$ μV, $SD = 4.24$) and sleep deprived participants ($M = -5.59$ μV, $SD = 3.32$); $t(47) = -1.36$, $p = .180$. Given that error rates differed between groups, there was an unequal number of trials in the aforementioned ERN average waveforms (Controls: M = 36.7, SD = 22.7, range 5–92 trials); Sleep Deprived: M = 63.0, SD = 20.8, range 6–94 trials). In addition, there was a negative correlation between accuracy and ERN amplitude for both controls ($r = -.53$, n = 24, $p = .008$) and sleep deprived participants ($r = -.46$, n = 25, $p = .021$). This correlation indicated that those who made more errors on the Flanker task had smaller ERN amplitudes.

Given the relationship between ERN amplitude and accuracy and since sleep deprived participants were expected to systematically make more errors, follow up analyses were run to investigate the hypothesized effect of sleep deprivation on the ERN using a comparable number of trials for each group in the ERP. Specifically, a subsample (n = 20 per group) of participants were investigated who made at least 20+ errors on the Flanker task. The first 20 artifact free error responses for each participant were included to compare groups on ERN amplitude. In this analysis that controlled for the number of trials, a Group by Accuracy (error, correct) ANOVA yielded a significant interaction, $F(1, 38) = 7.17$, $p < .001$, $\eta^2 = .32$). Follow-up independent t-tests showed that groups differed on both error (Control: $M = -7.78$, $SD = 4.24$; Sleep Deprived: $M = -5.18$, $SD = 3.22$; $t(38) = -2.19$, $p = .035$) and correct trials (Control: $M = 2.08$, $SD = 3.91$; Sleep Deprived: $M = -0.32$, $SD = 2.93$; $t(38) = -2.19$, $p = .034$). Paired t-tests to compare the amplitude between error and correct trials for groups separately showed that amplitude was larger to error trials than correct trials, for both groups (*ps* < .001). The significant interaction may be explained by the fact that the difference in amplitude between error and correct trials was greater for controls ($M_{diff} = 9.86$) than for sleep deprived ($M_{diff} = 4.85$) participants. See Fig. 1 for response-locked Flanker electrophysiology.

The relationship between the number of trials and the ERN amplitude could have been due to either signal attenuation because of a larger number of trials or due to neurocognitive habituation to processing errors over time. To explore this, the sample was further reduced to a subgroup of participants who made at least 40+ errors to allow investigation of the change in ERN overtime (n = 9 for controls and 11 for sleep deprivation). A Group (Control, Sleep Deprivation) by Error Block (First 20 trials, Last 20 trials) mixed model ANOVA was run to investigate changes in ERN amplitude across time on the Flanker task. There was a significant

Table 3
Effects of sleep deprivation on stimulus-locked ERPs in the Flanker task.

	Control				Sleep deprivation			
	Congruent		Incongruent		Congruent		Incongruent	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
# trials	113.79	19.64	209.67	40.27	63.42	15.71	63.71	16.85
N2 at FCz								
Amplitude (μV)	−4.33	2.77	−4.90	2.66	−4.74	2.30	−5.16	2.20
Latency (ms)	260.38	18.06	270.33	13.61	268.83	18.17	276.71	14.80
P300 at FCz								
Amplitude (μV)	7.80	3.35	7.78	2.94	6.58	2.70	6.21	2.81
Latency (ms)	358.63	25.80	374.17	25.23	366.88	30.55	391.79	35.47
P300 at Pz								
Amplitude (μV)	9.50	3.14	9.58	2.95	8.80	2.82	8.13	2.87
Latency (ms)	341.46	33.60	347.13	59.77	369.68	50.27	398.16	35.78

Note: *M* = means; *SD* = standard deviation; N = 24 for controls and 25 for SD.

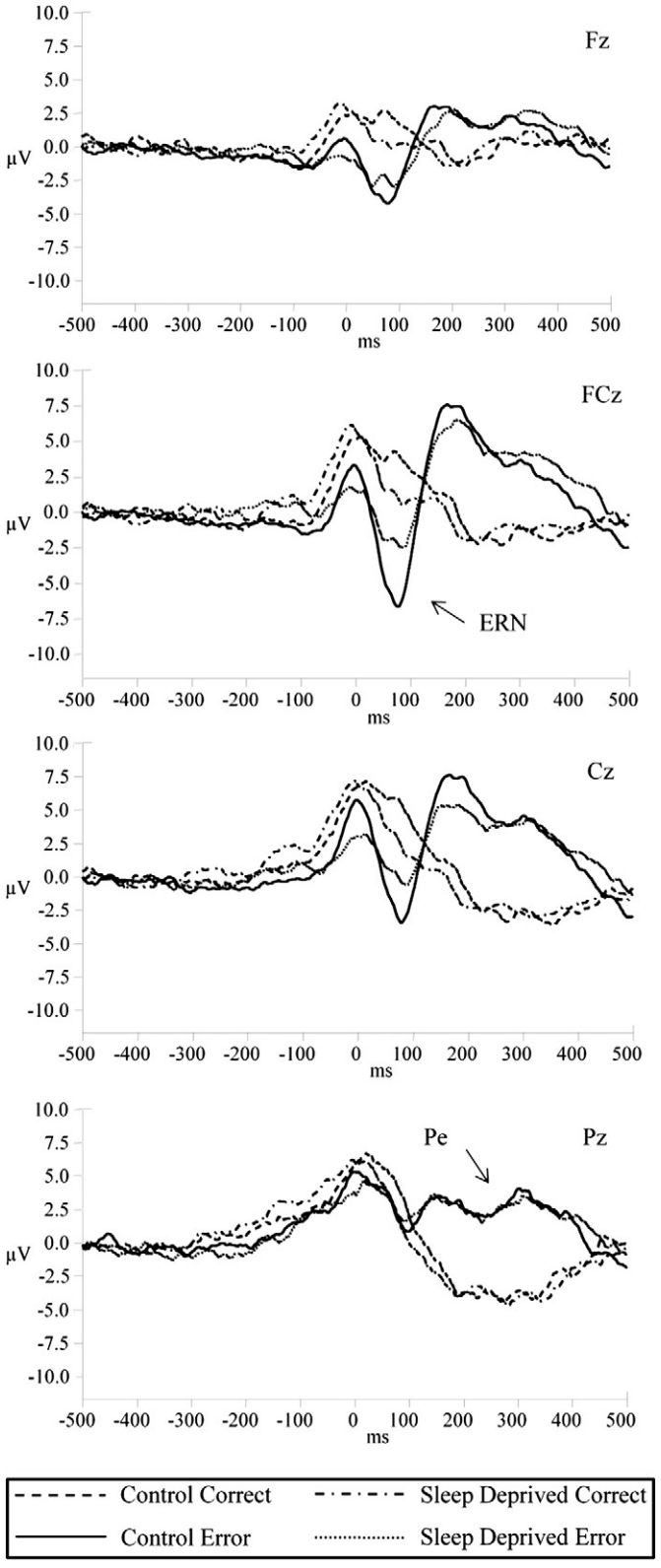

Fig. 1. Response-locked averages the first 20 artifact free correct and error responses superimposed between groups. Midline Fz, FCz, Cz, and Pz are illustrated. Dashed lines represent correct trials and solid lines represent error trials in the Control group whereas Dash-dotted lines represent correct trials and Dotted lines represent error trials in the Sleep Deprivation group. The ERN deflection is largest at FCz and Pe is largest at Pz. Grand averages are filtered 1–20 Hz FIR.

Group by Error Block interaction, $F(1, 18) = 5.06$, $p = .037$, $\eta^2 = .22$. Follow-up paired samples t-tests showed that the sleep deprived group did not differ in ERN amplitude between the first 20 and last 20 artifact free errors ($p = .63$), whereas the controls differed moderately between blocks, $t(8) = -2.22$, $p = .057$.

For the Pe component, an independent samples *t*-test initially showed no significant difference in Pe amplitude between controls ($M = 2.74$ μV, $SD = 3.04$) and sleep deprived participants ($M = 3.23$ μV, $SD = 3.38$); $t(47) = -.527$, $p = .601$. Pearson correlations were computed to assess any relation between Pe mean amplitude and performance on the Flanker task. There was a positive correlation between accuracy and Pe mean amplitude for both controls ($r = .46$, n = 24, $p = .024$) and sleep deprived participants ($r = .42$, n = 25, $p = .039$). Since accuracy impacted Pe mean amplitude, a subsample of participants was investigated if they made more than 20+ errors on the Flanker task. A Group by Stimulus Type (error, correct) ANOVA yielded a main effect for Stimulus Type ($F(1, 38) = 93.37$, $p < .001$, $\eta^2 = .71$), confirming larger Pe peaks to errors, but no significant main effect of Group or interaction. Given that the ERN component reduced as error rate increased, similar analysis procedures were carried out for the Pe component. A Group (Control, Sleep Deprivation) by Error Block (First 20/Last 20) mixed model ANOVA was run to investigate changes in Pe amplitude across time on the Flanker task. There was a significant main effect of Group, $F(1, 18) = 6.00$, $p = .025$, $\eta^2 = .25$, but no effect of Error block or interaction. When trials were collapsed across error block, the sleep deprived group ($M = 2.00$ μV, $SD = 1.98$) had a significantly larger Pe mean amplitude than controls ($M = 0.20$ μV, $SD = 1.52$).

3.6. Response inhibition: behavioral performance on the Go–NoGo task

Group (Control, Sleep Deprivation) by Stimulus Type (Go, NoGo) mixed-model ANOVAs were run to investigate the differences in response accuracy and RT. A main effect of Stimulus Type indicated that all participants made more errors to NoGo ($M = 43.95\%$, $SD = 16.56$) compared to Go stimuli ($M = 6.8\%$, $SD = 7.72$), $F(1, 47) = 283.3$, $p < .001$, $\eta^2 = .86$. A group main effect indicated that the sleep deprived group ($M = 29.6\%$, $SD = 10.0$) made more errors than controls ($M = 21.3\%$, $SD = 9.50$), $F(1, 18) = 8.91$, $p = .004$, $\eta^2 = .16$, collapsed across both trial types. Although there was no interaction, given the fundamental differences in stimulus types, follow-up t-tests were run on both Go and NoGo trials. Sleep deprived participants performed significantly worse than controls on Go trials, $t(47) = 4.75$, $p < .001$, but groups did not differ in a statistically robust way on NoGo trials ($p = .11$), although there was an 8% difference in accuracy between groups (see Table 4). For RT, a main effect of Stimulus Type indicated that all participants responded faster to unsuccessful NoGo inhibitions ($M = 299.04$ ms, $SD = 29.83$), compared to properly executed Go trials ($M = 337.56$ ms, $SD = 35.10$), $F(1, 47) = 293.7$, $p < .001$, $\eta^2 = .86$.

3.7. Response inhibition: stimulus-locked N2 and P300 ERPs on the Go/NoGo task

A Group (Control, Sleep Deprivation) by Stimulus Type (Go, NoGo) interaction, $F(1, 43) = 11.5$, $p = .002$, $\eta^2 = .21$, was found for N2 amplitude at electrode site FCz. Sleep deprived individuals tended to elicit smaller NoGo-N2 components compared to the control group, $t(43) = -1.79$, $p = .080$, whereas groups did not differ on Go-N2 amplitude ($p = .11$). In addition, all participants experienced delayed NoGo-N2 ($M = 282.82$ ms, $SD = 29.29$) compared to Go-N2 ($M = 272.78$ ms, $SD = 27.45$) ERPs, $F(1, 43) = 7.25$, $p = .010$, $\eta^2 = .14$. An independent sample *t*-test showed sleep deprived participants had attenuated Go-P300 amplitudes $t(42) = 2.07$, $p = .045$ at Pz, but did not differ in Go-P300 latency compared to the control group at electrode site Pz. Sleep deprived and control groups did not differ significantly on NoGo-P300 amplitude or latency at electrode site FCz. See Table 5 for

Table 4
Effects sleep deprivation on accuracy and reaction time in the Go/NoGo task.

	Control			Sleep deprivation		
	N	*M*	*SD*	N	*M*	*SD*
Accuracy (%)						
Go	25	97.52	2.33	24	88.71	8.80
NoGo	25	59.80	15.57	24	52.15	16.96
RT (ms)						
Go	25	331.85	31.56	24	343.51	38.21
NoGo	25	291.73	29.55	24	306.66	28.76

Note: *M* = means; *SD* = standard deviation.

Go/NoGo stimulus-locked data and Fig. 2 for stimulus-locked electrophysiology.

3.8. Response inhibition: response-locked ERN and Pe on the Go/NoGo task

Group (Control, Sleep Deprivation) by Accuracy (Error, Correct) mixed-model ANOVAs were run to investigate the differences in ERN and Pe amplitude and latency at FCz and Pz respectively to unsuccessful NoGo inhibitions. A Group by Stimulus Type interaction was found for ERN amplitude, $F(1, 42) = 12.93$, $p = .001$, $\eta^2 = .24$. The sleep deprived participants ($M = -5.72$ μV, $SD = 3.29$) had significantly smaller ERN amplitudes to NoGo errors compared to the control ($M = 9.15$ μV, $SD = 3.15$) group, $t(42) = -3.53$, $p = .001$, but did not differ on correct trials. No main effects or interactions were found for ERN latency or Pe. There were no significant correlations observed between Go/NoGo performance and stimulus or response-locked electrophysiology. See Fig. 3 for response-locked Go/NoGo electrophysiology. The mean number of trials included in waveforms for NoGo errors was 29.78 ($SD = 11.47$) for the control group and 29.91 ($SD = 13.10$) for sleep deprived group. The mean number of trials included in waveforms for NoGo corrects was 273.23 ($SD = 82.03$) for the control group and 230.41 ($SD = 106.00$) for sleep deprived group.

4. Discussion

In a well-controlled experimental study of total sleep deprivation, performance monitoring was examined using two different tasks. Sleep deprived individuals responded slower on the Flanker task and tended to be less accurate on trials following incorrect responses suggesting a minor impairment in their remedial behavior. On the Go/NoGo task, sleep deprived participants had a lower accuracy for Go hit rate, and made false alarm responses to NoGO stimuli 8% more often than controls. As hypothesized, smaller ERN amplitudes for both tasks and smaller NoGo-N2 ERPs demonstrated that the performance monitoring system was impaired by sleep deprivation. Further, impaired attention was evident in the stimulus-locked P300 which was delayed in the Flanker task and smaller in amplitude to Go-trials in the Go/NoGo task relative to controls. Correlations between performance accuracy and ERN on the Flanker task showed that ERN amplitude attenuated as error rate increased. The control group showed habituation to errors in their ERN response over time (i.e., reduced recognition of errors), but sleep deprived participants failed to habituate. In a subsample of individuals who made a substantial number of errors, the Pe appeared larger in the total sleep deprivation group, possibly reflecting a greater emotional response to errors, compared to rested controls.

Table 5
Effects of sleep deprivation on stimulus-locked ERPs in the Go/NoGo task.

	Control		Sleep deprivation		*df*	*t*	*p*
	M	*SD*	*M*	*SD*			
NoGo # trials	43.13	19.64	34.00	15.41			
NoGo-N2							
Amplitude (μV)	−6.46	3.39	−4.79	2.82	43	−1.792	.080
Latency (ms)	277.91	26.72	287.95	31.55	43	−1.154	.255
NoGo-P300							
Amplitude (μV)	11.75	3.90	11.14	4.36	43	.496	.622
Latency (ms)	391.09	32.12	410.00	42.87	43	−1.680	.100
Go # trials							
Go-P300	286.22	74.34	234.50	79.96			
Amplitude (μV)	6.84	2.62	5.15	2.80	42	2.069	.045
Latency (ms)	295.83	32.43	312.45	32.29	41	−1.680	.101

Note: *M* = means; *SD* = standard deviation; *df* = degrees of freedom; N = 23 for controls and 22 for sleep deprived, except for Go-P300 (−1 sleep deprived).

4.1. Error monitoring in the Flanker task

The current study found support for the hypothesized attenuation in ERN amplitude as a result of sleep deprivation during the Flanker task. These data reflect impairment in the performance monitoring system, which may more specifically reflect deficits in error detection and compensation processes (Gehring et al., 1993), conflict detection processes (Botvinick et al., 1999), or the dopaminergic negative reinforcement learning system (Holroyd and Coles, 2002). While theories on the functional significance of the ERN are varied at present, it generally reflects controlled attention during performance monitoring; such deficits in information processing due to sleepiness are particularly impactful in environments where continuous monitoring of changing events is essential such as driving and industry operations. Inconsistent outcomes in the previous literature may stem from the type of protocol employed. Murphy et al. (2006) and Asaoka et al. (2010) found no ERN effect after prolonged wakefulness (20 h awake, i.e., about a four-hour bedtime delay for most adults) and sleep inertia protocols respectively. In contrast, the current study and others (Scheffers et al., 1999; Hsieh et al., 2007) observed significant attenuation of the ERN using more extreme sleep deprivation. It is possible that the frontal neural physiology remains unaffected or mildly affected in subtle levels of sleepiness produced by a few hours of prolonged wakefulness or sleep inertia. Balkin et al. (2002) reported that the functional connectivity between the ACC and other brain regions is stable at 5 and 20 min post-awakening response (a time of high sleep inertia); this may explain why the ERN was not affected by sleep inertia.

The ERN correlated with error rate such that the larger the error rate, the smaller the ERN. This relationship was first observed by Gehring et al. (1993) who reported that the ERN was smallest when speed was stressed over accuracy, unchanged when speed and accuracy were equally stressed, and largest when accuracy was stressed over speed. This relationship was also observed by Hajcak et al. (2003) and Herrmann et al. (2004) such that participants who made fewer errors produced larger ERNs. A response control hypothesis developed by Pailing et al. (2002) purported that individuals with larger ERNs were expected to have smaller error rates and smaller response RT differences reflecting a more controlled response strategy. Pailing et al. (2002) did not find evidence for a significant ERN/error rate correlation but did report a relationship between RT differences and ERN suggesting that those with less of a RT difference between correct and error trials produced larger ERNs. Although this hypothesis seems plausible, Hajcak et al. (2003) noted that the relationship between the ERN and error rate could also represent a 'habituation' response to making errors. The data reported here lend support for this habituation effect. Specifically, data illustrate that ERN amplitude changed as a function of error block as indicated by the interaction observed between the first and last 20 errors in two blocks in a subsample of participants who made more than 40+ errors. The ERN for well-rested controls was smaller in the last error block (last 20 error trials) compared to the first error block (first 20 trials). This effect was not found in the sleep deprived group; the ERN remained constant despite changes in error rate. This habituation interpretation is supported by Holroyd and Coles' (2002) reinforcement learning dopamine hypothesis. If error signals are fed back to the ACC

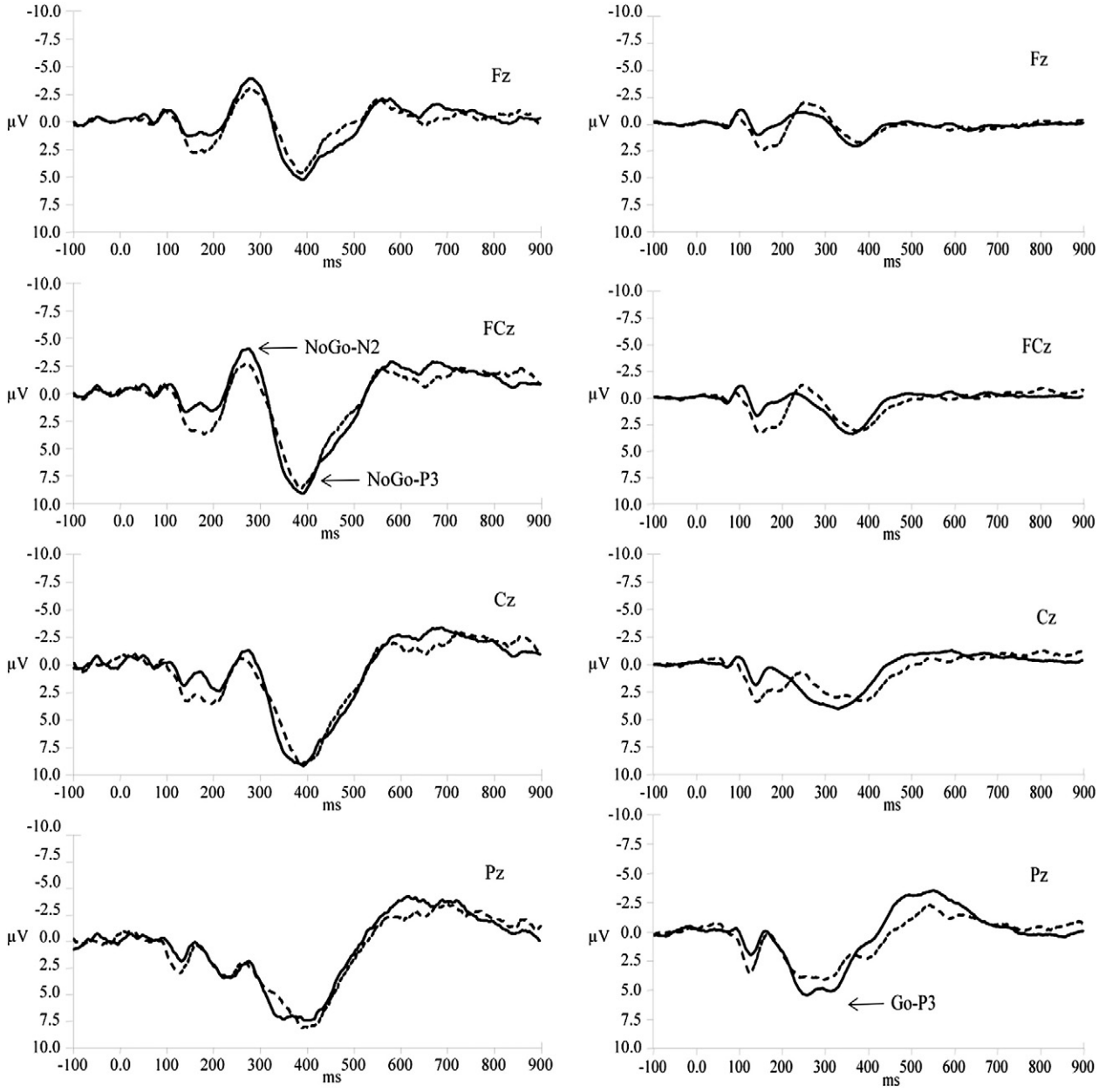

Fig. 2. Stimulus-locked averages to correct Go (right-side) and NoGo (left-side) responses in a Go/NoGo task superimposed between groups. Midline Fz, FCz, Cz, and Pz are illustrated. Black solid lines represent Control whereas black dashed lines represent Sleep Deprivation. The NoGo-N2 and NoGo-P300 deflections are largest at FCz. The Go-P300 deflection is largest at Pz. Grand averages are filtered 1–30 Hz FIR.

via the dopamine system to modify performance, and there is no consequence with repeated errors, then habituation may occur leading to reduced activity in the ACC. The habituation effect may also explain the inconsistent results observed between prior sleep deprivation studies that did not control for the number of trials in the ERP average. This result illustrates the importance of selecting not only an equivalent number of trials for response-locked averages, but also an early set of trials to draw ERN waveforms because habituation may reduce the ERN effect in well-rested controls.

In the Flanker task, the Pe did not differ between groups when all trials were included in the ERP average; however, when a subsample of individuals who committed a substantial numbers of errors was investigated, the Pe actually appeared larger in the sleep deprivation group. Previous research has reported a smaller Pe following both subtle (Murphy et al., 2006; Asaoka et al., 2010) and more extreme degrees of sleep loss sleep deprivation (Tsai et al., 2005; Hsieh et al., 2010). It is possible that the finding of a larger Pe in the current study is spurious since it was only found in the smaller sub-sample of participants, although the means indicated a larger, albeit non-significant, Pe for the sleep deprived group in the full sample. Alternatively, emotional evaluation of errors may depend on the degree of sleep loss and the number of errors made. Specifically, a subtle degree of sleep loss may impact mood regulation leading to apathy or reduced concern for errors. However, in more extreme levels of sleep loss, where more errors are made, it is possible that participants become agitated with their poor performance. Imaging data lend support for total sleep deprivation leading to greater emotional reactivity (Yoo et al., 2007). Indeed the larger Pe may represent perseveration of errors in poor performing sleep deprived individuals, which would be in keeping with diminished frontal lobe function (Harrison and Horne, 2000), and the prefrontal limbic top-down disconnect (Yoo et al., 2007).

The current study showed that sleep deprivation led to deficits in RT and RT variability which is consistent with previous research (Scheffers et al., 1999; Tsai et al., 2005; Hsieh et al., 2009, 2010), but did not find support for accuracy differences reported previously. As the sleep

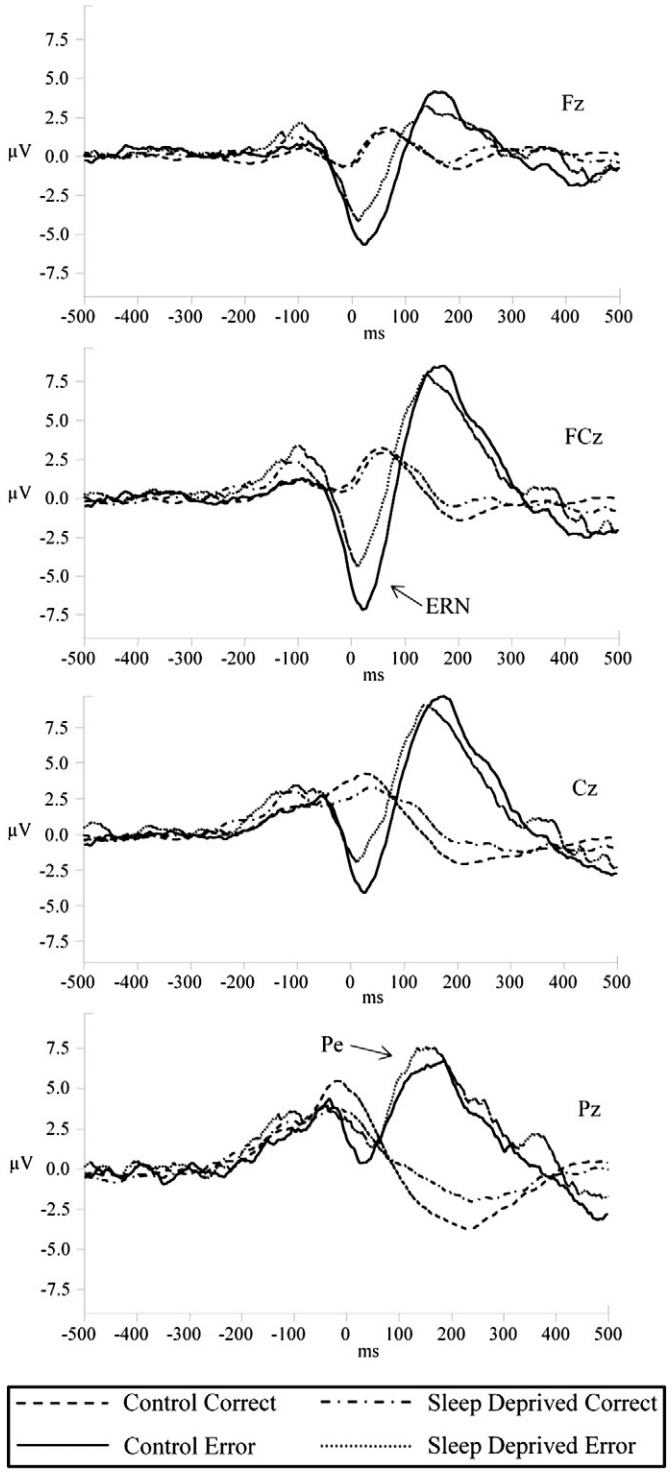

Fig. 3. Response-locked averages to incorrect responses in a Go/NoGo task superimposed between groups. Midline Fz, FCz, Cz, and Pz are illustrated. Dashed lines represent correct trials and solid lines represent error trials in the Control group whereas dash-dotted lines represent correct trials and dotted lines represent error trials in the Sleep Deprivation group. The ERN deflection is largest at FCz and Pe is largest at Pz. Grand averages are filtered 1–20 Hz FIR.

deprived participants also had delayed P300 ERP waveforms to Flanker stimuli, the slow RT is likely due to deficits in stimulus evaluation (Donchin and Coles, 1988) as a result of sleep deprivation. Despite previous literature reporting impairments in remedial behavior, post-error slowing and accuracy (Tsai et al., 2005; Murphy et al., 2006), this study did not find evidence for a slowing effect, but did find a trend in post-error accuracy suggesting sleep deprived participants were less accurate on trials following an error. The null accuracy differences observed on the Flanker and lack of support for a post-error slowing effect may be explained by task difficulty. Harrison and Horne (2000) argued that sleep deprived individuals expend greater motivation and compensation for more complex and "rule-based" tasks. This has been observed in an imaging study by Chee and Choo (2004) who showed behavioral performance changed as a function of task complexity after sleep deprivation. They also showed increased frontal activation during a complex task and interpreted this as a compensation strategy to overcome the effects of sleep deprivation. Similarly, reports have shown associations with Pe amplitude and remedial behaviors like post-error slowing (Nieuwenhuis et al., 2001; Hajcak et al., 2003) where unperceived errors typically have smaller Pe amplitudes (Nieuwenhuis et al., 2001). Our study and others (Falkenstein et al., 2000; Dywan et al., 2004) have shown a relationship such that the more errors of commission, the smaller the Pe amplitude. Task difficulty has been shown to affect Pe amplitude (Falkenstein, 2004), therefore, it may be that the difficulty of Flanker task employed in the present study resulted in unperceived errors which ultimately nullified a post-error slowing effect.

4.2. Response inhibition

Reductions in the commonly studied stimulus-locked ERPs, the NoGo-N2 and Go P300, during the response inhibition task were consistent with Breimhorst et al. (2008) and Schapkin et al. (2006). These ERP differences reflect impairment in the inhibitory network (probably at a pre-motor level; Breimhorst et al., 2008) and resources necessary for normal levels of information processing after sleep deprivation. The current study was the first to investigate response-locked electrophysiology to a Go/NoGo response inhibition task after total sleep deprivation. Consistent with the findings on the Flanker task, reduced ERN amplitudes were observed to false-positive NoGo stimuli in the sleep deprived group. No significant differences were observed in the Pe component. These data support the hypothesis that sleep deprived individuals have impaired error monitoring as indexed by attenuation in ERN amplitude. To date performance monitoring has been investigated with modified Flanker tasks and our data add to a body of evidence that error monitoring is impaired on tasks that require response inhibition as well. There were no correlations between ERN or Pe amplitude and accuracy on the response inhibition task; this may be because there were a comparable number of trials in the group averages for errors, and less inter-subject variability, on the Go–NoGo task.

In the current study, although the sleep deprived group failed to inhibit their response to NoGo stimuli 8% more often than controls, the group differences was not statistically robust. This may be explained by the relatively low accuracy performance in controls in this study (60% successful inhibitions to NoGo trials). The task was designed such that 'X' stimuli were Go trials and '+' stimuli were NoGo trials; this may have introduced some degree of cognitive interference and increased task difficulty. Alternatively the control participants may have had reduced motivation due to the lengthy paradigm. Future research should investigate response inhibition tasks that manipulate both difficulty and motivation.

4.3. Conclusions and implications

The reduced amplitude ERNs observed in the sleep deprived group in the current study clarifies an equivocal literature and extends our understanding of deficits in performance monitoring of tasks that involve response inhibition. These data lend support for the hypothesis that sleep deprivation leads to a deficit in frontal regions of the brain (Harrison and Horne, 2000). Sustained attention is largely influenced by proper PFC function. Given that both the ERN and NoGo-N2 have been shown to be generated in the dorsal ACC (an area of the mPFC: van Veen and Carter, 2002), their alteration in sleep deprived participants

illustrates that PFC function underlies performance instability during sleep loss. Where dorsal ACC regions appear impaired following total sleep deprivation, these areas may not be impaired during minor sleep delays or sleep inertia.

The current study also provided new evidence for failure to habituate and perseveration of errors on the Flanker task in sleep deprived individuals. Well-rested individuals habituated with increased error rate (supported by a reduction in ERN amplitude as error rate increased), whereas sleep deprived individuals remained stable throughout the task regardless of error rate. The consequence of failing to habituate to stimuli or errors during sleepiness may be that individuals process all stimuli as novel or meaningful; allocating unnecessary extensive processing to repeated stimuli would reduce attention resources available for efficient information processing. This novel finding of failure to habituate to errors in sleep deprived individuals needs further systematic study. The larger Pe response in sleep deprived individuals who made a substantial number of errors is also a novel finding which may represent perseveration of errors and be a marker of the degree of emotion dysregulation during sleepiness. These data raise the possibility that Pe may be modulated by the degree of sleep deprivation or individual differences in performance. Further research on individual differences in error monitoring following sleep deprivation in larger samples is needed to garner a better understanding of factors related to emotional evaluation of errors indexed by the Pe.

Understanding of the neural basis of deficits in performance monitoring abilities is particularly important for our increasingly sleep deprived society and for safety and productivity in situations like driving and the workplace. The impact of sleep deprivation on frontal lobe function and performance monitoring may be especially relevant for adolescents and older adult age groups because of the age-related susceptibility to compromised frontal lobe function. Poorly defined frontal function (Luna and Sweeney, 2004), along with chronic sleep deprivation (Carskadon et al., 1998), combine to negatively impact the performance and well-being of adolescents. Similarly, previous research has reported performance monitoring deficits (Dywan et al., 2004), and poorer and more fragmented sleep in older adults (Bliwise, 2011). Further research is needed in samples like adolescences and older adults to determine if sleep deprivation leads to greater impairment in performance monitoring in these groups.

Conflict of interest

There are no conflicts of interest.

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