The influence of partial sleep restriction on five ERP components

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Abstract

Sleep loss can have multiple mild to disastrous effects on human functioning, especially attention. Several distinct neuronal networks are believed to reflect the different aspects of visual attention. Event Related Potentials (ERPs) provide an insight into the neuronal networks involved whilst the Attentional Network Test (ANT) enables measurement of the three attentional networks known as the alerting, orienting and executive control networks. Sleep loss is known to have an effect on both attention as measured with the ANT and on ERP components. However, the combination of these three aspects, namely the effect of sleep loss on ERP components as measured during the ANT is still not clear. This study researched the effects of three consecutive days of partial sleep restriction (4 hours in bed) on the P1, N1, P2, N2 and P3 ERP components, measured during the ANT, compared to a normal sleep condition (8 hours in bed). Ten healthy participants took part in the experiment which lasted a total of seventeen consecutive days, including a week of baseline and the two sleep conditions, counterbalanced across participants. Unexpectedly, the reaction times, accuracy and network scores did not significantly worsen due to sleep restriction. Analysis of the five ERP components was difficult as those components were not present in the ERP waveforms for all participants leading to extremely low sample sizes for statistical tests. Equivalence testing showed the parietal P1 component was not significantly different in amplitude and latency for the two sleep conditions. The frontal P3 component rendered a non-significant trend towards a decrease in amplitude and increase in latency due to sleep restriction. The frontal P3 amplitude and latency were found to be the most sensitive markers for sleep restriction. An application of the findings as well as recommendations for future research were discussed.

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Introduction

General introduction

Recent global surveys revealed that 62% of adults admit their sleep could be improved (Philips, 2019) and only 49% of people are satisfied with their sleep (Philips, 2020). Moreover, looking back at prior demographic studies, it seems that adults in this time and age sleep significantly less than the generation before us (Liew & Aung, 2021; Luyster et al., 2012). Indeed, in 1985, about 22% of the adult population in the United States of America reported sleeping 6 hours or less per night (Schoenborn, 1986). Results from between 2005 and 2008 showed that already 37.1% of American adults sleep less than 7 hours a night, with inter-individual and cross cultural differences (Centers for Disease Control and Prevention (CDC), 2005-2008). Current worldwide statistics show people sleep on average 6.8 hours per night (Horan, 2021) which is about ±1.2 hours less than the recommendations (7 to 9 hours). These statistics are worrisome, as shorter and longer sleep durations compared to the recommendations are associated with increased mortality and morbidity (CDC, 2005-2008; Watson et al., 2015). Loss of sleep by itself could have immense effects on peoples' daily life, resulting in, for example, large-scale health problems and cognitive impairments such as lapses in attention and challenges related to executive control (Krause et al., 2017; Liew & Aung, 2021).

Aside from studying ways to prevent sleep loss, research aims to develop interventions to support people struggling with its consequences during the wake phase. In order to be successful, one must define and evaluate monitoring tools to quantify the impact of sleep loss on attention and gain a better understanding of the underlying mechanisms. This report aims to investigate such a monitoring tool to enable the quantification of the effects of sleep loss, specifically the effects on attention. *In fine*, this knowledge could be applied among sleep-disorder patients.

Sleep deprivation

In the event that sleep loss leads to a decrease in both performance and attention as well as a deterioration in health, sleep deprivation occurs. This can be due to a decrease in sleep quantity or due to impaired sleep quality. A decrease in sleep quality equals multiple awakenings or arousals during the night. Sleep debt results from chronic sleep loss and cannot be recovered from (Abrams, 2015). A review by Durmer and Dinges (2005) gave a summary of neurocognitive performance decrements observed in studies among healthy participants that have been imposed a sleep deprivation to be consistent with decrements seen in real-life sleep deprived people due to, for example, disease-related sleep fragmentations. Furthermore, they distinguished three types of studies, namely short- (<45 hours) and long-term (>45 hours) total sleep deprivation (TSD) and partial sleep restriction (<7 hours per day). Appendix A provides a more thorough explanation of sleep and why it is important for humans.

Causes of sleep deprivation

Sleep deprivation can have multiple causes, a major one being one's lifestyle: shift work, late night events and stress can all lead to sleep deprivation (Abrams, 2015; Liew & Aung, 2021). Furthermore, light exposure can have an effect on sleep onset as well as awakening time. For example, the use of light-emitting electronic devices hours before bedtime can lead to the suppression of melatonin (hormone of darkness) secretion as well as an increased alertness before bedtime which both lead to a delayed sleep onset (Chang et al., 2015). Normally, humans are synchronized to the 24 hour solar day; they awake around sunrise and fall asleep around sunset (Gooley, 2017). However, due to indoor lighting and daily social activities, most people deviate from the sun imposed day-night rhythm.

Demographic characteristics also play a role in sleep deprivation: racial background, the level of education as well as age affect the amount of hours people sleep (Liew & Aung, 2021). Lastly, sleep deprivation can be caused by medical conditions such as hypertension and obesity, both related to sleep apnea, a condition affecting breathing during sleep. Other sleep conditions like insomnia and restless leg syndrome are also defined by a reduced sleep period (Abrams, 2015; Cowie, 2017; Liew & Aung, 2021). Besides, one's mental health as well as alcohol and drugs usage can have a negative effect on their sleep length (Krause et al., 2017; Roehrs & Roth, 2015).

Consequences of sleep deprivation in cognition

Sleep deprivation can lead to many adverse health consequences including mood disturbances and impaired cognitive processing. A single night of sleep loss might not immediately lead to adverse health consequences such as diabetes or stroke, but it could already have an effect on one’s cognitive processing and therefore their cognitive performance (Durmer & Dinges, 2005). Alertness, attention and vigilance are degraded after sleep loss (Killgore, 2010) which could lead to dangerous situations in, for example, traffic or workplaces (Luyster et al., 2012). According to a nation-wide survey, 23.2% of the US population reported having problems concentrating and 18.2% reported difficulties remembering things, both due to daytime sleepiness. Furthermore, 13.5% of the US adult population reported having three or more such sleep-related daytime difficulties, meaning they could not function optimally due to sleep problems the night(s) before (CDC, 2005-2008). Sleep deprivation has been shown to alter neural processing which could be the underlying reason for such observations (Boonstra et al., 2007; Magnuson et al., 2022).

Often, various tasks are used to examine cognitive performances, and therefore, indirectly, the neural processing needed to complete these tasks. These tasks often measure vigilance, attention or declarative memory in terms of reaction time and accuracy, but variations exist to also measure other types of cognitive capacities. For example, in the study by Magnuson et al. (2022), healthy participants performed two tasks measuring their inhibitory performance. They were found to perform worse on these tasks when they were sleep deprived (24 hours without sleep) compared to having slept a normal night. From these results, in combination with the results from their electroencephalography (EEG) study, they concluded that sleep deprivation led to slowed neural processing. Multiple previous studies have shown people become progressively worse on these kind of tasks, the longer they are working on

them. This worsening in cognitive performance over time is classically called the “fatigue effect” which has also been shown to exacerbate due to sleep loss (Durmer & Dinges, 2005). However, not only long cognitive tasks that induce the fatigue effect are prone to sleep loss, the performances on very brief cognitive tasks have also shown to be affected by sleep restriction. For most tasks, the accuracy will decrease and the reaction time will increase due to sleep deprivation (Durmer & Dinges, 2005; Hoedlmoser et al., 2011). These slowed reaction times could become a problem in daily life activities like hitting the break in response to a red traffic light.

Healthy participants in a study by Van Dongen and colleagues (2003) were restricted to 8, 6 or 4 hours of sleep per night for 14 consecutive days, or to TSD for 3 consecutive days. Every two hours they performed multiple tasks to examine their cognitive performances. Results showed lower levels of alertness, decreased cognitive accuracy, and decreased speed in the 4 and 6 hour sleep conditions compared to the 8 hour sleep condition. Interestingly, the subjective sleepiness increased only slightly during the last few days whilst cognitive performances were the worst those days. The authors suggest that, unlike performance measures, the subjective sleepiness ratings appeared to show adaptation to chronic partial sleep restriction. However, a ceiling effect is out of the question because participants in the TSD condition did report higher sleepiness levels. Furthermore, they suggest people cannot reliably evaluate their own sleepiness once sleep loss is chronic. It seems, therefore, crucial to have non-subjective measures to quantify the effects of sleep restriction.

Furthermore, sleep loss increases the intra-individual variability of cognitive performances and makes these performances dependent on compensatory mechanisms. These mechanisms can be seen as the brain eliciting extra neural activity in certain brain networks to compensate for the decline in performance due to other brain networks not functioning as they are supposed to. However, people are, even with increased neural activity, not able to compensate enough to completely mask the effects of sleep loss (Durmer & Dinges, 2005; Krause et al., 2017).

Other cognitive processes affected by sleep deprivation are learning and memory (Krause et al., 2017). Cognitive consequences include impairments in recall and working memory, in problem solving and decision making, as well as in maintaining focus (Durmer & Dinges, 2005; Krause et al., 2017; Szelenberger et al., 2005).

More information about the consequences of sleep deprivation on peoples’ physical and mental health as well as on their mood and emotions can be found in Appendix B.

Attention in relation to sleep deprivation

One of the most prominent cognitive processes affected by sleep loss is attention. Attention can be defined as the ability to process specifically chosen information whilst ignoring other details in the environment. Attention has a limited capacity and duration, e.g. people with a higher working memory capacity are better able to suppress irrelevant information and process only the relevant information (Cherry & Susman, 2021; Stevens & Bavelier, 2012). Often, attentional deficits due to sleep loss are seen as the cause of decreased performance (Durmer & Dinges, 2005; Martella et al., 2011). A reduction in performance on various attentional detection tasks is therefore described as generic attentional deficits or general reduction in tonic alertness (Riontino & Cavallero, 2022). Attentional impairments often lead to response failures

during tasks which are called “lapses” or “microsleeps”. These deficits accumulate as the period of sleep deprivation prolongs. The inter-individual variability in attentional impairments is high following sleep deprivation (Krause et al., 2017).

Attentional networks

Throughout the years, the attention system has been divided into three different but interrelated subsystems, namely the alerting-, orienting-, and executive control network (Fan et al., 2005; Posner & Petersen, 1990). The alerting network reflects the maintenance of an alert or vigilant state, the orienting network reflects an attentional shift to particular locations, and the executive control network reflects top-down conflict detection as well as inhibition of distracting information (Jeong et al., 2022; Posner & Petersen, 1990).

The alerting network, sometimes called vigilance network, is involved in the maintenance as well as the preparation of attention in order for people to attend to specifically specified stimuli or signals. This network of alertness can be further divided into tonic and phasic alertness. Tonic alertness is the maintenance of an alert state (also called vigilance) for a prolonged time period. Whilst phasic alertness can be seen in response to a warning signal resulting in a shift to an alert state. Alertness affects reaction time to stimuli, moreover, increased alertness is associated with faster reaction times. However, these faster reactions might be accompanied by a higher error rate as alertness does not have an effect on the amount of information people can engage with in the short time period before they can react to the stimuli (Cunningham et al., 2018; Fan et al., 2009).

The orienting network is involved in searching for and selection of information for further processing. Two separate brain systems are involved in this network, namely the dorsal attention system which provides the top-down processing of visuospatial information, and the ventral attention system which enables the bottom-up reorienting (Cunningham et al., 2018; Petersen & Posner, 2012).

The executive control network corresponds to the conflict detection as well as the inhibition capacities of the human brain. Often, the Stroop task as well as the flanker task are used to study cognitive conflict. Two relatively independent brain systems are involved here to produce top-down control, namely the frontoparietal control system which regulates the moment-to-moment task meaning switching and initiating tasks, and the cingulo-opercular system which maintains a stable background to increase overall task performance (Jeong et al., 2022; Martella et al., 2011; Petersen & Posner, 2012).

The Attentional Network Test (ANT) developed by Fan and colleagues in 2002 can be considered the golden standard to research these three attentional networks. They combined the flanker task (Eriksen & Eriksen, 1974) with the Posner’s cued reaction time task (Posner, 1980). The ANT has, among others, been used to study the altered attentional networks in people with autism and Alzheimer disease, as well as attentional changes due to sleep deprivation (Riontino & Cavallero, 2022).

Effect of sleep deprivation on ANT performance

As previously explained, sleep deprivation has a negative effect on attention. Most articles using the ANT concluded that reaction times increase and accuracy decreases due to

sleep restriction (Jugovac & Cavallero, 2012; Martella et al., 2011; Riontino & Cavallero, 2022; Roca et al., 2012). Trujillo and colleagues (2009), for example, found these results after 24 hours of TSD compared to a full 8 hour night. The effect of sleep deprivation on the separate components of attention are less well understood and even conflicting results are found about the effect of sleep deprivation on the three networks (see Table 1 for an overview). Roca and colleagues (2012) used an ANT with added alerting auditory cues. They found that the alerting network score decreased significantly due to 25.5-27.5 hours of TSD compared to the normal sleep condition, meaning the alerting effect of the auditory cue decreased significantly after one night of complete sleep deprivation. They, however, did not find such differences for the other two networks. Martella and colleagues (2011), on the other hand, found the orienting and executive control network scores to increase due to 24 hours of TSD and did not find such results for the alerting network. Compared to Roca et al. (2012), Martella et al. (2011) found significant results for different networks, namely the orienting and executive control instead of the alerting network. Furthermore, they found increasing scores compared to Roca et al. (2012) finding decreasing network scores. Moreover, Jugovac and Cavallero (2012) found different results than the articles described above. They found 24 hours of TSD to result in a decreased executive control effect compared to baseline whilst no such effect was found for the alerting and orienting networks.

Riontino and Cavallero (2022) researched the effects of sleep deprivation on attention as well, but used a version of the ANT with differing cue-stimulus intervals. For both the 400 and 800 ms cue-stimulus interval, the alerting effect was larger after 24 hours of TSD compared to baseline. Regardless of the cue-stimulus interval, the executive control effect was larger after sleep deprivation compared to baseline indicating sleep deprivation impairs the response conflict management. Unfortunately, Riontino and Cavallero (2022) did not calculate the regular orienting network score but rather calculated four different types of orienting network scores instead, making their results regarding the orienting network not comparable to the results from other articles.

Trujillo and colleagues (2009) did not calculate the three network scores, but evaluated the cue types separately. They found a significant effect of cue type on reaction time, but not for accuracy. People reacted quicker to spatial cue trials than to no or neutral cue (similar to center cue) trials and quicker to neutral cue trials than to no cue trials, regardless of sleep condition. Moreover, they found that people after 24 hours of sleep deprivation had more target misses (defined as no behavioral response within a 2000ms time limit) than they had after a full 8 hour night of sleep.

None of the articles described above found effects of sleep restriction on all three attentional networks. Because they could not find effects for all networks, they concluded no evidence was found for a global attention deficit but rather specific alerting networks were affected by sleep restriction. It is important to mention that they substantiated these conclusions on different results (i.e. finding or not finding alerting effects) (Jugovac & Cavallero, 2012; Riontino & Cavallero, 2022).

The studies described above all imposed ± 24 hours of acute TSD on participants and mostly used university students, yet found different results (see Table 1). Since no effect sizes were reported, the results from these studies cannot be easily compared and no conclusions can be drawn about the reliability of the results. The differences in results could be due to these

studies using different variations of the ANT and calculating different types of network scores or not calculating scores and just comparing cue and stimuli conditions. Furthermore, most articles fail to analyze interactions between ANT cue and stimulus types, but rather see them as options within the same factor in their analysis (i.e. (1) no cue, (2) double cue, (3) center cue, (4) spatial cue, (5) congruent and (6) incongruent). It could, for example, be that participants gain more from an alerting double cue when the stimuli is congruent whilst the double cue distracts them when reacting to the incongruent stimuli. This statement is speculative, nevertheless research is needed regarding these interaction effects during sleep restriction.

Overall, there is a need for more research on the effect sleep restriction has on the three network scores, especially on the effects of chronic and partial sleep restriction instead of 24 hours of acute TSD.

Table 1

Effect of Sleep Restriction on the ANT Networks, Compared to Normal Sleep.

					ANT networks		
First author	Year	Sleep restriction[a]	Task	Participants	Alerting	Orienting	Executive control
Jugovac	2012	24h of TSD (w)	ANT	30 adults (mean age unknown)	ns	ns	↓
Martella	2011	24h of TSD (w)	ANT	18 male students (mean age 23 years)	ns	↑	↑
			ANT-R 0 ms[b]		ns		↑
Riontino	2022	24h of TSD (w)	ANT-R 400 ms	50 students (mean age 22.5 years)	↑		↑
			ANT-R 800 ms		↑		↑
Roca	2012	25.5-27.5h of TSD (w)	ANTI-V[c]	26 students (mean age 21 years)	↓	ns	ns

Note. Only significant effects were reported. TSD = total sleep deprivation. ns = non-significant.
[a] Study was within (w) or between (b) subject design.
[b] Revised ANT with respectively 0, 400 and 800 ms cue-stimulus interval.
[c] Attention Network Test for Interactions and Vigilance.

Event Related Potentials

Since sleep deprivation affects neural processing, researchers are often interested in the brain activity during cognitive tasks. Therefore, throughout the years, electroencephalography (EEG) has been widely used in sleep deprivation research to register event-related potentials (ERPs) in the brain cortex. ERP data can, among other purposes, be used to investigate visual, cognitive and motor networks that are time-locked to stimuli (Durmer & Dinges, 2005; Jeong et al., 2022; Picton et al., 2000). Moreover, since the three attentional networks are based on separate but interacting networks with corresponding brain regions, ERPs are often used to clarify the interactions between the attentional networks as well as between their corresponding brain networks (Cunningham et al., 2018; Galvao-Carmona et al., 2014). Furthermore, ERP waveforms can be used to study what happens in the brain between cue and stimulus, and between stimulus and response, which is not possible when only using the ANT. (Luck et al., 2000).

More information about ERPs and their acquisition from EEG data can be found in Appendix C. ERP components reflect the neural processes following the stimulus, the first components (roughly within 100ms after stimulus onset) correspond to the sensory processing of the stimulus and are sometimes called exogenous. The succeeding components, sometimes called endogenous, correspond to the evaluation of the stimulus and include decision making and response-related neural processes. The differences of voltage measured by the different electrodes located on the scalp can be used to determine the neuroanatomical places where the specific processes are happening. Furthermore, the latency of the components can be used to estimate the time course of specific cognitive processes. Lastly, the amplitude of the components is believed to indicate cortical responsiveness. For example, a reduced amplitude of the N1 component is associated with reduced cortical responsiveness to incoming stimuli and therefore with reduced attention (Boonstra et al., 2007; Luck et al., 2000; Sur & Sinha, 2009).

The P1, N1, P2, N2 and P3 ERP components explained

Previous studies researched the P1, N1, P2, N2 and P3 ERP components, recorded during the ANT, and their correlations with the ANT outcomes (reaction times, accuracies and the three networks) (Kaufman et al., 2016; Neuhaus et al., 2007; Neuhaus et al., 2010; Trujillo et al., 2009; Williams et al., 2016). The P1 and N1, for example, were shown to be related to the alerting and orienting networks whilst the N2 and P3 were shown to be related to the executive control network (Williams et al., 2016). These five ERP components have also been studied in sleep restriction research and some components were shown to change in amplitude and latency due to sleep deprivation (compared to normal sleep) (Boonstra et al., 2007; Cote et al., 2003; Hoedlmoser et al., 2011; Kusztor et al., 2019; Trujillo et al., 2009; Zhang et al., 2019). Therefore, it was chosen to further research these specific ERP components. More information about these ERP components can be found in Appendix D, a summary is given in Table 2.

Table 2

Summary of the Five ERP Components Used in the Current Study.

	P1	N1	P2	N2	P3
Latency	100ms	100ms	130-250ms	200-350ms	300ms
Component elicited by…	Visual processing	Sensory processing	Object perception	Stimulus discrimination & inhibitory control	Surprising attended events or unattended events producing orienting

The five ERPs in sleep restriction research

An overview of the effects of sleep restriction on the amplitude and latency of the five ERP components can be found in Table 3 and 4, respectively. The articles presented in the

tables and below are not a full literature review, but include studies that are considered relevant due to their methodologies (tasks and manipulation). Furthermore, only articles regarding at least one of the five ERP components relevant for this study were included.

Table 3

Effect of Sleep Deprivation on the Amplitude of Five ERP Components Measured During Several Tasks.

					ERP components				
First author	Year	Sleep restriction[b]	Task	Participants	P1	N1	P2	N2	P3
Hoedlmoser	2011	24h of TSD (w)	PVT	20 young adults (mean age of 23.45 years)	↓	ns			
Stojanoski	2019	1 night of 5h (w)	PVT	26 adults (20-35 years old)	ns	ns			↓
Trujillo	2009	24h of TSD (w)	Modified ANT using exogenously and endogenously cued targets	15 young cadets from the US Military Academy and 14 young US Army soldiers	ns	↓[c]	↑		↓
Cote	2003	2 nights of fragmented sleep (w)	Auditory discrimination task	8 adults (mean age = 33.25)		↓			
Corsi-Cabrera	1999	40h of TSD (w)	Visual vigilance task	8 males (22-30 years old)		↓			
Smith	2002	1 night of TSD (w)	n-back working memory task	16 adults (21-32 years old)			↓		
Zhang	2019	36h of TSD (w)	n-back working memory task	31 male postgraduates (23-27 years old)			↓	↓	↓
Qi	2010	43h of TSD (b)	Go/NoGo task	24 male undergraduates (mean age 19)			↑	↓	↓
Peng	2020	36h of TSD (w)	3 types of two-back working memory tasks	16 college students (21-18 years old)			↑	ns	↓
Renn	2013	34h of TSD (b)	Flanker task	49 adults (18-30 years old)				ns	ns
			Go/NoGo task					NoGo↓; Go ns	Go↓; NoGo ns
Liu	2015	72h of TSD (b)	Go/NoGo task	20 male adults (18-30 years old)				ns	↓
Kaneda[a]	1999	1 night of TSD (w)	Auditory target discrimination task	Unknown					↓
Morris[a]	1992	1 night of TSD (w)	Unknown	15 adults (age unknown)					↓
Gosselin	2019	36h of TSD (measured every 6 hours) (w)	Go/NoGo task	12 students (18-26 years old)					↓

Note. Only significant effects were reported. TSD = total sleep deprivation. ns = non-significant.
[a] From Jones & Harrison review.
[b] Study was within (w) or between (b) subject design.
[c] Amplitude reduction only at parietal sites for endogenously cued targets.

P1 in sleep research.

Hoedlmoser and colleagues (2011) restricted their participants to 24 hours without sleep. During the night the participants performed the PVT hourly whilst measuring EEG. They found the amplitude of the P1 component to decrease over the course of the night whilst its latency remained the same. However, Stojanoski et al. (2019) and Trujillo et al. (2009) did not find such results for the P1 component. Stojanoski and colleagues (2019) found no effect of mild acute

sleep restriction (one night of 5 hours sleep) on the P1 amplitude whilst Trujillo and colleagues (2009) found no effect after 24 hours of TSD. Unfortunately, both Stojanoski et al. (2019) and Trujillo et al. (2009) did not research the latency of the P1 component.

Table 4

Effect of Sleep Deprivation on the Latency of Five ERP Components Measured During Several Tasks.

					ERP components				
First author	Year	Sleep restriction[b]	Task	Participants	P1	N1	P2	N2	P3
Hoedlmoser	2011	24h of TSD (w)	PVT	20 young adults (mean age of 23.45 years)	ns	ns			
Cote	2003	2 nights of fragmented sleep (w)	Auditory discrimination task	8 adults (mean age = 33.25)		ns			
Krull	1993	30h of TSD (w)	Visual reaction time task	54 male adults (age unknown)		↑			
Smith	2002	1 night of TSD (w)	n-back working memory task	16 adults (21-32 years old)			ns		
Zhang	2019	36h of TSD (w)	n-back working memory task	31 male postgraduates (23-27 years old)			↑	mismatching: ↑; matching: ns	↑
Qi	2010	43h of TSD (b)	Go/NoGo task	24 male undergraduates (mean age 19)		↑	↑	↑	↑
Peng	2020	36h of TSD (w)	3 types of two-back working memory tasks	16 college students (21-18 years old)			↑	↑	ns
Renn	2013	34h of TSD (b)	Flanker task	49 adults (18-30 years old)				ns	ns
			Go/NoGo task					ns	↑
Kaneda[a]	1999	1 night of TSD (w)	Auditory target discrimination task	Unknown					↑
Morris[a]	1992	1 night of TSD (w)	Unknown	15 adults (age unknown)					↑
Gosselin	2019	36h of TSD (measured every 6 hours) (w)	Go/NoGo task	12 students (18-26 years old)					↑

Note. Only significant effects were reported. TSD = total sleep deprivation. ns = non-significant.
[a] From Jones & Harrison review.
[b] Study was within (w) or between (b) subject design.

N1 in sleep research.

In a sleep restriction paradigm with auditory prompts to any sleep onset, Cote and colleagues (2003) observed, among eight participants, a decrease in N1 amplitude (by comparison to the baseline and recovery night) in response to an auditory discrimination task. Not only auditory stimuli result in N1 amplitude reduction after sleep deprivation, also visual- and motor-evoked potentials have shown to elicit this effect (Corsi-Cabrera et al., 1999; Boonstra et al., 2005, 2007). In 1999, Corsi-Cabrera and colleagues found amplitude reductions in multiple ERP components including the N1 after 40 hours of TSD and measured during a visual vigilance task. More recently, in a research described above, Trujillo and colleagues (2009) found the N1 amplitude to decrease at parietal sites for endogenously cued targets, but did not find this effect at other sites or for exogenously cued targets. Therefore, the N1 amplitude reduction due to sleep deprivation indicates reduced responsiveness of sensory areas to peripheral stimuli due to reduced attention (Boonstra et al., 2007).

Contrastingly, both Hoedlmoser et al. (2011) and Stojanoski et al. (2019) found no changes for the amplitude of the N1 component due to sleep deprivation. Moreover, Hoedlmoser (2011), Cote (2003) and their colleagues found sleep deprivation to have no effect on the N1 latency. Krull and colleagues (1993), however, did find 30 hours of TSD to increase the N1 latency. This last finding would suggest that the initial stimulus detections slowed down due to sleep deprivation. It seems that severe sleep restriction (e.g. 40 hours of TSD) results in a reduced N1 amplitude whilst less severe sleep restriction does not (e.g. 24 hours of TSD).

P2 in sleep research.

In a study by Zhang and colleagues (2019), participants performed a pronunciation working memory two-back task before (baseline) and after 36 hours of TSD as well as after an 8 hour recovery night. For both task conditions (matching or mismatching letters), the authors found the P2 amplitude to decrease, primarily in the frontal regions (F3, Fz and F4), due to sleep deprivation compared to baseline and recovery sleep, indicating a reduced cortical activity. More specifically, the resources used for matching processes and sensory inputs were reduced.

Smith and colleagues (2002) performed a study using a n-back spatial location working memory task with low memory load (n=1) and a high memory load trials. Participants had to stay awake for a full night in the laboratory during which they performed the task five times spread over the night. Results showed a decrease in the P2 amplitude half way into the night (at 1:30AM) and a slight recovery of the P2 amplitude later on in the night (at 3:30AM and 5:00AM). From these results, Smith et al. (2002) suggested that a short delay of sleep onset already results in a decay in the attention focused on stimuli and working memory.

Surprisingly, Trujillo and colleagues (2009) found the P2 component to increase in amplitude after 24 hours of TSD compared to a full 8 hour night. Since this is contradicting most results regarding ERPs in sleep deprivation studies, the authors searched for an alternative explanation. They proposed the increase, rather than decrease, of the P2 amplitude could be due to practice effects, as they conducted the control condition before the sleep deprived condition. Previous studies showed task repetition to increase the P2 amplitude and since Trujillo did not counterbalance the design of this study, this could be a well sustained explanation (Johnson et al., 2005; Shelley et al., 1991). Similar results were found by Peng and colleagues (2020) after 36 hours of TSD. They measured EEG during three different two-back working memory tasks, namely the two-back pronunciation working memory task, the two-back spatial working memory task which was similar to the one used by Smith et al. (2002), and the two-back object working memory task. Peng also found the P2 amplitude to be increased in the TSD condition compared to the baseline condition. However, they speculate this result is due to functional compensation of the brain in order for people to maintain normal cognitive functioning. They base this argument on the fact that brain sources decrease after sleep deprivation and the simultaneous excessive activation of the dorsolateral prefrontal cortex (Choo et al., 2005; Drummond et al., 2004). This argument is in line with the review by Durmer and Dinges (2005) which stated that sleep deprived individuals use compensatory mechanisms to mask or compensate for the cognitive deficits resulting from sleep loss. Lastly, Qi and colleagues (2010) also found the P2 amplitude to increase due to 43 hours of TSD compared to

normal sleep. They argue that the capacity to withdraw attention from the unimportant stimuli (e.g. NoGo-stimuli) is impaired after sleep restriction (Lorenzo-López et al., 2002). Regarding the P2 latency, Zhang (2019), Peng (2020), Qi (2010) and their colleagues found an increase due to sleep restriction whilst Smith et al. (2002) found no such effect.

N2 in sleep research.

In the same article explained above, Zhang et al. (2019) found similar results for the N2 component regarding the amplitude as for the P2 component. Regarding the latency, sleep deprivation resulted in a longer N2 latency in the mismatching condition. However, no such effects were found in the matching condition. Furthermore, Qi and colleagues (2010) studied the effects of 43 hours of TSD on several ERP components measured during a Go/NoGo-task. For the N2, they found similar results as Zhang et al. (2019), namely a decreased N2 amplitude and increased N2 latency in the sleep restriction condition compared to the normal sleep condition. The study by Peng et al. (2020), as explained above, showed the N2 amplitude decreased due to 36 hours of TSD, but this decrease was not statistically significant. It was suggested that the absence of N2 amplitude change, as for the P2 amplitude increase, was due to compensatory responses. Furthermore, a significant increase in N2 latency was found compared to the baseline condition, reflecting the increase in RT relative to baseline.

Renn and Cote (2013) recorded participants' EEG data during a flanker task as well as a Go/NoGo-task before and after 34 hours of TSD. For the flanker task, they found no significant effect of sleep deprivation on the N2 amplitude or latency at electrode FCz. However, for the Go/NoGo-task, sleep deprivation led to decreased N2 amplitude for the NoGo trials whilst no differences were found for the Go-N2 amplitude. The decreased NoGo-N2 amplitude is thought to reflect the impaired performance monitoring system due to sleep deprivation.

P3 in sleep research.

According to a review by Jones and Harrison (2001), multiple studies in the 1990's already showed the effects of sleep deprivation on the P3 component. All of these studies found the same effects, namely an increase in P3 latency and decrease in P3 amplitude due to a single night of TSD. More recently, these exact results were found after 34 hours at electrode Pz (Renn & Cote, 2013), after 36 hours (Zhang et al., 2019) and after 43 hours of TSD at electrode Cz (Qi et al., 2010). Peng et al. (2020), Trujillo et al. (2009) and Stojanoski et al. (2019) found the P3 amplitude to decrease after sleep restriction. Unfortunately, both Trujillo et al. (2009) and Stojanoski et al. (2019) did not study the effects on the P3 latency. Peng et al. (2020) found a prolonged P3 latency after 36 hours of TSD, but this was not statistically significant. Contrastingly, Renn and Cote (2013) found no effect of sleep deprivation on the P3 amplitude as measured during the flanker task. For the P3 measured during the Go/NoGo-task, only the Go-P3 amplitude decreased due to sleep deprivation. Furthermore, the P3 latency as recorded during the flanker task was increased at the Pz electrode due to sleep deprivation whilst for the Go/NoGo-task this effect was not found. According to Renn and Cote (2013), the increased P3 latency during the flanker test as well as the decreased amplitude during the Go trials indicated impaired attention due to sleep deprivation.

Regarding the P3a and P3b, Gosselin and colleagues (2005) found reduced amplitudes after 36 hours of TSD compared to baseline. Since performances on the auditory oddball task

decreased as well, they concluded that the whole attentional network was affected by sleep deprivation. In a later study they found the amount of sleep deprivation to have an effect on the P3 component during a Go/NoGo-task (Gosselin et al., 2019). They measured the latency and amplitude of the P3 every 6 hours during 36 hours of TSDand found the latency to increase and the amplitude to decrease with increasing time spent awake. After 12 hours of wakefulness, the P3 amplitude was already significantly reduced compared to baseline. Furthermore, after 36h awake, the P3 amplitude reduced by almost 50% compared to baseline. Even after a recovery night of about ten hours, the P3 had not returned to its baseline properties. Since both the Go and NoGo P3 amplitudes are decreased due to sleep loss, it is thought the sleep deprivation affects detection instead of inhibition processes.

Lastly, caffeine administration results in opposite effects as sleep deprivation, namely a decreased P3 latency and increased P3 amplitude. These results, together with the findings that the P3, as well as the N1, are influenced by dopaminergic, noradrenergic and cholinergic systems (systems known to mediate arousal and attention), support the view of (a lack of) attention having an effect on the P3 and the N1 (Boonstra et al., 2007).

Discrepancies in effects of sleep deprivation on ERP components.

As can be seen in Table 3, the P3 amplitude rendered the most consistent findings, while the P2 amplitude rendered mixed effects and the P1, N1 and N2 amplitude showed a decrease or null effects. Based on the latency changes shown in Table 4, if results were significant, all components render a longer latency. Yet, there are quite some null findings.

The contradicting results the above mentioned articles reported regarding the amplitude and latency changes of ERP components due to sleep deprivation could be due to the use of different tasks, but also to the application of different styles of sleep restriction. In some studies participants were restricted to TSD (Corsi-Cabrera et al., 1999; Gosselin et al., 2019; Hoedlmoser et al., 2011; Kaneda et al., 1999; Krull, 1993; Morris et al., 1992; Peng et al., 2020; Qi et al., 2010; Renn & Cote, 2013; Smith et al., 2002; Trujillo et al., 2009; Zhang et al., 2019), whilst in other studies they were restricted to partial sleep restriction or fragmented sleep (Cote et al., 2003; Stojanoski et al., 2019). Moreover, the duration of the sleep restriction varied from one (Hoedlmoser et al., 2011; Kaneda et al., 1999; Morris et al., 1992; Smith et al., 2002; Stojanoski et al., 2019; Trujillo et al., 2019) to several consecutive days (Corsi-Cabrera et al., 1999; Cote et al., 2003; Gosselin et al., 2019; Krull, 1993; Peng et al., 2020; Qi et al., 2010; Renn & Cote, 2013; Zhang et al., 2019). Ecological validity, the extent to which the results can be generalized to real-life situations, plays an important role here. Acute TSD, for example, has a lower ecological validity than chronic partial sleep restriction. Moreover, the order of the conditions could have an effect on the results. Gosselin et al. (2019), for example, carried out baseline, then sleep restriction and then a recovery night whilst Trujillo et al. (2009) carried out the control and then sleep restriction condition. As mentioned above, learning could play a role when performing the same task multiple times, leading to the possibility of results showing these learning effects instead of the effects of the sleep restriction. Besides, a wash-out period between the conditions is often not employed leading to the possibility of the effects of the first condition to ‘leak’ into the second condition. Studies with a counterbalanced crossover design and sufficient wash-out period between sessions could overcome these problems, because the learning effects are then canceled out over both conditions.

Another possible reason for the contradicting results is the variety of participants. The study by Trujillo et al. (2009), for example, used United States Military Academy cadets as well as US Army soldiers, and the study by Lui et al. (2015) used male astronauts. Both these participant groups cannot be seen as a good representation of society. Moreover, participants' age could have an effect on the results. Kaufman et al. (2016) as well as Williams et al. (2016) studied the effects of age on ERP components measured during the ANT. Kaufman et al. (2016), for example, found for all cue types, older adults (64.8 ± 8.0 years) had smaller N1 amplitudes compared to younger adults (22.9 ± 4.0 years). Likewise, smaller P3 amplitudes were recorded for both stimulus types for the older adults compared to the younger adults (Kaufman et al., 2016). Moreover, Williams et al. (2016) reported older adults (60–76 years) were slower than younger adults (18–29 years), for all ANT trial types.

Lastly, inter-individual as well as intra-individual variability is often not taken into account in the mentioned articles as they average over all participants. Due to sleep restriction these variabilities increase. Firstly, one participant might just be better at a specific cognitive task than another participant. It could be that one sleep-deprived participant's worst performance is superior to another non-sleep-deprived participant's best performance. Secondly, due to learning effects, participants could improve on a repeated cognitive task regardless of sleep restriction diminishing their cognitive abilities. Thirdly, sleep restriction might immensely impair the cognitive abilities of a participant whilst it might enhance the abilities of another participant (Durmer & Dinges, 2005; Krause et al., 2017).

Therefore, future research should include participant differences as well as any interactions between participant factor and sleep condition in the analysis. Aside from the amount of sleep restriction, the task type and the amount of participants could be arguments for the differences in results, when inspecting Table 3 and 4, no trends were found regarding the impact of these factors on the results. Lastly, to increase ecological validity, there is a need for studies researching the five mentioned ERP components after chronic partial sleep restriction with a counterbalanced crossover design.

The five ERPs during the Attentional Network Test

Whilst the ANT is a commonly used task to monitor attention, it has, at least to our knowledge, not been used in studies investigating the impact of sleep restriction on ERP components. Earlier research did study the relationship between ERP components and the results of the ANT itself, but not during a sleep restriction paradigm. Some of those recent studies regarding the five ERP components relevant for this study are explained below. Williams and colleagues (2016) measured the P1 at occipital (O1, Oz, O2) and parietal (P3, Pz, P4) sites during the different cue conditions of the ANT. They found the P1 amplitude to be larger in the no cue condition in comparison to the double cue condition, and in center cue trials in comparison to spatial cue trials. Such results seem to indicate more cortical activation is needed to respond during these conditions (no cue, center cue). These results echo with observations of longer RT's found for the no cue and center cue trials compared to the double and spatial cue trials, respectively, indicating more processing was needed for the no cue and center cue trials. For these four conditions, the P1 amplitude was more positive at the occipital sites compared to the parietal sites.

Neuhaus and colleagues (2010) found the pooled N1 amplitude (interpolation of all analyzed electrodes) in double and spatial cue conditions to be larger than in the no cue and center cue conditions, respectively. These findings correspond respectively to the alerting and orienting effects. The alerting effect was most pronounced at the parietal electrodes (P3, Pz and P4) as at those sites, the double and no cue differed the most. Moreover, this effect was to a lesser extent present at other analyzed electrodes (O1 and O2). Surprisingly, the alerting effect was reversed over the PO9 and PO10 electrodes: the double cue resulted in a smaller N1 amplitude compared to the no cue condition. The orienting effect was mostly found in the occipital electrodes (O1 and O2); at those sites, the spatial and center cue differed the most. Similar effects were found by Kaufman et al. (2016) and Williams et al. (2016), namely a main alerting and orienting effect for the N1 amplitude. Furthermore, Williams et al. (2016) reported the alerting effect to be larger at parietal than occipital sites whilst the orienting effect was larger at the occipital sites (O1, Oz, O2) compared to the parietal sites (P3, Pz, P4).

The P2 and N2 components have seldom been studied in ANT research. The article by Trujillo et al. (2009) reported smaller P2 amplitudes for exogenous compared to endogenous ANT trails. Furthermore, P2 amplitudes were larger for spatial cue trials than for neutral and no cue trials. Regarding the N2 amplitude, Williams et al. (2016) only found a significant interaction effect, namely the fronto-central N2 amplitude was larger during incongruent trials compared to congruent trials for younger adults (18-29 years old) and not for older adults (60–76 years old). Similar results were found in the study by Renn and Cote (2013) including only younger adults: compared to congruent trials, they found a larger amplitude and longer latency during incongruent trials. Another study showed no flanker congruency effects on the N2 amplitude, in this study the N2 latency was not analyzed (Neuhaus et al., 2007).

In contrast, the P3 component has been widely studied in ANT research. Williams et al. (2016) analyzed this component averaged over centro-parietally (Pz, CPz) and fronto-centrally (Cz, FCz) sites. Firstly, the P3 was larger and appeared earlier at centro-parietal sites than at fronto-central sites. At centro-parietal sites, the P3 amplitude was reduced and its latency was increased for incongruent compared to congruent stimuli. No such effects were found at fronto-central sites. Regarding the amplitude, Neuhaus et al. (2007) found the same results at Pz as Williams et al. (2016) found at centro-parietal sites. Similarly, they found the same P3 latency increase at Cz as Williams et al. (2016) found at fronto-central sites. According to Neuhaus et al. (2007), the reduced P3 amplitude for the increased task difficulty (incongruent vs congruent stimuli) is seen frequently in healthy participants. The increased P3 latency for incongruent stimuli indicates people need more time to cognitively process incongruent compared to congruent stimuli.

In a later study by Neuhaus and colleagues (2010), they found P3 amplitude at Pz to be lower for incongruent compared to congruent trials. However, they also found the opposite results at the Fz electrode, namely an increased P3 amplitude for incongruent compared to congruent trials. According to Neuhaus et al. (2010), these results reflect response inhibition. Moreover, they did not find any differences in the P3 latency for the congruent and incongruent stimuli. Lastly, Kaufman et al. (2016) also found the P3 amplitude to decrease for incongruent stimuli compared to congruent stimuli; they did not analyze the P3 latency.

As for the effects of sleep restriction on the ERP components, the articles regarding these components during the ANT (without sleep restriction) also report contradicting results.

This, again, could be due to differences in participants, but the manner in which the ERPs are analyzed could also play a role here. Some articles analyzed the ERP components at certain brain sites (Hoedlmoser et al., 2011; Peng et al., 2020; Williams et al., 2016; Zhang et al., 2019) whilst other articles analyzed them at other sites or at a single electrode (Cote et al., 2003; Gosselin et al., 2019; Qi et al., 2010; Renn & Cote, 2013), still other articles analyzed them as an average of all electrodes (Neuhaus et al., 2010; Stojanoski et al., 2019). Furthermore, some articles analyzed the ANT network scores (Jugovac & Cavallero, 2012; Martella et al., 2011; Neuhaus et al., 2007; Riontino & Cavallero., 2022; Roca et al., 2012) whilst others analyzed the trial types separately (Neuhaus et al., 2010; Trujillo et al., 2009; Williams et al., 2016). This makes comparisons between these articles' results difficult, resulting in issues evaluating the relationship between the ERP components and attention. Further research in this field is needed in order for these relationships to become apparent and therefore useful for attentional monitoring tools.

Research questions & hypothesis

As stated above, the P1, N1, P2, N2 and P3 ERP components are widely studied in both sleep restriction and attentional research. However, the studies regarding the effects of sleep restriction on these ERP components were often measured during several different cognitive tasks (see Table 3 and 4 for an overview), but not during the ANT, a task monitoring three forms of attention. Similarly, the studies regarding the ANT and these ERP components were often not measured during sleep deprivation. Likewise, the studies that researched the effects of sleep deprivation on the ANT outcomes did not measure ERP components. Moreover, discrepancies can be observed in the results of the research done on the relationships between these three fields of research. Therefore, it is concluded that a literature gap is present in the combination of these three fields of research, more specifically the effect of sleep deprivation on the ERP component measured during the ANT. This gap is highly relevant because the ERP components could provide an underlying explanation for the effects of (the lack of) sleep on attention. Furthermore, when the relationships between these three factors are known, monitoring tools can be developed to track the effects sleep loss has on peoples' attention through the measurement of the ERPs. Eventually such monitoring tools could lead to the development of interventions to support patients suffering from sleep loss. The literature gap as well as the discrepancies described above lead to the following main research question:

What are the effects of 3 consecutive days of partial sleep restriction on the P1, N1, P2, N2 and P3 ERP components (measured during an Attention Network Test) compared to a normal sleep condition?

To better answer this question, it is divided in two investigations: (a) *to what extent are there differences in the amplitude of the P1, N1, P2, N2 and P3 ERP components between the sleep restriction and the normal sleep condition;* (b) *to what extent are there differences in the latency of the ERP components between the sleep restriction and the normal sleep condition?.*

All hypotheses regarding these two investigations were based on the articles and their conclusions regarding the effects of sleep deprivation on the ERP components presented in

Table 3 and Table 4. The P3 seems to be the most sensitive to sleep loss since it was already reduced in amplitude and increased in latency due to only a few hours of sleep loss. The other components are less sensitive to sleep loss (e.g., only reducing in amplitude after longer periods of sleep loss) and some components seemed to show no clear moderations in amplitude and latency as a function of the sleep condition.

Regarding the first investigation, similar results as Stojanoski et al. (2019) and Trujillo et al. (2009) are expected, that is to say a lack of change in the P1 amplitude, therefore the first hypothesis is:

Hypothesis 1:
Partial sleep restriction will not lead to a change in P1 amplitude during the ANT compared to the normal sleep condition.

No effects were found on the N1 amplitude within a single day of sleep deprivation (Hoedlmoser et al., 2011; Stojanoski et al., 2019). However, more chronic sleep deprivation led to reduced N1 amplitudes (Corsi-Cabrera et al., 1999; Cote et al., 2003). Since the participants in the current study will undergo partial sleep restriction for three consecutive nights, the N1 amplitude is expected to decrease compared to normal sleep. Regarding the N2 and P3 amplitude, most articles reported a decrease in amplitude due to sleep restriction. Therefore, the second hypothesis is:

Hypothesis 2:
Partial sleep restriction will lead to a decreased N1, N2 and P3 amplitude during the ANT compared to the normal sleep condition.

Smith et al. (2002) and Zhang et al. (2019) found the P2 to decrease in amplitude due to sleep restriction. However, Peng et al. (2020), Qi et al. (2010) and Trujillo et al. (2009) reported an increase in the P2 amplitude. Since Smith et al. (2002) and Zhang et al. (2019) measured the P2 during a n-back working memory task whilst this current study will measure the P2 during the ANT, like Trujillo et al. (2009) did, the results by Smith et al. (2002) and Zhang et al. (2019) are considered less relevant in site of the current research question. Therefore, the third hypothesis is:

Hypothesis 3:
Partial sleep restriction will lead to an increased P2 amplitude during the ANT compared to the normal sleep condition.

Regarding the second investigation, Hoedlmoser et al. (2011) found no significant effect of sleep restriction on the P1 latency. Therefore, the fourth hypothesis is:

Hypothesis 4:
Partial sleep restriction does not lead to a change in the latency of the P1 ERP components obtained during the ANT compared to the normal sleep condition.

The studies by Krull et al. (1993) and Qi et al. (2010) found a significant increase in N1 latency due to sleep deprivation whilst the studies by Hoedlmoser et al. (2011) and Cote et al. (2003) found no such significant results. Cote et al. (2003) applied an auditory discrimination task and since the current study will apply a visual ANT, the results by Cote et al. (2003) are considered less relevant than results from studies that apply visual tasks. Besides, Krull et al. (1993) and Qi et al. (2010) apply chronic TSD whilst Hoedlmoser et al. (2011) applied only 24 hours of sleep restriction. Moreover, Krull et al. (1993) and Qi et al. (2010) used more participants than Cote et al. (2003) and Hoedlmoser et al. (2011), so their results have higher statistical power. Since the current study will apply chronic sleep restriction, the N1 latency is expected to increase due to sleep restriction compared to normal sleep.

Regarding the P2, only Smith et al. (2002) reported no effect of sleep deprivation on its latency after a single night of TSD. Since Peng et al. (2020), Qi et al. (2010) and Zhang et al. (2019) reported an increase in P2 latency due to more chronic sleep restriction, the P2 latency is expected to increase. Regarding the N2 and P3, most articles concluded the latencies of these ERP components increased due to sleep deprivation. Therefore, the fifth hypothesis is:

Hypothesis 5:

Partial sleep restriction will lead to an increased latency of the N1, P2, N2 and P3 ERP components obtained during the ANT compared to the normal sleep condition.

Furthermore, we exploratively investigate to what extent are there any differences in the sleep-dependent moderations in the amplitude and latency of ERP components between the orienting, alerting and executive functioning networks of the ANT?
Currently, there is no data on the effect sleep deprivation has on the P1, N1, P2, N2 and P3 ERP components within the different networks of the ANT. Therefore, these analyses will be more exploratory in nature.

Lastly, we'll explore whether the behavioral markers, obtained with the ANT, or the EEG-derived metrics are more sensitive to chronic partial sleep restriction, this will be done by comparing effect sizes.

Methods

Design

To investigate how three consecutive days of partial sleep restriction influences the P1, N1, P2 and P3 ERP components, a counterbalanced crossover experiment with a within-subject design was applied. The independent variable was the amount of sleep participants got; either restricted or normal. The dependent variables were the performances on the ANT and the physiological EEG measurements. During the whole experiment ecological validity was kept as high as possible. People with sleep problems sleep only a few hours per night for sometimes multiple nights in a row, to best simulate this, chronic partial sleep restriction was applied. Moreover, during the whole experiment, participants were asked to wake up at the same time they would normally do and go to bed either four or eight hours prior.

It is important to note that this experiment was part of a larger study by PhD candidate Vaida Verhoef and colleagues aiming to develop a sensitive protocol to assess in-the-field sleepiness, to later validate this among patients suffering from excessive daytime sleepiness as a result of sleep disorders. Verhoef's study has the same experimental design and independent variable, but includes more dependent variables. Her study included not only the ANT, but also the Karolinska Drowsiness Test (KDT) and the PVT, during which EEG, electromyography (EMG), electro-oculography (EOG), electrocardiography (ECG) and pupillometry were performed. Furthermore, it included field measures, namely daily questionnaires measuring sleepiness and fatigue and a daily PVT. Lastly, Verhoef will include twenty participants instead of the ten included in the current study. More details about Verhoef's method can be found in the pre-registration of the study *Field Measures of Sleepiness* (Verhoef et al., 2022).

Participants

Ten healthy participants (7 male, 3 females) with a mean age of 22.6 years (SD = 4.8; range 19-32 years) were included in this study. All participants were right handed and randomly selected from the JSF participant database. Inclusion criteria were: good health, minimum age of 18 years old, normal or corrected to normal vision, understanding and answering in English, regular Android (version $\geqq$ 10) or IOS (version $\geqq$ 13) smartphone user with a Qwerty keyboard. Furthermore, participants were required to have a habitual sleep duration of 8 ± 1 hour and were screened to not have any sleep disorder or irregular sleep schedule (Pittsburgh Sleep Quality Index), to not have an extreme chronotype (ultra-short version of the Munich Chronotype Questionnaire), and to not have traveled between continents in the 3 months preceding the sampling period. Additionally, people were excluded from participation when they consumed recreational drugs, chronic medication, or smoked cigarettes. Lastly, for safety precautions, participants had to be able to travel to the laboratory at university campus by means of public transport, by bike or by foot, and they had to be able to refrain from using heavy machinery and driving motorized vehicles, during their participation.

Due to recording errors, the EEG data from one participant, recorded during the restricted sleep condition, could not be used for analysis. Another participant was excluded due

to extremely high ANT reaction times and extremely low ANT accuracies compared to other participants. Upon inspection of the results of this participant, it was concluded this participant did not fully understand the ANT, in particular whether to answer to the central or flanker arrows. Therefore, all reported data was from the remaining eight participants.

All participants provided written informed consent at the start of the experiment and received a €130 compensation with the possibility of an additional €20 bonus upon answering at least 75% of the Experience Sampling Questionnaires.

This study, including all experimental procedures, was approved by the Ethical Review Board of Eindhoven University of Technology (TU/e). Furthermore, two sleep experts from the Kempenhaeghe Sleep Center evaluated this study to have no major risks regarding the well-being of the participants. Lastly, according to the Medical Ethical Board Review (METC, Máxima Medical Center), this study does not fall under the rules of the Medical Research Involving Human Subjects Act.

Procedure

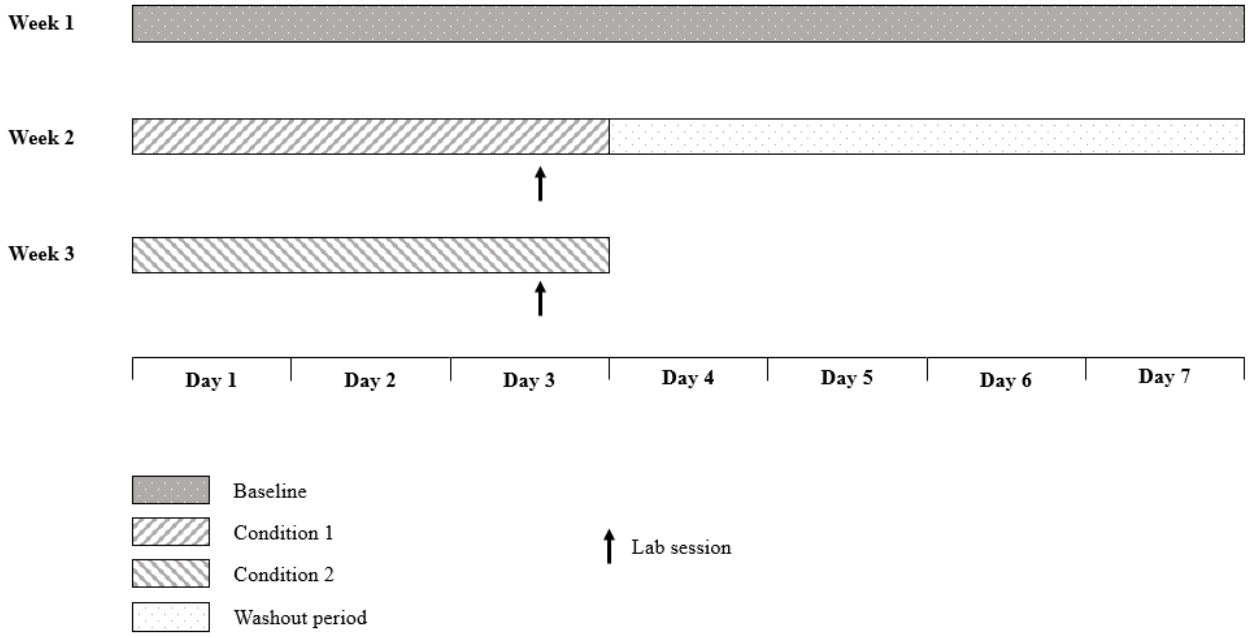
Figure 1 illustrates the time course of the study that lasted 17 consecutive days for each participant. The first week was a baseline during which participants slept according to a normal schedule (8 ± 1h of sleep per night). Participants could choose the time they went to bed and had to wake up 8 hours later. The first three days of the second week, participants either followed the normal sleep condition (as baseline) or the restricted sleep condition (3 consecutive nights of 4 hours in bed). The first three days of the third week they followed the other condition; to acquire a counterbalanced study design, the order of these two conditions differed among participants. The last four days of the second week were ‘wash-out’ days during which the participants slept according to the baseline schedule. At 3PM on the third day of both the second and third week, participants were expected in the laboratory.

The procedure of the two laboratory sessions was identical, except from the explanation of this procedure to the participants upon first arrival in the laboratory. Using the data from the actiwatch participants wore during the full experiment, it was checked whether they adhered to the agreed sleep schedule. Subsequently, three disposable adhesive ECG electrodes were placed on the torso: one just underneath the right collarbone and one under the lowest left rib, the ground was placed underneath the left collarbone. In accordance with the guidelines drawn up by Picton et al. (2000), EOG and EMG were recorded to facilitate artifact removal. Face electrodes were placed lateral to both eyes and above and below the left eye to measure horizontal and vertical eye movements, respectively. Furthermore, two electrodes were placed as a bipolar to measure jaw movements. The ground for the face electrodes was a disposable adhesive electrode placed behind the right ear. For the EEG measurement, thirteen electrodes (Fp1, Fp2, F3, F4, C3, Cz, C4, P3, P4, O1, O2, M1 and M2) were placed according to the 10-20 system. It was tested whether the measurement equipment captures the needed signals, and subsequently recordings were started. Participants were placed in a dark room in front of a laptop and were instructed to sit still as much as possible. They performed two other cognitive tasks, namely the KDT and the PVT (see Verhoef et al., 2022 for more details) before completing the ANT. Responses to the ANT were recorded with a box with two buttons that participants held in their hands. After the ANT, participants were thanked and debriefed. During

the whole experiment, alcohol and caffeine consumption were limited to 15 alcohol units per week and 4 caffeine units per day. The caffeine units included coffee, tea and energy drinks.

Figure 1

Time Course of Study



Note. Day 1 did not have to start on a Monday.

Materials and settings

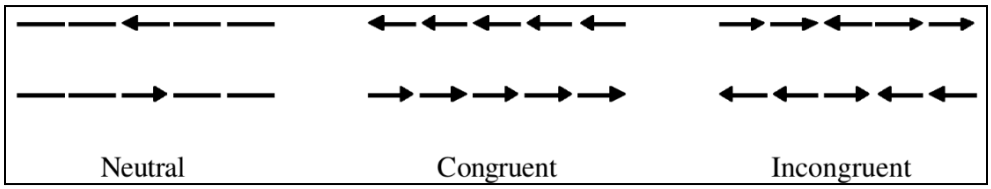
ANT

Participants' attention was measured with the ANT, during which participants had to indicate the direction of a central arrow appearing on a screen by pressing the corresponding button (left or right). On each side of this arrow, flanker arrows could be present either pointing in the same direction or in the opposite direction. This, respectively, created the congruent and incongruent conditions. Furthermore, a neutral condition was present in which the central arrow was surrounded by neutral lines instead of arrows (see Figure 2A). These arrows, with or without flankers, were shown either above or below a fixation point (+) which was positioned at the center of the screen. Before the arrows were shown, in some trials, participants received a cue (*) as shown in Figure 2B. When the cue was presented above or below the fixation (spatial cue), the arrow would appear respectively above or below the fixation. The center and double cues did not provide information about the location of the upcoming arrow, but only that the arrow was coming soon. In research, the center cue is often used as a control cue, because it attracts attention to a single location whilst the double cue keeps the attention spread over the two potential target locations. Lastly, a no cue condition existed during which only the fixation point was shown (Fan et al., 2002; PST Admin, n.d.).

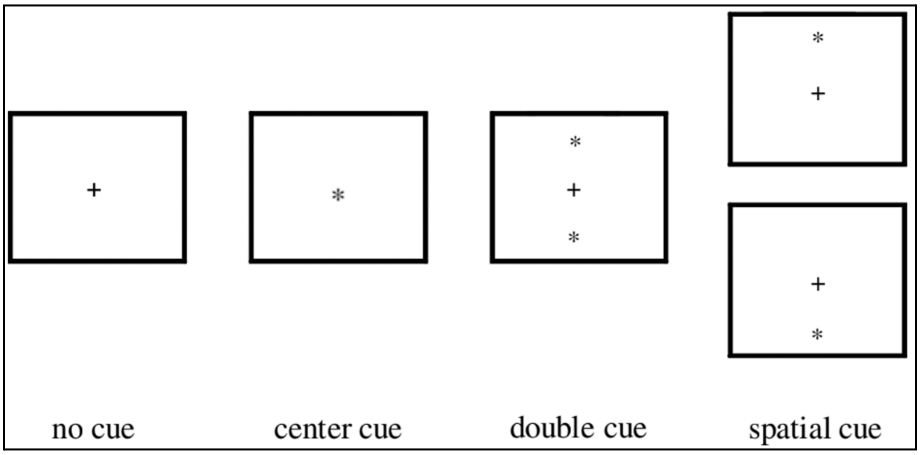
Figure 2

The Six Stimuli Options (A) and the Four Cue Types (B) used in the Attentional Network Test

A



B



Note. Adapted from Fan et al. (2002).

As can be seen in *Formula 1*, the alerting effect was obtained by calculating the difference between the mean RT of the no cue condition and the mean RT of the double cue condition. Upon presentation of these two cue types, no spatial information about the arrow location was provided. However, the alerting network reflected the improvement in RT due to the alerting effect of a nonspatial double cue compared to being shown no cue (Martella et al., 2011).

(1) $Alerting\ effect\ =\ mean\ RT_{no\ cue}\ -\ mean\ RT_{double\ cue}$

The spatial cue was the only cue type that provided information about the location of the upcoming target arrow, allowing the participants to orient their attention to the appropriate location in anticipation of the target arrow. Therefore, the difference between the mean RT of the center cue condition and the mean RT of the spatial cue condition resulted in the orienting effect (see *Formula 2*). Since both these cue types also served as an alerting cue, orienting to a certain location was the only degree of freedom here (Fan et al., 2002, 2009; Martella et al., 2011).

(2) $Orienting\ effect\ =\ mean\ RT_{center\ cue}\ -\ mean\ RT_{spatial\ cue}$

Upon presentation of an incongruent target stimulus, one had to first recognize the conflict between the direction of the central arrow and the flankers. Subsequently, one had to inhibit the action that was elicited by the flankers as they did not correspond to the correct answer. Upon presentation of a congruent target stimulus, no detection of a conflict or inhibition of the flanker information was needed. Therefore, the difference between the mean RT of the incongruent condition and the mean RT of the congruent condition resulted in the executive control functioning (see *Formula 3*). Note that the cue type did not play a role here (Martella et al., 2011).

(3) $Executive\ control\ effect\ =\ mean\ RT_{incongruent}\ -\ mean\ RT_{congruent}$

EEG

To record the EEG signal, a TMSi SAGA device with a 64 channel cap was used. EOG, EMG and ECG were recorded to facilitate artifact removal. A Mobi device enabled bipolar EOG and EMG signal recording. Bipolar ECG recordings were done using the TMSi SAGA device.

Measurements

ANT

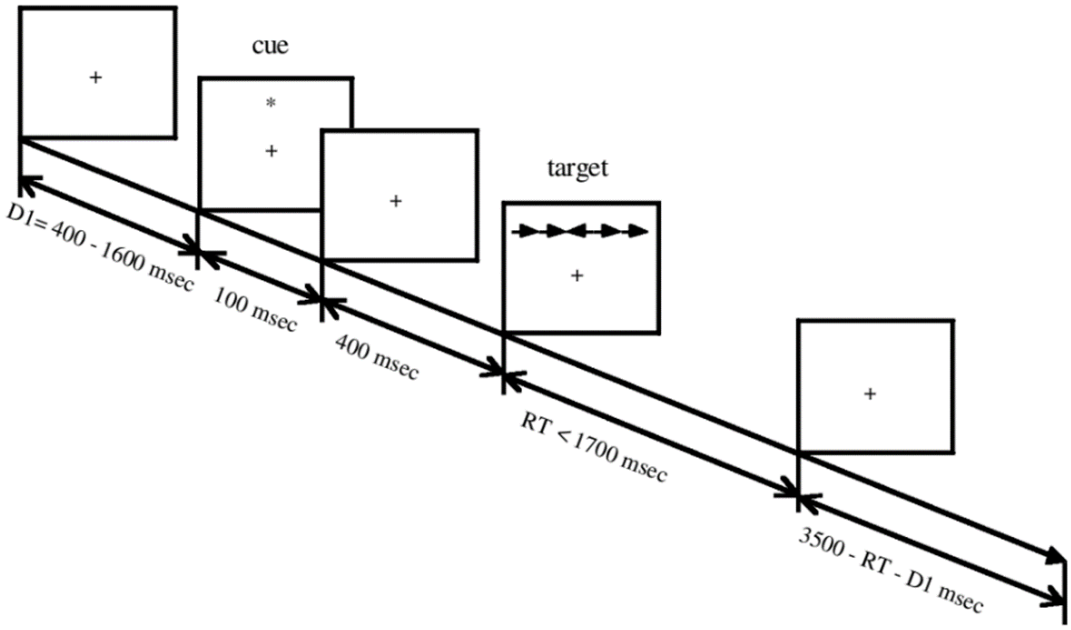
E-Prime software was used to program and administer the ANT. The ANT was programmed as described above and in accordance with the guidelines drawn up by Fan et al. (2002). The timeline of an ANT trial is shown in Figure 3 and always lasted 4000 ms. The fixation period before the cue was shown had a randomly varying duration (400 to 1600 ms). The cue was shown for 100 ms after which the fixation cross was presented for another 400 ms. During the no cue condition, participants saw the fixation cross for an extra 100 ms instead of a cue. Subsequently, the stimulus (called ‘target’ in Figure 3) was presented until the participant responded, but never longer than 1700 ms. Failing to answer within those 1700 ms was considered a lapse and included in the incorrect responses. After the participants’ response, the fixation cross appeared for a variable duration (3500 minus the duration of the first fixation period and minus the RT). Immediately after every trial, the next trial began. One full test lasted about 21 minutes and consisted of 24 practice trials and three blocks of 96 trials each. All stimulus and cue types were presented an equal amount of time. Furthermore, during half of the trials the stimulus appeared at the top of the screen and the other half at the bottom. The reaction times (RT’s in ms) as well as the responses (correct vs incorrect) were recorded.

EEG

The AFz was used as a ground and potentials were measured in reference to the electrode average. The sampling frequency was set at 4000 Hz and the electrode impedances were remained below 5kΩ. On acquisition, a highpass and lowpass filter of respectively 0 and 2000 Hz were applied. Bipolar EOG and EMG signals were recorded with a sampling frequency of 2048 Hz and an electrode behind the right ear was used as the ground.

Figure 3

Timeline of a Single ANT Trial.



Note. From "Testing the Efficiency and Independence of Attentional Networks," by J. Fan, 2002, *Journal of cognitive neuroscience, 14*(3), p. 341 (https://doi.org/10.1162/089892902317361886)

Data analysis

Pre-processing

Performance data

Fan et al. (2002) found no differences between left- and right-pointing target arrows, so they were combined in the analysis. The same yields for the stimuli being shown at the top or bottom of the screen. For the analysis of the ANT, which was done in Python, the practice trials were excluded. Accuracies were then calculated per participant, condition and stimulus type (averaged over cue type), per participant, condition and cue type (averaged over stimulus type) and per condition, stimulus and cue type (averaged over participant) as well as, per participant, overall accuracies per condition (averaged over stimulus and cue type). Note that responses slower than 1700 ms were considered lapses and thus considered incorrect responses. Further analysis was done only on correct trials with RTs longer than 100 ms. RTs shorter than 100 ms were excluded as this exceeds human performance limits and is most likely a result of a false start (Basner & Dinges, 2011; Dinges & Powell, 1985; Stojanoski et al., 2019; Yun et al., 2015). Outliers were removed using Cook's Distance with a threshold of 4/N, with N being the total number of data points. Subsequently, mean reaction times were calculated per participant and condition (averaged over stimulus and cue type), per condition, stimulus and cue type (averaged over participant), per condition and stimulus type (averaged over cue type and participant), and per condition and cue type (averaged over stimulus type and participant). Furthermore, per participant, mean reaction times were calculated for both sleep conditions.

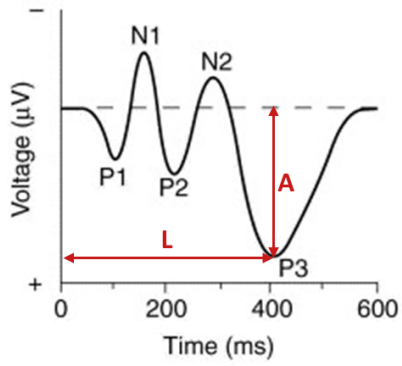
Lastly, the same data as used for the reaction time analysis was used for the analysis of the ANT network scores. These scores were computed according to Formulas 1-3 for every participant in both conditions.

Physiological data

The ERP analysis was done in Python using the MNE package. First, the EEG data was matched in time to the ANT trials. Then, EEG data corresponding to practice trials as well as incorrect responses was dropped. Data was re-referenced to the average of the mastoids. Blinks and eye movement artifacts were removed using Independent Component Analysis (ICA). Per dataset, ten components were fitted and manually compared to EOG and EMG plots. Components that were believed to reflect artifacts were removed from the EEG data. Subsequently, a highpass filter of 0.1 Hz and a lowpass filter of 45 Hz, both finite impulse response filters with zero-phase, were applied using MNE standard settings. EEG epochs were formed from 200 ms pre-stimulus onset to 800 ms post-stimulus onset. Baseline correction was applied by subtracting the mean of the baseline (200 ms pre-stimulus onset) from the entire epoch. Epochs with peak-to-peak signal amplitudes (PTPs) exceeding 100 μV were rejected. In other words, per epoch the PTP was calculated for every channel, when the PTP of at least one channel exceeds 100 μV, the respective epoch was rejected. Furthermore, epochs were manually viewed and epochs for which all electrodes contained a sudden change in signal amplitude or epochs containing signal drift were excluded, see Appendix E for examples of such epochs. ERP signals were obtained by averaging the EEG signal of every dataset in two groups: one containing the first 144 trials and the other containing the last 144 trials. This resulted in two ERP signals at every electrode site for all participants in both conditions.The electrode sites used were prefrontal (Fp1 and Fp2), frontal (F3 and F4), central (C3, Cz and C4), parietal (P3 and P4) and occipital (O1 and O2). Per ERP signal, the amplitude and latency of the five components of interest were computed as shown in Figure 4.

Figure 4

Computation of the Amplitude (A) and Latency (L) of the ERP Components.



Note. The P3 component is taken as an example here.

The P1 and N1 components are related to the alerting and orienting attentional networks (Williams et al., 2016). According to Rueda and Posner (2013), the frontal area, parietal area and thalamus are involved in the alerting attentional network whilst the frontal eye field, superior

parietal lobe, temporoparietal junction, pulvinar and superior colliculus are involved in the orienting attentional network. Since EEG can only record data from the cortex, the P1 and N1 components were analyzed at frontal and parietal electrode sites. Likewise, the N2 and P3 components are related to the executive control network (Williams et al., 2016). The anterior cingulate gyrus and the prefrontal cortex are involved in the executive control network (Rueda & Posner, 2013). Since the anterior cingulate gyrus is not part of the brain cortex, the N2 and P3 were analyzed at prefrontal electrode sites. Lastly, the P2 is likely not directly related to a certain attentional network, but based on previous sleep deprivation research, this component was analyzed at frontal electrode sites (Peng et al., 2020; Zhang et al., 2019).

The latency at which the five ERP components were expected and analyzed was based on previous research in the field of sleep restriction. The average of the latencies used by previous sleep restriction studies was used to determine the latency windows of the current study (see Table 5). However, if the components are not visible in their respective latency windows, they might be analyzed at a different latency.

Table 5

Latency Windows of Five ERP Components used in Current Study.

P1	N1	P2	N2	P3
50-200 ms	70-200 ms	130-250 ms	200-350 ms	250-450 ms

Note. (Gosselin et al., 2005; Hoedlmoser et al., 2011; Peng et al., 2020; Zeng et al., 2021; Zhang et al., 2019).

Statistical analysis

Performance data

As previously explained, sleep deprivation is expected to lead to slowed and worse performances on cognitive tasks like the ANT. Therefore, to check whether the sleep manipulation worked, two paired sample t-tests were performed. One to test the increase in reaction time and one to test the decrease in accuracy, both due to sleep restriction compared to normal sleep. Furthermore, three paired sample t-tests were performed to check whether the three ANT network scores were indeed higher in the sleep restriction condition compared to the normal sleep condition.

A 2 (sleep condition: normal sleep, restricted sleep) × 3 (stimulus type: neutral, congruent, incongruent) × 4 (cue type: center, double, spatial, no cue) repeated measures analysis of variance (ANOVA) was performed twice, once on the accuracy data and once on the reaction time data. These analyses were performed to inspect the effects of sleep condition on the differences in performance on the different trials (both stimulus and cue types and their interaction).

As explained before, the fatigue effect as observed during cognitive tasks becomes more pronounced after sleep loss compared to normal sleep conditions. Therefore, a multi-level analysis was performed to examine whether the impact of expired time during the ANT on the reaction times differed between the two sleep conditions, see Formula 4.

$$(4)\ Reaction\ time = sleep\ condition + trial\ number + trial\ number{:}\ sleep\ condition + (1|participant) + (1 + sleep|participant) + \varepsilon$$

Sleep condition (either normal sleep or restricted sleep) as well as trial number (1-288) were added as fixed effects to the model. The trial number by sleep condition interaction fixed effect was added as a predictor to the model to encompass the possibility of participants becoming slower over time when they were sleep deprived compared to them being not sleep deprived. The random effect of participant was added to predict the random intercepts due to inter-individual variability (i.e. some participants might overall be quicker than others). Lastly, a random slope was added to the model to predict the differences in how participants react to sleep restriction (i.e. participant A reacts differently to sleep restriction than participant B). This analysis was done in RStudio (version 4.2.1) using the lme4 and lmerTest packages (Bates, Maechker, Bolker, & Walker, 2015; Kuznetsova, Brockhoff, & Christensen, 2017). Initially an extra term was included in the model to explain the random effect of lab visit nested in participant ($1|participant{:}\ visit$). This term predicted the random intercept due to inter-individual variability (i.e. some participants might be quicker during the first lab visit whilst others might be quicker during the the second lab visit). Unfortunately, the model failed to converge because of this term and was thus left out for this analysis.

Lastly, two paired sample t-tests were performed to check whether a practice effect was present. To do so, the mean reaction times and accuracies per participant for the second lab session were compared to these results for the first lab session. Except for the muli-level model explained above, all other analyses on the performance data were done in Python using the Pingouin package version 0.5.1 and the Statsmodel package version 0.13.2.

Physiological data

Equivalence testing, more specifically two one-sided paired sample t-tests (TOST), was used to test Hypothesis 1, *partial sleep restriction will not lead to a change in P1 amplitude during the ANT compared to the normal sleep condition*. Ideally, the threshold between a negligible and useful effect (also called smallest effect size of interest) would be based on effect sizes from previous similar studies. However, unfortunately, such previous studies did not report effect sizes and did not use equivalence testing. Therefore, an equivalence interval [-δ, δ] was defined, based on the standardized mean differences (Cohen's d_z). According to a power analysis, the equivalence interval to achieve 80% power with a sample size of 4 was [-1.8, 1.8]. The null-hypothesis for the TOST was that the mean difference between the sleep conditions would fall outside this equivalence interval (i.e., $\mu_{normal\ sleep} - \mu_{restricted\ sleep} \leqq -\delta$ or $\mu_{normal\ sleep} - \mu_{restricted\ sleep} \geqq \delta$) and the alternative hypothesis was that the mean difference between the sleep conditions would fall within the equivalence interval (i.e., $\mu_{normal\ sleep} - \mu_{restricted\ sleep} > -\delta$ or $\mu_{normal\ sleep} - \mu_{restricted\ sleep} < -\delta$). This analysis was done in R-Studio using the TOSTER package version 0.4.2 (Lakens, 2017). The exact same statistical analysis was done to test Hypothesis 4 (*partial sleep restriction does not lead to a change in the latency of the P1 ERP components obtained during the ANT compared to the normal sleep condition)*.

To test Hypothesis 2 (*N1, N2 and P3 amplitude decreases due to partial sleep restriction compared to the normal sleep condition)* and 3 (*P2 amplitude increases due to partial sleep restriction compared to the normal sleep condition*), four separate multilevel models were made:

one for each ERP amplitude. An example of such a model for the N1 amplitude is shown in Formula 5. Sleep condition was a fixed factor with two levels: normal and restricted sleep. Due to the fatigue effect as explained before (Durmer & Dinges, 2005), the ANT performances were expected to decrease over the duration of the test. Since this could lead to amplitude and latency changes, time was included in the model as a dichotomous fixed factor with the levels first and last (144 trials). The time by sleep condition interaction effect was added as a fixed factor, because the amplitude could change differently over time when participants were sleep restricted compared to having slept normally. A random intercept of participant was added, because some participants might have a higher overall amplitude than others. Another random intercept was added for the laboratory visit nested in participant, because it could be that the amplitude was generally different during the two laboratory visits per participant. Moreover, a random slope for sleep was added to the model to account for the possibility of participants reacting differently to the sleep restriction.

(5) $$N1\ amplitude = sleep\ condition + time + time{:}\ sleep\ condition + (1|participant) + (1|participant{:}\ visit) + (1 + sleep|participant) + \varepsilon$$

To validate Hypothesis 5, another four multilevel models were made as explained above, but the dependent variable was the latency of the ERP components instead of the amplitude.

Unfortunately, there was not enough data to perform these mentioned multilevel analyses on the physiological data (see Results section for further explanation). Therefore, where possible, a repeated measures ANOVA was performed instead (this was for the P3 amplitude and latency). The repeated measures ANOVAs on the physiological data were performed in Python using Pingouin package version 0.5.

Since many different models were run on the same data, for every test an alpha level of 0.01 was used to reduce false positives.

Results

Performance data

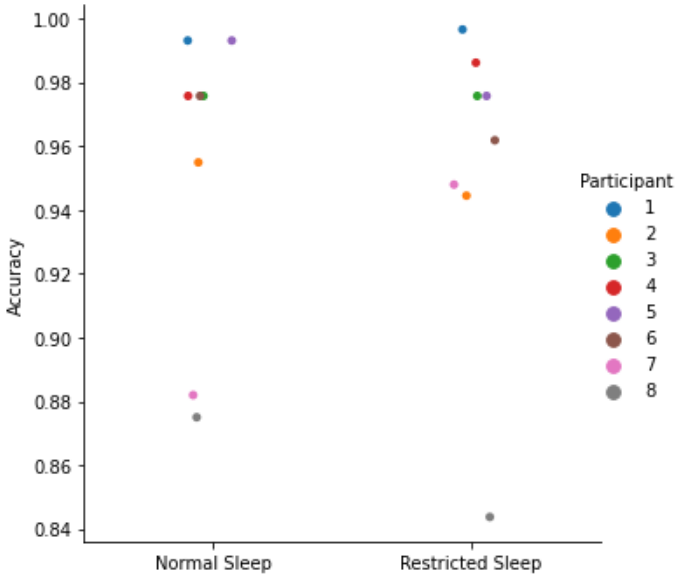
Three output variables of the ANT were analyzed namely the accuracy, reaction time and network scores.

Accuracy

For the remaining eight participants, the average accuracy was 0.95 (SD = 0.21). For both sleep conditions, the accuracies were calculated per participant and are shown in Figure 5. Whilst not clearly visible from this figure, these accuracies did not significantly deviate from a normal distribution, although the W-statistics are very low (W = 0.76, p = 0.012 for normal sleep and W = 0.77, p = 0.013 for restricted sleep). Results from a paired sample t-test showed that the accuracies for the restricted sleep condition (M = 0.95, SD = 0.048) were not lower than the accuracies for the normal sleep condition (M = 0.95, SD = 0.048), t(7) = -0.084, p = 0.532, with a very small effect size (d = 0.018).

Figure 5

Mean Accuracy per Participant for Both Sleep Conditions



The mean accuracies per stimuli and cue type were calculated for both sleep conditions and are presented in Appendix F, Table F1. Figure 6 shows the mean accuracies for the different cue types per stimulus type. Based on visual inspection, the accuracy for incongruent trials seems to be lower than for the neutral and congruent trials. No clear differences are visible between the two sleep conditions or between the four cue types. Sleep condition seems to have the most pronounced effect on the incongruent, double cue trials. A 2 (sleep condition: normal sleep, restricted sleep) × 3 (stimulus type: neutral, congruent, incongruent) × 4 (cue type:

center, double, spatial, no cue) repeated measures ANOVA was planned to verify this. However, due to a ceiling effect (see Appendix F, Figure F1) this analysis was not possible. This ceiling effect was least pronounced in the incongruent trials. Therefore an alternative 2 (sleep condition: normal sleep, restricted sleep) × 4 (cue type: center, double, spatial, no cue) repeated measures ANOVA was performed on the accuracy of the incongruent trials. The original non-transformed data was the closest to reaching normality for all cells and was therefore used in this analysis. All main and interaction effects were statistically non-significant (see Table 6). Interestingly, according to Cohen (1988, pp. 413-414), the effect size for the main effect of cue type was medium to large (η^2_G = 0.206) and the effect size for the sleep condition by cue type interaction was large (η^2_G = 0.297).

Figure 6

Mean Accuracy for the Four Cue Types per Stimulus type

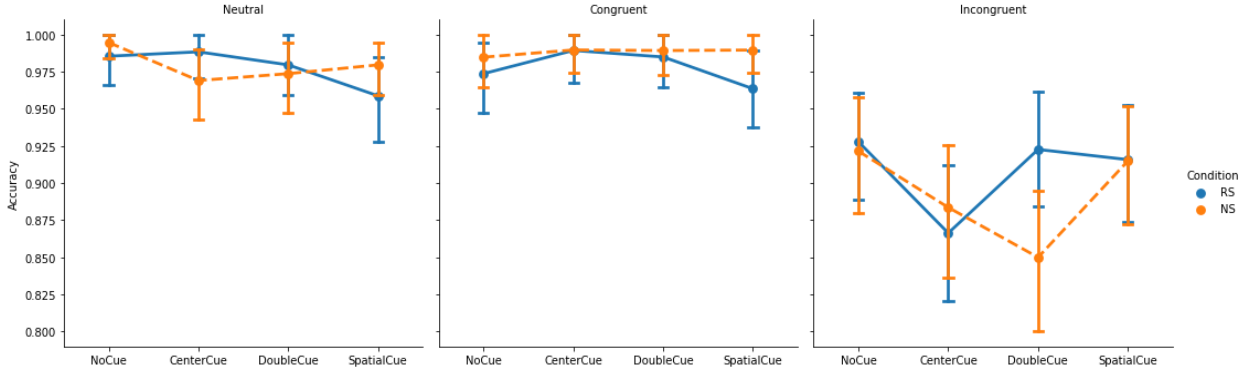


Table 6

Repeated Measures ANOVA on the Accuracy of the Incongruent Trials.

Factor	F-statistic	Numerator df	Denominator df	p-value	η^2_G
Sleep condition	0.521	1	7	0.494	0.069
Cue type	1.821	3	21	0.174	0.206
Sleep condition × Cue type	2.959	3	21	0.056	0.297

Note. df = degrees of freedom. η^2_G = generalized eta-squared.

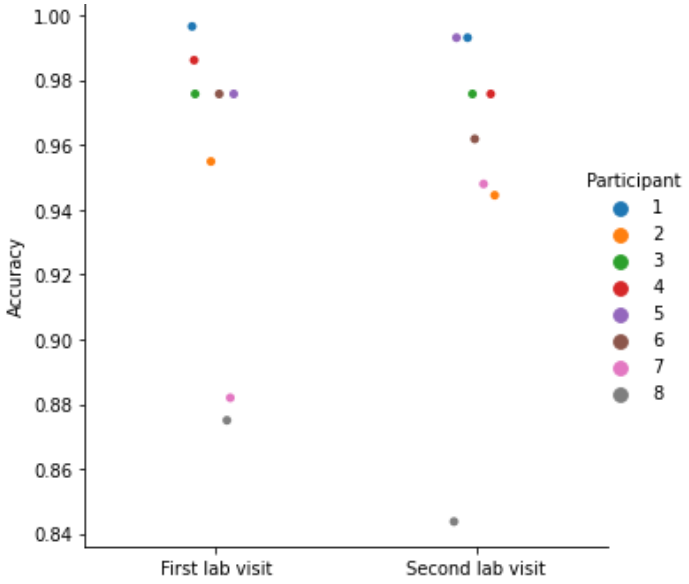
Practice effects

To check whether practice effects were present in the ANT, the mean accuracies were calculated per participant for their first and second lab visit, see Figure 7. The accuracies in Figure 7 do not seem to be normally distributed, but according to a Shapiro-Wilk test they did not significantly deviate from a normal distribution (W = 0.77, p = 0.015 for the first lab visit and W = 0.76, p = 0.010 for the second lab visit). A subsequent paired sample t-test showed participants were not more accurate in their second lab visit compared to their first, t(7) = -0.167,

p = 0.436, with a very small effect size (d = 0.036). These results indicate that no statistically significant practice effect was found on the accuracy of the ANT in the current study.

Figure 7

Mean Accuracy per Participant for Both Lab Visits



Reaction Times

None of the participants had a false start for any of the trials, so no trials were dropped due having a RT < 100ms. Outlier removal using Cook's Distance led to the removal of 4.19% of the correct trials. The mean reaction times were calculated per condition for each participant, see Figure 8. The mean reaction times were normally distributed for both the normal sleep (W = 0.96, p = 0.774) as well as the restricted sleep condition (W = 0.86, p = 0.126). Results from a paired sample t-test showed no statistically significant increase in the reaction times measured during the restricted sleep condition (M = 431.29, SD = 54.63) compared to the reaction times measured during the normal sleep condition (M = 409.82, SD = 35.32), t(7) = -2.031, p = 0.041, with a medium effect size (d = 0.467).

The mean reaction times per stimuli and cue type were calculated for both sleep conditions and are presented in Appendix F, Table F1. Figure 9 shows the mean reaction times for the different cue types per stimulus type. Based on visual inspection, a clear trend is visible for prolonged reaction times in the restricted sleep condition compared to the normal sleep condition. Furthermore, people seemed to answer the quickest to the spatial cue trials and the slowest to the no cue trials, regardless of stimulus type. Similarly, regardless of cue type, visual inspection suggests that people needed more time to answer correctly to incongruent trials compared to neutral and congruent trials. A 2 (sleep condition: normal sleep, restricted sleep) × 3 (stimulus type: neutral, congruent, incongruent) × 4 (cue type: center, double, spatial, no cue) repeated measures ANOVA was performed on response speed (1/RT transformed data) instead of reaction time to ensure normal distributions. The analysis resulted in statistically significant main effects for both stimulus ($F_{2,14}$ = 498.650, p < 0.001) and cue type ($F_{3,21}$ = 37.751, p <

0.001). Furthermore, the stimulus by cue type interaction effect was significant ($F_{6,42} = 5.299$, $p < 0.001$). All other interaction effects as well as the main effect for sleep condition were non-significant (see Table 7). Unfortunately, due to this analysis having three factors, effect size calculation in Python was not possible.

Figure 8

Mean Reaction Time per Participant for both Sleep Conditions

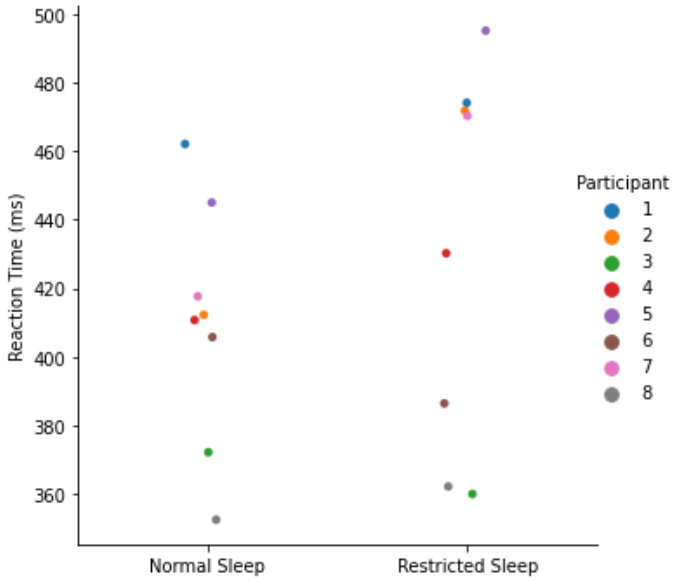


Figure 9

Mean Reaction Times for the Four Cue Types per Stimulus type

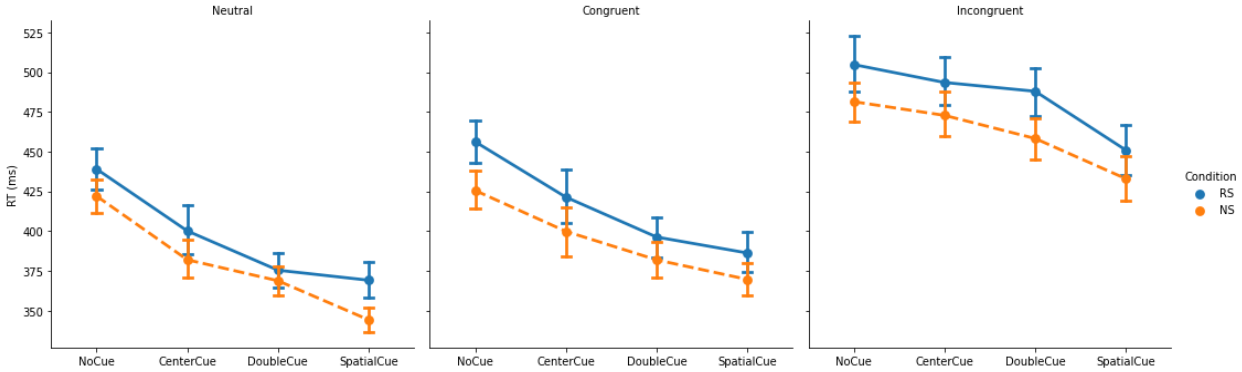


Table 7

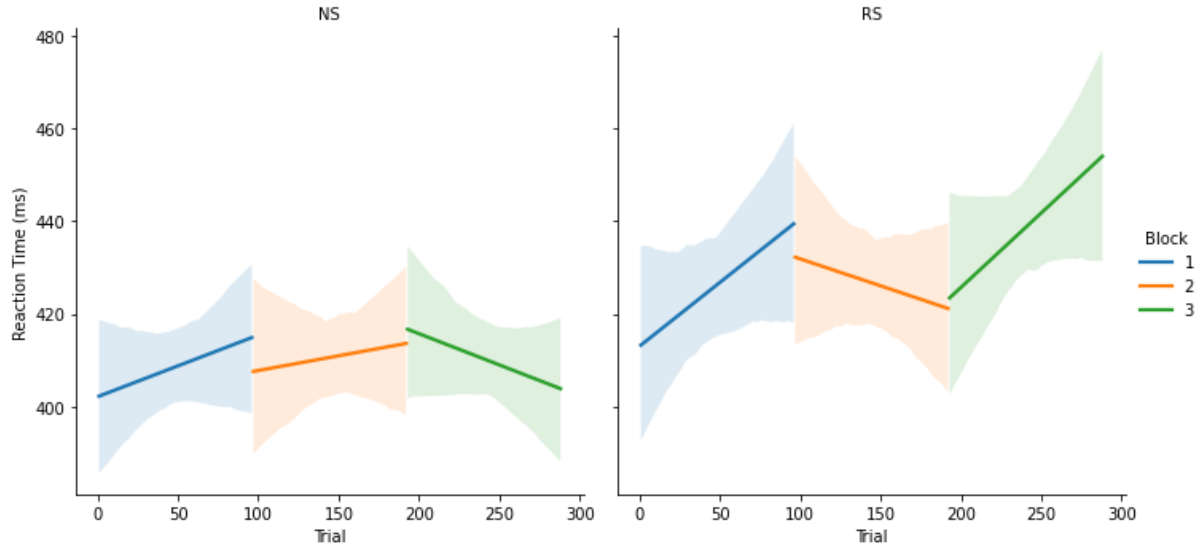
Repeated Measures ANOVA on the Reaction Time of the Incongruent Trials.

Factor	F-statistic	Numerator df	Denominator df	p-value
Sleep condition	2.384	1	7	0.167
Stimulus type	498.650	2	14	< 0.001
Cue type	37.751	3	21	< 0.001
Sleep condition × Stimulus type	1.233	2	14	0.321
Sleep condition × Cue type	0.621	3	21	0.610
Stimulus type × Cue type	5.300	6	42	< 0.001
Sleep condition × Stimulus type × Cue type	1.272	6	42	0.291

Note. df = degrees of freedom.

Figure 10

Mean reaction times over trial number for the three ANT blocks.



Note. Linear regressions are plotted with a 99% confidence interval.

Fatigue effect

Figure 10 shows how the reaction times (averaged over participants) change during the ANT. In this figure, the reaction times for the first and third block as measured during the restricted sleep condition seem to increase. However, when taking the confidence intervals into account, this effect is likely not statistically significant. To examine the impact of expired time during the ANT on the reaction times, a multilevel analysis was performed on the

log-transformed reaction time data (see Formula 4). No statistically significant main or interaction effects of sleep condition and trial number were found with this analysis (see Table 8). The variance of the random slope for sleep condition was very low (Var = 0.0009, SD = 0.03), however removing it from the model would worsen it (LRT(2) = 90.46, p < 0.001). This means the slope for the two sleep conditions variates between the participants, however, very little. These results suggest there was no indication of a fatigue effect in the reaction time data obtained in the current study.

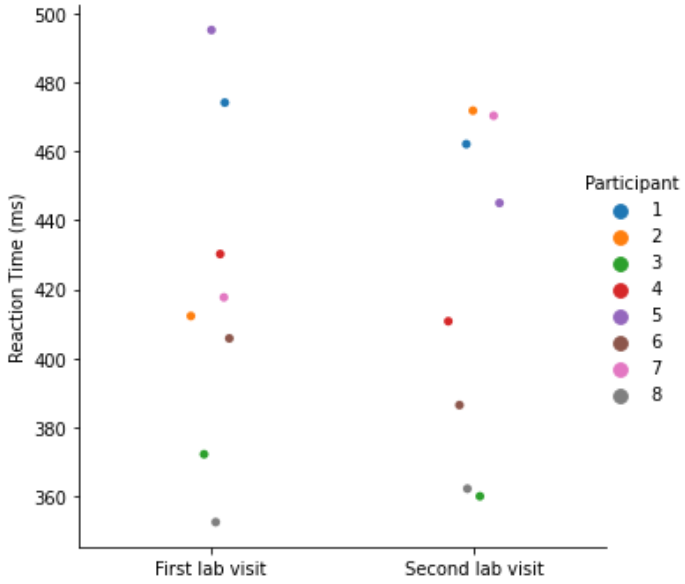
Table 8

Results of the fixed effects of the multilevel analysis on Reaction Time.

Predictor	Estimate	Standard error	Degrees of freedom	t-value	p-value
Intercept	2.600	0.014	7.853	188.897	< 0.001
Sleep condition	1.029e-2	0.012	9.794	0.877	0.402
Trial number	1.213e-5	< 0.001	4191	0.630	0.529
Sleep condition : Trial number	6.342e-5	< 0.001	4191	1.991	0.047

Figure 11

Mean reaction time per participant for both lab visits.



Practice effects

To check whether practice effects were present in the ANT reaction times, the mean reaction times were calculated per participant for their first and second lab visit, see Figure 11. For both the first and second lab visit, the mean reaction times were normally distributed

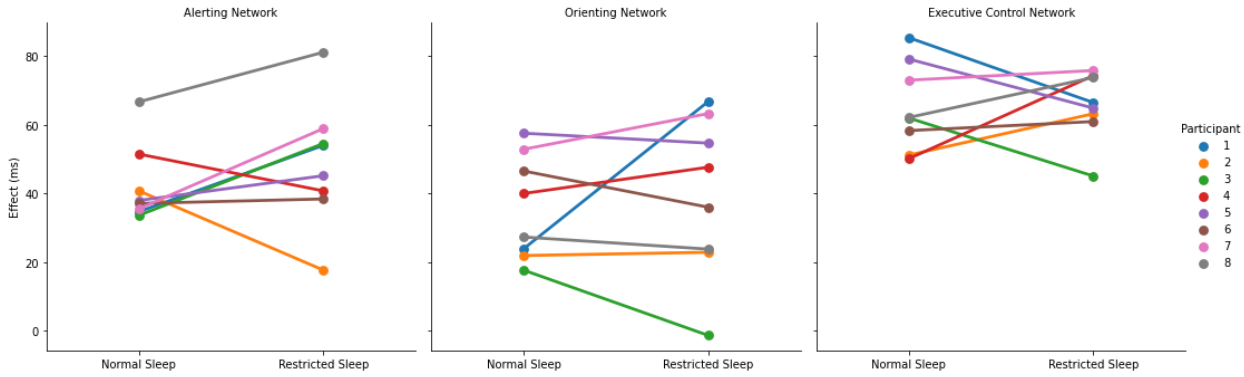
(respectively W = 0.96, p = 0.813 and W = 0.87, p = 0.148). Results from a paired sample t-test showed no statistically significant decrease in reaction time during the second lab visit (M = 421.09, SD = 47.38) compared to the first lab visit (M = 420.01, SD = 47.43), t(7) = -0.082, p = 0.531, with a very small effect size (d = 0.023). These results, together with the results of the accuracy, indicate there was no evidence for a practice effect.

Attentional Networks

Figure 12 shows the network scores per participant for both sleep conditions. For all three networks, the varying intercepts and varying slopes of the lines indicate a strong inter-individual variability. Based on visual inspection, no clear pattern regarding the difference between the two sleep conditions can be observed. The alerting network effect of participant 8 is for both sleep conditions larger than for the other participants. This indicates participant 8 gained the most reaction time improvement due to the alerting effect of a nonspatial double cue compared to being shown no cue. Upon inspection of the orienting network effects in Figure 12, a clear example of the varying slopes is visible between participant 1 and 3. Participant 1 has an increasing slope meaning they gained more improvement in reaction time, due to spatial cues allowing orientation of their attention to the location where the target arrow would appear, during the restricted sleep condition compared to the normal sleep condition. For participant 3 the opposite applies, for this participant the orienting network effect was smaller in the sleep restriction compared to the normal sleep condition. Furthermore, participants could have an increasing score for one network whilst having a decreasing score for another. An example of this is participant 3 for the alerting and orienting network, respectively. This participant gained more from the alerting effect of a nonspatial double cue during the restricted sleep condition than during the normal sleep condition. However, as seen in this participant's orienting scores, they gained more from the spatial orienting cues during the normal sleep condition than during the restricted sleep condition.

Figure 12

Change in the Alerting, Orienting and Executive Control Network scores from the Normal Sleep to the Restricted Sleep Condition.



To check whether the alerting scores were higher for the restricted sleep condition than for the normal sleep condition, a paired sample t-test was performed, because two Shapiro-Wilk

tests showed the alerting scores for the normal sleep condition (W = 0.76, p = 0.011) as well as for the restricted sleep condition (W = 0.97, p = 0.87) did not significantly differ from a normal distribution. The paired sample t-test showed the alerting scores were not significantly higher during the restricted sleep condition (M = 48.85, SD = 18.35) than during the normal sleep condition (M = 42.26, SD = 11.37), t(7) = -1.126, p = 0.149, with a medium effect size (d = 0.432).

Regarding the orienting network, scores for both the normal and restricted sleep conditions were normally distributed (W = 0.91, p = 0.376 and W = 0.95, p = 0.684, respectively). The orienting network score was found to have a mean of 39.25 ms (SD = 23.32ms) during the restricted sleep condition, and a mean of 36.01 ms (SD = 15.26 ms) during the normal sleep condition. A paired sample t-test revealed that no significant increase in the orienting network scores was observed, t(7) = -0.494, p = 0.318, with a small effect size (d = 0.164).

Since the executive control network scores for the two groups were normally distributed (W = 0.930, p = 0.518 for normal sleep and W = 0.880, p = 0.188 for restricted sleep), a paired sample t-test was performed to check whether the scores for this network were higher for the restricted sleep condition than for the normal sleep condition. No significant evidence was found to substantiate an higher executive control network score for restricted sleep compared (M = 65.58, SD = 9.98) to normal sleep (M = 65.17, SD = 12.83), t(7) = -0.074, p = 0.472, with a very small effect size (d = 0.036).

Physiological data

P1 component

Frontal ERP waveform

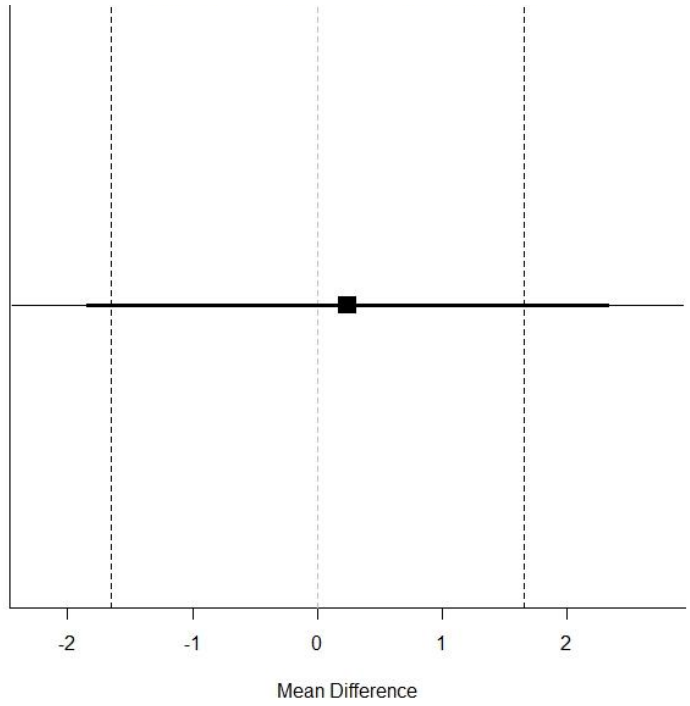
Figure 13 shows the frontal ERP waveforms for all eight participants in both sleep conditions. Similar graphs including ERPs for the two time groups (first and second 144 trials) are visualized in Appendix H, Figure H1. ERP components P1, N1 and P2 are expected to be most pronounced at frontal brain sites. However, between 50 and 130 ms in Figure 13, clear P1 components are only visible in the graphs for participants 2 and 5. The descriptive statistics of the P1 amplitudes and latencies for these two participants are shown in Appendix F, Table F2. Unfortunately, the data from these two participants is not enough to perform a statistical test to investigate the effect of sleep condition on the frontal P1 amplitude or latency.

Parietal ERP waveform

Figure 14 shows the parietal ERP waveforms for all eight participants in both sleep conditions. Again, similar graphs including the ERPs for the two time groups can be found in Appendix H, Figure H2. Both the P1 and N1 components are expected to be present in the parietal brain area. Only for participants 2, 3, 5 and 7 the frontal P1 component is visible between 50 and 130 ms. The amplitude of this component for participant 7 is very small and could also be noise, coincidentally at the correct place in the plot, instead of a P1 component. These small amplitudes can also be seen in Appendix F, Table F3 which shows the descriptive statistics of the parietal P1 amplitudes and latencies for these four participants.

Figure 16

Difference between Mean Parietal P1 Amplitudes in Normal Sleep and Restricted Sleep.



Note. The thick horizontal line indicates the 99% confidence interval from the TOST ([-1.845; 2.330]), the light dashed vertical line indicates the null hypothesis, and the dark dashed vertical line indicates the equivalence bounds in raw scores (-1.655 and 1.655). The black square indicates the mean difference which was 0.242.

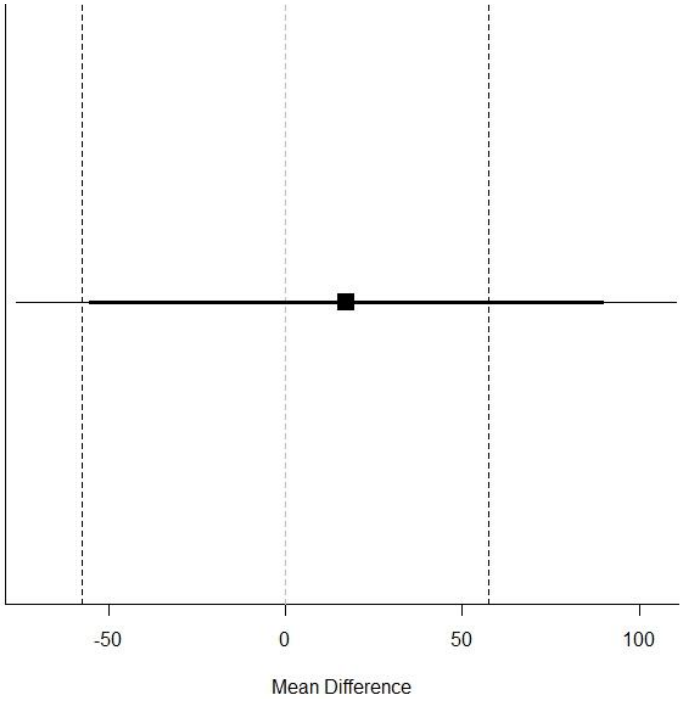
Equivalence testing was done to test hypothesis 1, *partial sleep restriction will not lead to a change in P1 amplitude during the ANT compared to the normal sleep condition*. The difference between means in the normal (M = 2.17, SD = 0.67) and restricted sleep condition (M = 1.93, SD = 0.86) is shown in Figure 16. The correlation of the parietal P1 amplitudes between the two sleep conditions was 0.30. The equivalence test was non-significant, t(3) = -3.07, p = 0.027, given equivalence bounds of -1.655 and 1.655 (on a raw scale). Since the null-hypothesis cannot be rejected, there is no evidence for the parietal P1 amplitude in the normal sleep condition being statistically equivalent to the parietal P1 amplitude in the restricted sleep condition. Upon inspection of the means, the parietal P1 amplitude seems to be larger for the normal sleep condition compared to this amplitude for the restricted sleep condition. However, there is no statistical evidence for this possible trend. A repeated measures ANOVA on the parietal P1 amplitude was also performed (see Appendix G).

To test hypothesis 4, *partial sleep restriction does not lead to a change in the latency of the P1 ERP components obtained during the ANT compared to the normal sleep condition*, another equivalence test was done. The difference between means in the normal (M = 102.63, SD = 7.98) and restricted sleep condition (M = 85.50, SD = 24.30) is shown in Figure 17. The correlation of the parietal P1 latencies between the two sleep conditions was -0.94. The equivalence test was non-significant, t(3) = -2.52, p = 0.043, given equivalence bounds of

-57.502 and 57.502 (on a raw scale). This indicates there is no evidence for the parietal P1 latency in the normal sleep condition being statistically equivalent to the parietal P1 latency in the restricted sleep condition. Upon inspection of the means, the parietal P1 latency seems to be larger for the normal sleep condition compared to the latency in the restricted sleep condition. However, there is no statistical evidence for this possible trend.

Figure 17

Difference between Mean Parietal P1 Latencies in Normal Sleep and Restricted Sleep.



Note. The thick horizontal line indicates the 99% confidence interval from the TOST ([-55.403; 89.653]), the light dashed vertical line indicates the null hypothesis, and the dark dashed vertical line indicates the equivalence bounds in raw scores (-57.502 and 57.502). The black square indicates the mean difference which was 17.125.

N1 and P2 component

For all participants, no frontal N1 or P2 components are visible in Figure 13 during their respective expected latency windows (70-200 ms and 130-250 ms, respectively). Similarly, the N1 component is not visible between 70 and 200 ms after stimulus onset. Therefore, no multilevel analysis could be performed to validate hypothesis 2 and 3 regarding the amplitude of the N1 and P2 component, respectively. For the same reason, hypothesis 5 regarding the N1 and P2 latency could not be validated.

N2 component

The N2 and P3 ERP components are expected to be present at the prefrontal brain area. Figure 15 shows the prefrontal ERP waveforms of all participants in both conditions; a

more detailed figure with the two time groups included can be found in Appendix H, Figure H3. For participant 6, the prefrontal ERP waveform from the normal sleep condition seems to be considerably more negative compared to this waveform for the restricted sleep condition as well as compared to waveforms from other participants. Something similar can be seen for this participant in the frontal ERP waveform (see Figure 13F). For none of the participants, a clear prefrontal N2 component is visible between 200 and 350 ms after stimulus onset.

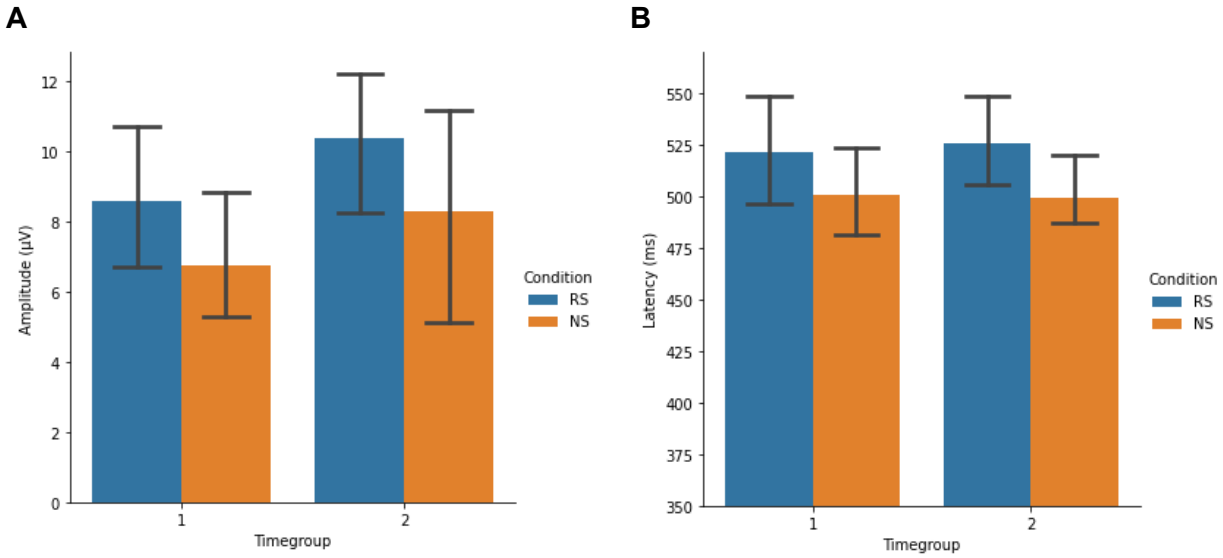
Since the P3 component happens at a larger latency than expected (see explanation below), the N2 might also happen later than expected. It could be that the negative dip before the signal amplitude increases (i.e. right before 450 ms), represents the N2. Clear N2 components are visible in the frontal ERP waveforms for participants 1, 3, 5 and 6 (see Figure 13A, C, E and F) as well as in the parietal ERP waveforms for participants 1, 2, 3, 5 and 6 (see Figure 14A, B, C, E and F) and prefrontal ERP waveforms for participants 1, 3 and 5 (see Figure 15A, C and E). Since it is unlikely that the N2 component is more distinguishable than the P3 component, not all of these negative peaks are likely N2 components. The negative parietal peak around 500 ms after stimulus onset for participant 1 (see Figure 14A) is, for example, likely not a N2 component as its amplitude is large (around 7 to 11 μV) and it does not precede a P3 component. Similarly, the negative frontal peak preceding the P3 component for participant 6 (see Figure 13F) is likely not a N2 component since its amplitude is larger than the P3 amplitude which is unlikely. Therefore, it is unfortunately not possible to classify any of these negative peaks as the N2 component. This means that hypothesis 2 regarding the N2 amplitude as well as hypothesis 5 regarding the N2 latency could not be validated.

P3 component

No clear prefrontal P3 components are visible in Figure 15 between 250 and 450 ms after stimulus onset. For most participants, however, the prefrontal signal amplitude increases after about 450 ms, this is best visible for participant 4 (see Figure 15D). Such late peaks are also visible in certain frontal and parietal plots, e.g. the frontal and parietal plots for participant 7 (see Figure 13G and Figure 14G, respectively). These peaks with positive amplitudes exceeding 5 μV are expected to be P3 components, even if they appear later than 450 ms after stimulus onset. In the frontal ERP waveforms these P3 components are best visible (see Figure 13). In other brain regions, there are amplitudes exceeding 5 μV as well, but those waveforms do not form peaks as the expected P3 would. For example, the parietal ERP waveform for participant 2 (see Figure 14B) shows a clear increase in amplitude after 450 ms, however, the signal amplitude stays increased (around 9 to 10 μV) for a prolonged time (i.e. 250 ms) instead of going back to the baseline of the signal which is around 0 μV. Therefore, this ERP waveform, and waveforms that look similar, are not classified as P3 components. The frontal ERP waveforms of participant 2, 4, 6 and 7 (see Figure 13B, D, F and G, respectively) as well as the parietal ERP waveform of participant 7 (see Figure 14G) and the prefrontal ERP waveform of participant 4 (see Figure 15D) are considered to include P3 components as their amplitude exceeds 5 μV after 450 ms and decreases again within 100 ms. Since most P3 components are visible in the frontal ERP waveforms, it was decided to statistically analyze the P3 component at frontal sites for only four participants. Interestingly, all ERP waveforms from the frontal brain region increase after 450 ms, however, the amplitude, latency and duration of this increase differs substantially per participant.

Figure 18

Mean Amplitude and Mean Latency of Frontal P3 Components for Participants 2, 4, 6 and 7.



Note. **A** Mean amplitude. **B** Mean latency. Y-axis in **B** starts at 350 ms. Error bars indicate the 99% confidence intervals. NS = normal sleep. RS = restricted sleep.

Frontal P3 amplitude.

The mean amplitude and latency of the frontal P3 component are visualized in Figure 18. Regardless of time group, all bars for the restricted sleep condition are higher than for the normal sleep condition. To test hypothesis 2 regarding the P3 amplitude (*P3 amplitude decreased due to partial sleep restriction compared to the normal sleep condition*) a multi-level model was planned. Unfortunately, not enough data was acquired to do so and a 2 (sleep condition: normal sleep, restricted sleep) × 2 (time: first 144 trials, second 144 trials) repeated measures ANOVA was performed on the frontal P3 amplitudes for participants 2, 4, 6 and 7 instead. Both main effects as well as the interaction effect were not statistically significant (see Table 9). However, the main effect for sleep condition was close to reaching significance ($F_{1,3}$ = 21.679, p = 0.019, η^2_G = 0.183), which indicates a trend towards higher P3 amplitudes after 3 consecutive days of partial sleep restriction compared to normal sleep. Interestingly, the effect sizes for both main effects are, according to Cohen (1988, pp. 413-414), medium.

Frontal P3 latency.

To check hypothesis 5 regarding the P3 (*partial sleep restriction will lead to an increased latency of the P3 ERP component obtained during the ANT compared to the normal sleep condition*), a 2 (sleep condition: normal sleep, restricted sleep) × 2 (time: first 144 trials, second 144 trials) repeated measures ANOVA was performed on the frontal P3 latencies for participants 2, 4, 6 and 7. Again, this was done instead of a multi-level analysis because of the deficit in the acquired data. Both main effects as well as the interaction effect were non-significant (see Table 10). The effect size for the main effect of sleep condition is, however, large (η^2_G = 0.263).

Table 9

Results Repeated Measures ANOVA on Frontal P3 Amplitude of Participants 2, 4, 6 and 7.

Factor	F-statistic	Numerator df	Denominator df	p-value	η^2_G
Sleep condition	21.679	1	3	0.019	0.183
Time	3.092	1	3	0.177	0.143
Sleep condition × Time	0.031	1	3	0.872	0.001

Note. df = degrees of freedom. η^2_G = generalized eta-squared.

Table 10

Results Repeated Measures ANOVA on Frontal P3 Latency of Participants 2, 4, 6 and 7.

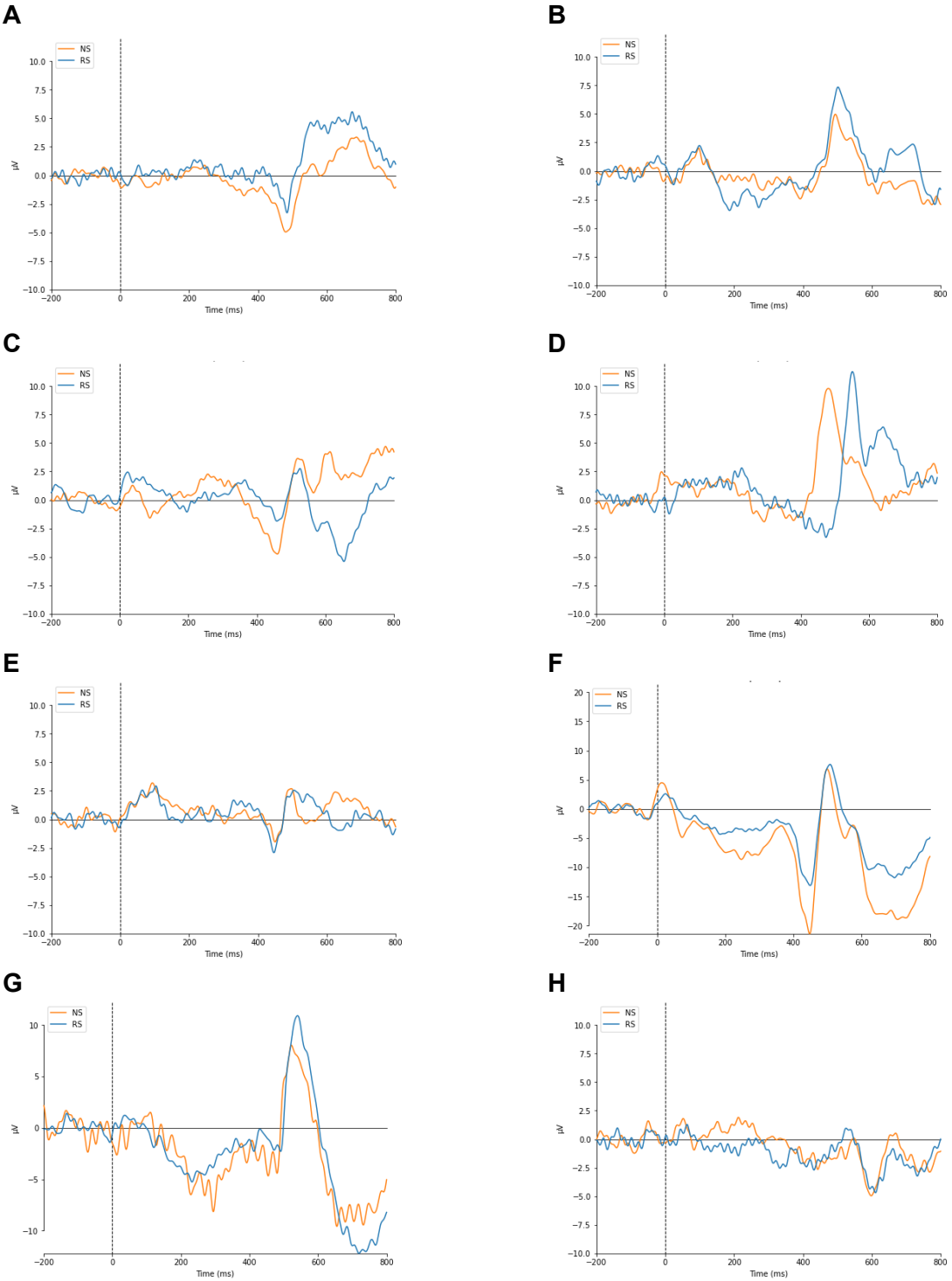
Factor	F-statistic	Numerator df	Denominator df	p-value	η^2_G
Sleep condition	2.266	1	3	0.229	0.263
Time	0.147	1	3	0.727	0.001
Sleep condition × Time	1.821	1	3	0.270	0.005

Note. df = degrees of freedom. η^2_G = generalized eta-squared.

Although not used for analysis, the plots of the central ERP waveforms are shown in Appendix H, Figure H4 and H5. In these plots, some of the mentioned ERP components are visible. However, less clear as for the electrode sites analyzed above. Therefore, no additional analysis was performed on the central ERP waveforms. Regarding the occipital waveforms, the O1 and O2 channels contained a lot of noise resulting in the deletions of sometimes more than half of their epochs. This eventually led to not enough epochs being left over to obtain ERP waveforms, therefore they were not visualized.

Figure 13

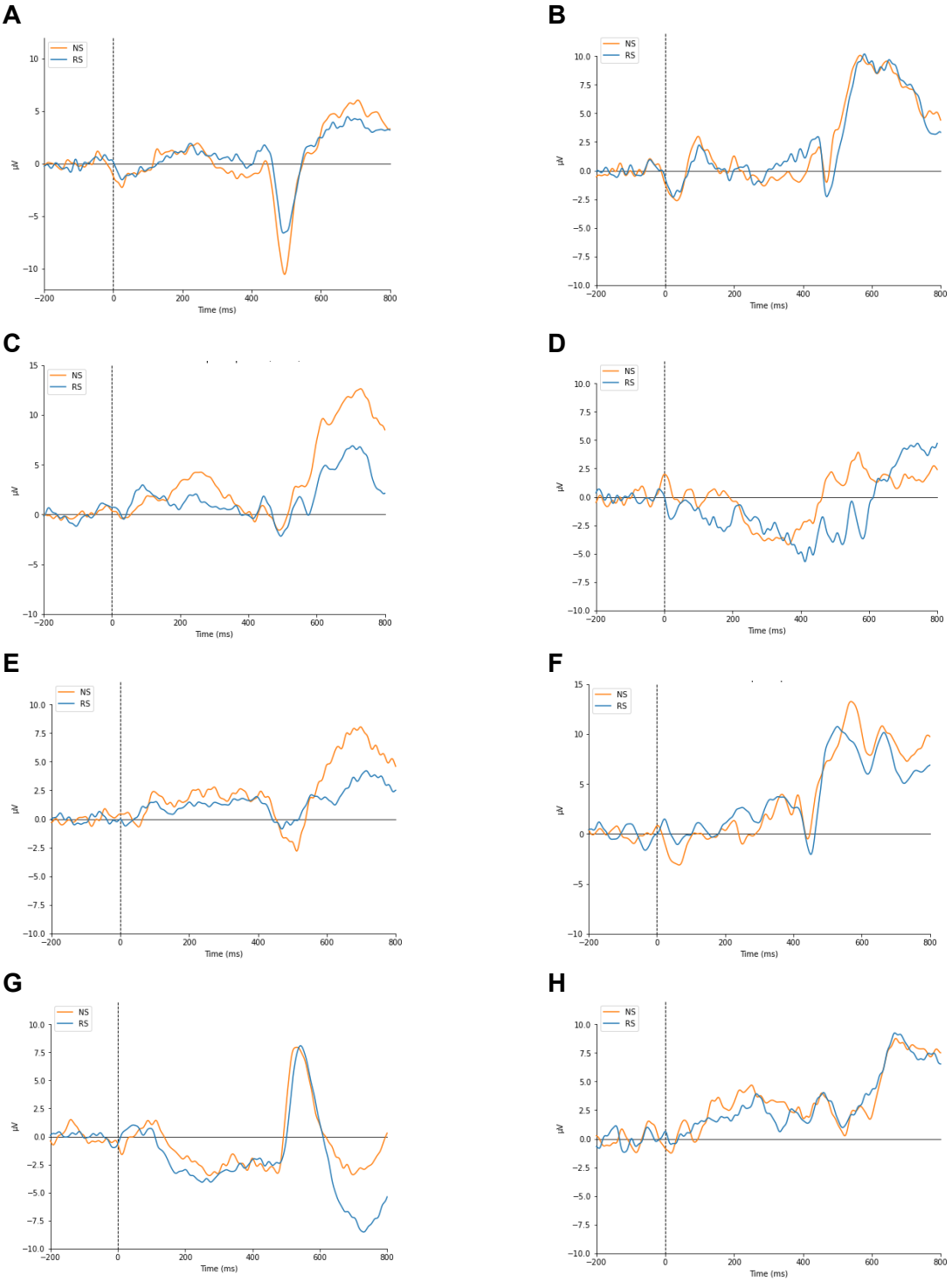
Frontal ERP Waveforms for Participant 1-8 for both Sleep Conditions.



Note. **A** - **H** represent the frontal ERPs of, respectively, participants 1 - 8. NS = normal sleep. RS = restricted sleep. Y-axes for the plots regarding participants 6 and 7 are different from the other plots which have a y-axis running from -10 to 10 μV. Dashed vertical line represents stimulus onset time.

Figure 14

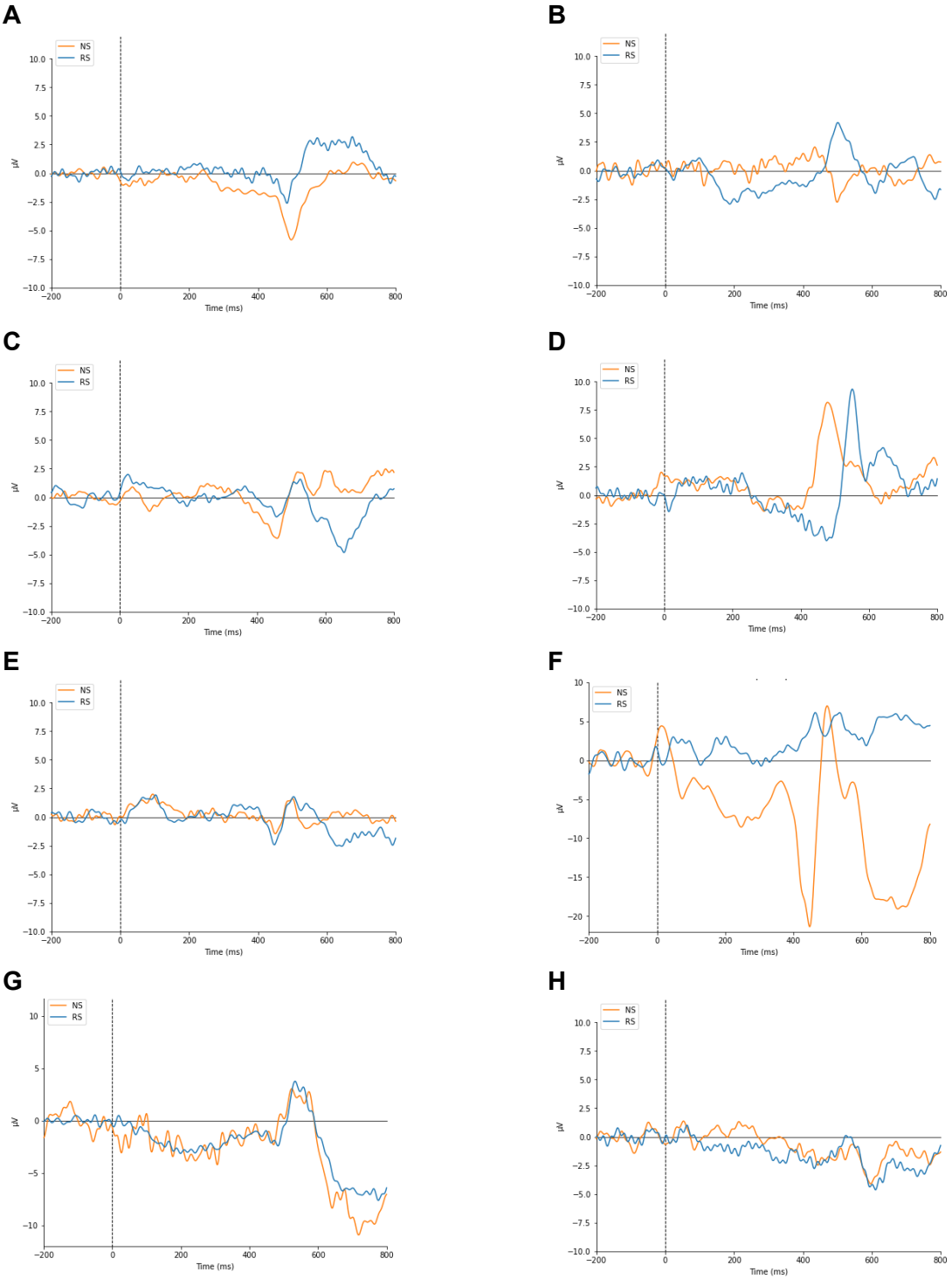
Parietal ERP Waveforms for Participant 1-8 for both Sleep Conditions.



Note. **A** - **H** represent the parietal ERPs of, respectively, participants 1 - 8. NS = normal sleep. RS = restricted sleep. Y-axes for the plots regarding participants 1, 3 and 6 are different from the other plots which have a y-axis running from -10 to 10 μV. Dashed vertical line represents stimulus onset time.

Figure 15

Prefrontal ERP Waveforms for Participant 1-8 for both Sleep Conditions.



Note. **A** - **H** represent the prefrontal ERPs of, respectively, participants 1 - 8. NS = normal sleep. RS = restricted sleep. Y-axes for the plots regarding participants 6 and 7 are different from the other plots which have a y-axis running from -10 to 10 μV. Dashed vertical line represents stimulus onset time.

Discussion

This study aimed to investigate the effects of sleep loss on brain activity as well as attention. More specifically, the reaction of the brain, quantified as ERP components, to stimuli presented as part of the ANT after partial sleep restriction or non-restricted sleep. To this end, a laboratory session was scheduled during which brain activity was monitored during the ANT after three consecutive nights of sleep restricted to 4 hours vs. a habitual sleep episode of 8 hours. The knowledge gained in this study could be applied to investigate monitoring tools to enable the quantification of the effects of sleep loss, specifically the effects on attention, but also inform the development of interventions to support people struggling with the consequences of sleep loss during the wake phase.

Previous research showed sleep loss leads to attentional deficits which in turn causes a decrease in cognitive performance (Durmer & Dinges, 2005; Martella et al., 2011). One manner to quantify attentional deficits due to sleep loss is with the ANT. It has been shown that accuracies decrease, reaction times increase and the attentional network scores increase as well due to sleep restriction imposed on participants (Jugovac & Cavallero, 2012; Martella et al., 2011; Riontino & Cavallero, 2022; Roca et al., 2012). A second manner of quantifying attentional deficits due to sleep loss is with ERP components. Previous studies showed the amplitude and latency of such components change due to sleep loss. Interestingly, ERP components are thoroughly studied in relation to the ANT, but not in relation to the ANT during periods of sleep loss. To make up for this deficiency in literature the current study focused on the effects of sleep loss on ERP components as measured during the ANT.

Performance (ANT)

Accuracy

On average, the accuracy scores for the ANT found in the current study were similar to ones of previous studies (Martella et al., 2011; Riontino & Cavallero, 2022; Roca et al., 2012). Previous articles found people performing the ANT after sleep restriction make more mistakes than people who slept normal 8 hour nights (Jugovac & Cavallero, 2012; Riontino & Cavallero, 2022; Roca et al., 2012). Therefore, the participants in the current study were expected to reach a lower mean accuracy after partial sleep restriction compared to normal sleep. However, the result does not show a significant trend and was only very small in effect size. These results are surprising considering Riontino & Cavallero (2022) as well as Roca et al. (2012) found very similar mean accuracy values, but also found decreased accuracies with medium to very large effect sizes (η^2_G = 0.11 and η^2 = .60, respectively). When inspecting the data, no ceiling effect is visible, so this could not be the reason for the unexpected results.

Reaction Times

Regarding reaction times, compared to previous research, participants in the current study reacted very fast, independent of the sleep condition. Compared to the current study, previous research found reaction times of at least 100 ms slower and often even 150 to 200 ms

slower. Indeed, Martella and colleagues (2011) found, for the exact same ANT as applied in the current study, mean reaction times were approximately 100 ms longer than present results across baseline and after 24 hours of TSD. Similarly, Roca and colleagues (2012) found reaction times for both the normal sleep condition and after 25.5-27.5 hours of TSD to be around 200 ms slower than those in the current study. Furthermore, Riontino and Cavallero (2022) performed a revised ANT (ANT-R) and found higher reaction times as well. For ANT-R trials with the same cue-stimulus interval as applied in the current study (namely 400ms) and not including invalid cues, they found a mean reaction time of 530 ms for the baseline sleep condition and a mean reaction time of 573 ms after 24 hours of TSD. Lastly, participants in the study by Fan et al. (2002) performed the ANT under normal sleep conditions and reached a mean reaction time of 513 ms.

This difference between the current and previous results could be due to the participants being younger or more intelligent than the participants in the other studies. However, this is unlikely as the participants in the studies of Martella et al. (2011), Riontino and Cavallero (2022) and Roca et al. (2012) were also students with similar mean age and probably similar IQ's since they were all recruited from universities. Since the accuracies found in the current study were similar to those found in previous research, the lower reaction times cannot be due to a more pronounced speed-accuracy tradeoff for the current study compared to other studies. Since both the current and previous studies used (a version of) the ANT that was programmed in accordance with the guidelines drawn by Fan et al. (2002), the test itself could not have led to the difference in reaction times found. A possible explanation, however unlikely to have such an effect, could be that the researchers performing the current study may have emphasized more to the participants the need to react quickly. Lastly, a calibration error of the measurement equipment (i.e. the laptop used to administer the ANT) could be a feasible explanation for the reaction time difference. For future research with the same equipment, it is advised to run a few trial sessions comparing the current equipment to equipment that is known to be well calibrated. By checking the calibration of the measurement equipment, the fast results of the current study can be validated.

Previous research has shown increased ANT reaction times after 24 hours of TSD compared to normal sleep (Jugovac & Cavallero, 2012; Martella et al., 2011; Riontino & Cavallero, 2022; Roca et al., 2012). Statistical values of the current study show a non-significant trend in the expected direction with a medium effect size that could potentially become significant if the sample size would have been larger. As for the ANT accuracy, the effect of sleep restriction on ANT reaction time was expected to be present based on previous studies applying 24 hours of TSD. The current study, with its limitations, fails to provide evidence that the same effect can be found after three consecutive days of partial sleep restriction.

No statistically significant fatigue effect was found in the reaction time data of the current study which means participants did not become slower as the ANT prolonged, the effect size was extremely low. Luckily, no indications of practice effects were found for either accuracy or reaction time.

Attentional Networks

Regarding the three networks of the ANT, no significant increase in network scores were found due to sleep restriction compared to normal sleep. These results are not in line with the expected increase in network scores due to sleep restriction compared to normal sleep, based on previous findings that applied the ANT in sleep restriction research (Jugovac & Cavallero, 2012; Martella et al., 2011; Riontino & Cavallero, 2022; Roca et al., 2012). The sample size of the current study was very low leading to a higher chance of obtaining non-significant results. However, when inspecting the results by Jugovac and Cavallero (2012), non-significant effects were found for the alerting and orienting network. Likewise, Roca and colleagues (2012) found non-significant effects for the orienting and executive control network. These studies used, respectively, 30 and 26 participants, so even with larger sample sizes, changes in the network scores were not always found.

Interestingly, a very small effect size was found for the paired sample t-test regarding the executive control network scores, indicating that the effect of sleep on this network was very small. This is in contrast with multiple previous studies that found an effect of sleep restriction on this network (Jugovac & Cavallero, 2012; Martella et al., 2011; Riontino & Cavallero, 2022). Contrastingly, a medium effect size was found for the effect of sleep restriction on the alerting network scores whilst for only two previous studies significant effects were found of sleep restriction on this network score (Riontino & Cavallero, 2022; Roca et al., 2012).

As explained for participant 3, but also visible for other participants, sleep restriction could have different and even opposite effects on the different attentional network scores. The increase in alerting and decrease in orienting score for the restricted sleep compared to the normal sleep as seen for participant 3, could be a result of compensatory mechanisms. The attentional brain network involved in alerting (i.e. frontal area, parietal area and thalamus) might be over active to compensate for the reduced function of the brain network involved in orienting (i.e. frontal eye field, superior parietal lobe, temporoparietal junction, pulvinar and superior colliculus) (Rueda & Posner, 2013). This compensatory behavior of the brain is a perfect example of the dependence of cognitive performances on compensatory brain mechanisms as explained by both Durmer & Dinges (2005) and Krause et al. (2017).

Since the accuracy did not statistically significantly decrease and the attentional network scores did not increase due to sleep restriction compared to normal sleep, it cannot be concluded that the experimental sleep restriction manipulation was effective. However, reaction time results show a positive non-significant trend with a medium effect size from normal to restricted sleep, indicating the experimental sleep restriction manipulation might have been effective.

Individual differences

Of the eight participants analyzed, participant 8 was both the fastest and least accurate participant. Contrastingly, participant 1 was the slowest and most accurate. Both these participants nicely illustrate a speed-accuracy tradeoff which is an often seen effect of alertness on reaction time (Fan et al., 2002). Interestingly, these specific findings were rather similar for both sleep conditions, and therefore seemed to be independent of sleep condition.

Based on visual inspection, some participants seemed to be more affected than others by the sleep restriction when it comes to reaction time. Participants affected by sleep restriction showed reaction time increases whilst the reaction times of other participants stayed the same or even decreased a bit, indicating inter-individual variability. For most participants, the accuracy did not seem to change significantly between the restricted and normal sleep condition. Interestingly, one participant showed an increase in both accuracy and reaction time from the normal sleep to the restricted sleep condition. Therefore, when comparing this participant's sleep restriction to the normal sleep data, a speed-accuracy tradeoff was visible. This indicates this tradeoff is, as opposed to previously mentioned, not for all participants independent of sleep condition.

The above mentioned findings do not only indicate people differ in their baseline ANT performance, but also in their reaction to sleep restriction. This is in line with Krause et al. (2017) who explained everyone is affected differently by sleep restriction because of genetic differences. These genetic differences could also be the explanation for the opposite direction of the speed-accuracy tradeoff seen for participants 1 and 8.

The inter-individual differences seen in the results regarding the attentional network scores could also be assigned to the genetic composition of the participants. However, since all these results are snapshots from participants' daily lives, it could also be that the participants performed differently due to other circumstances that had nothing to do with genetics but with their daily experiences. Examples could be, stress for an exam or feeling a bit ill.

Unfortunately, all but one (the multilevel analysis for the fatigue effect) statistical analyses regarding the performance data were averaged over all participants instead of including participants in the models. This means statistically nothing can be said about the inter-individual differences. Previous ANT studies also failed to include the inter-individual variability in their statistical analysis. Yet, the current study provided, despite the small sample size, clear indications for inter-individual differences. For a better understanding of those inter-individual differences and for manners to apply those differences in future products, it is crucial to perform multilevel models with participants as random effects in future research.

Physiological (EEG)

The physiological results from the current study will be compared to previous research and implications of these results will be explained below.

P1 component

In the current study, the P1 component was analyzed at frontal and parietal electrode sites. The frontal P1 component was only visible for two of the eight participants, the parietal P1 component was visible for four of the eight participants. Therefore, only a statistical analysis could be performed on the parietal P1 component. In contrast to hypothesis 1 (*partial sleep restriction will not lead to a change in P1 amplitude during the ANT compared to the normal sleep condition*) no evidence was found for statistical equivalence in the parietal P1 amplitude between the normal and restricted sleep condition. Similarly, no evidence was found to confirm hypothesis 4 (*partial sleep restriction does not lead to a change in the latency of the P1 ERP*

components obtained during the ANT compared to the normal sleep condition). Although these two results are not in line with the hypotheses, they are not completely surprising. Previous studies found no statistically significant effects of sleep restriction on the P1 amplitude and latency by means of non-significant differences between the two sleep conditions (Hoedlmoser et al., 2011; Stojanoski et al., 2019; Trujillo et al., 2009). However, this does not mean that they found the P1 amplitude or latency to be equivalent for the two sleep conditions. Therefore, the results of non-equivalence found in the current study are not completely surprising as previous studies did not find such results either. The performed equivalence tests can, however, serve as an illustration for how such hypotheses can be tested across larger sample sizes.

Since there were only a few participants for which the P1 component was clearly visible in the frontal and parietal waveforms, the choice of electrode site to analyze this component at, may be called into question. Williams et al. (2016) also applied the ANT, but did not impose a sleep restriction and found the P1 component to be more positive at occipital sites (O1, Oz, O2) than at parietal sites (P3, Pz and P4). Two studies imposing sleep restriction on their participants, but applying the PVT instead of the ANT, analyzed the P1 component only at occipital sites (Hoedlmoser et al., 2011) or at the average of all channels (Stojanoski et al., 2019). Trujillo et al. (2009) imposed sleep restriction and applied the ANT and analyzed the P1 component also at the average of all channels. For the current study, the P1 component might have been clearly visible for multiple participants at the occipital sites. Unfortunately, in the current study, the occipital ERP waveforms could not be formed due to these channels containing extreme amounts of noise.

N1 component

The N1 component was analyzed at frontal and parietal electrode sites. Unfortunately, for none of the participants these ERP waveforms contained a N1 component. As for the P1 component, it may be called into question whether the frontal and parietal electrode sites were the correct sites to analyze this component at. Previous studies researching the effect of sleep restriction on the N1 component often used the Cz and Fz electrode (Cote et al., 2003; Qi et al, 2010). In contrast to previous research on the effect of sleep restriction, previous research on the N1 component as measured during the ANT often finds the N1 component at parietal and occipital electrode sites (Kaufman et al., 2016; Neuhaus et al., 2010). From these mentioned studies, it is surprising that the N1 component was in the current study not visible in any of the frontal, parietal or central waveforms. This component could have been visible in the occipital waveforms, but unfortunately, as mentioned before, these waveforms cannot be obtained in the current study.

P2 component

Previous research shows the P2 component was present at the frontal, central and parietal regions, but most pronounced at the frontal electrodes. Moreover, the difference in P2 amplitude between the sleep restriction and normal sleep condition was the largest at frontal sites (Peng et al., 2020; Zhang et al., 2019). Note that these effects of difference are in the opposite direction. Therefore, the P2 component was expected to be visible in the frontal ERP waveforms. Qi et al. (2010), however, analyzed the P2 component at Pz. Unfortunately, in the

current study no P2 components could be detected in the frontal, central or parietal ERP waveforms.

N2 component

The N2 component was not visible in the prefrontal ERP waveforms at its expected latency (200-350 ms after stimulus onset). However, since this component precedes the P3 component in time, which was found to be delayed, possible N2 components were detected at later latencies as well. Unfortunately, as explained among the results of this present study, it could not be determined with certainty that those negative dips were indeed N2 components. This means that the current study did not find the N2 component in the frontal, parietal, prefrontal and central ERP waveforms. These results are very unexpected, because previous research on the N2 component in sleep restriction studies found this component at frontal electrode sites (Peng et al., 2020; Qi et al., 2010; Renn & Cote, 2013; Zhang et al., 2019). Previous research on the N2 amplitude during the ANT analyzed this component at the Fz, FCz, Cz and Pz electrodes (Neuhaus et al., 2007; Williams et al., 2016).

P3 component

The P3 component was not detected at the expected latency (250-450 ms after stimulus onset). However, at later latencies, this component could be found in the prefrontal, frontal, parietal and central ERP waveforms from some of the participants. Previous research on the P3 component in sleep restriction studies found this component at frontal, central and parietal sites, mostly at the midline (i.e. Fz, Cz and Pz) (Cote et al., 2003; Gosselin et al., 2019; Peng et al., 2020; Qi et al., 2010; Zhang et al., 2019). Similarly, previous research on the P3 component as measured during the ANT, found this component at the exact same sites (Kaufman et al., 2016; Neuhaus et al., 2007; Williams et al., 2016). It is therefore not surprising that the current study also found this component at those electrode sites.

The prolonged latency of the P3 component at all electrode sites is, however, unexpected. Williams et al. (2016) found the P3 latency to be largest at the fronto-central sites (Cz and FCz) and Neuhaus et al. (2007) found this latency to be largest at the Cz electrode. Williams et al. (2016) detected P3 latencies of 600 ms after stimulus onset at fronto-central electrode sites compared to 400 ms after stimulus onset at centro-parietal electrode sites (Pz and CPz). These results are in line with the prolonged latencies found in the current study at the central electrode sites. However, the prolonged latencies at the prefrontal, frontal and parietal sites are still surprising. Moreover, the latencies found in previous research regarding the P3 component in sleep restriction studies were between 250 and 450 ms after stimulus onset and, to our knowledge, almost never later than 500 ms after stimulus onset (Gosselin et al., 2019; Peng et al.,2020; Qi et al., 2010; Zhang et al., 2019; Witkowski et al., 2015).

Williams et al. (2016) and Neuhaus et al. (2007) also found similar results on the P3 amplitude: at centro-parietal sites and at the Pz electrode, the P3 amplitude was the largest. The current study did not compare the amplitude and latency of the ERP components between the electrode sites, but from the ERP waveforms, there does not seem to be a clear difference in P3 amplitude and latency between the electrode sites. In the future, the differences in

amplitude and latency of the ERP components as measured during the ANT for both sleep conditions at different electrode sites is something that needs to be studied more.

The high P3 latency is especially surprising because the reaction times found in the current study are extremely low. As explained before, the P3 latency is believed to reflect stimulus evaluation- and categorization-time, independent of the response selection and subsequent action (McCarthy & Donchin, 1981; Reuter et al., 2019). Since participants first have to evaluate and categorize the stimuli presented to them before they can react to the stimuli, the P3 component always appears before the participants' answer. Surprisingly, the mean reaction time in the current study precedes the P3 in latency which is theoretically not possible.

Interestingly, Ramchurn and colleagues (2014) performed a study using a serial choice reaction time task and compared the P3 latency for the faster (2nd quartile of distribution) and slower (4th quartile of distribution) reaction times found. They found the P3 latency to not significantly differ between the faster and slower reaction times and concluded the P3 latency to not be associated with variations in behavioral reaction times. However, even for them the fastest reaction times did not precede the P3 component.

To our knowledge, Williams et al. (2016) is the only article in the field reporting ANT reaction times preceding the P3 in time. Interestingly, they found this result for healthy young adults (aged 18 to 29 years). They conclude the young adults already determined their response by the time the P3 occurs, but did not provide any further explanation. Williams et al. (2016) agree with the current study what kind of stimuli the P3 component is elicited by, which makes the absence of further explanation especially disappointing.

The peaks that were now identified as the P3 component could, according to their latency, be the P400, P500 or P600 component. However, these components are not expected in the current study because they are normally elicited by other stimuli. The P400, for example, is elicited in infants as a reaction to faces (Leppänen et al., 2007; Puce et al., 2013). The P500 is in adults linked to the recognition of inverted faces (Marks et al., 2000). The P600 is elicited by language comprehension (Brouwer et al., 2017; Delog et al., 2021). Since faces as well as language were not part of the ANT in the current study, one can assume that the peaks around 500 ms post stimulus onset are indeed P3 components. Lastly, such late components could have been the late positive complex (LPC) which is a positive deflection 600 ms after stimulus onset. The LPC is thought to be a marker of recollection of episodic details about the prior stimulus (Yang et al., 2019). When applied to the ANT, this would mean participants remember episodic details about the previous trial (e.g. stimulus location) which they recognize in the current trial. Therefore, the LPC could become visible for similar consecutive trials. Since every ANT trial is randomly generated, every time the ANT was run, the task was different and every task might contain more or less instances of similar consecutive trials. If the peak now defined as P3 was the LPC, this could explain why the late positive peak was detectable for some but not all participants. However, when a P3 component was detected, it was detected for both sleep conditions and the chance of the ANT task having similar consecutive trials for both conditions of one participant and not for neither of the conditions of another participant is very low. Moreover, the LPC often looks more like a complex (i.e. prolonged increase in amplitude) than a peak. Therefore, the peak that was classified as a P3 component in the current study is

very unlikely to be the LPC. Unfortunately, this means it remains the question how it is possible that the reaction times are shorter than the P3 latency.

To our knowledge, two other reasons for this strange finding could be true, namely the effect of an artifact that is still unknown to us, or people do not first have to evaluate and categorize the stimulus before they can react accurately.

Frontal P3 amplitude and latency.

Visual inspection of the four frontal ERP waveforms for which the P3 component was detected, suggest the P3 component to be larger in both amplitude and latency in the restricted sleep condition compared to the normal sleep condition. Although both main and interaction effects of the sleep condition and time in session on the frontal P3 amplitude were non-significant, a trend was found towards higher P3 amplitudes after 3 consecutive days of partial sleep restriction compared to normal sleep. Interestingly, this trend was close to reaching significance and with a medium effect size ($\eta^2_G = 0.183$). Research with larger sample sizes is needed to validate hypothesis 2 (*partial sleep restriction will lead to a decreased P3 amplitude during the ANT compared to the normal sleep condition*).

Similarly, the main effect of sleep condition on frontal P3 latency was not close to reaching statistical significance, but did render a large effect size ($\eta^2_G = 0.263$). This suggests that the sleep restriction could have a rather large effect on P3 latency in the frontal region. Since the P3 latency has been implied to reflect stimulus classification speed, this suggests people who are sleep restricted need more time to classify the stimulus presented to them.

Lastly, since no fatigue effect was found in the reaction time data of the current study, it was no surprise that the time factor in the repeated measures ANOVA for the P3 amplitude and latency was non-significant. This suggests, based on a very low sample size, that participants' attention and cortical brain activity was the same during the whole ANT.

Individual differences

Not surprisingly, inter-individual variability was also clearly visible in the physiological data. The ERP waveforms of every participant looked completely different and participants reacted differently to the sleep restriction imposed on them. For example, one participant had a frontal P3 component that shifted very clearly due to sleep restriction whilst this shift was only mildly visible for the other participants. It could be that a certain amount of sleep loss (i.e. threshold) is needed for the ERP components to change in amplitude and latency. This threshold could be different for different people. The participant for which the frontal P3 component shifted very clearly due to sleep restriction might have a lower threshold than a participant for which this ERP component did not make a clear shift. Further research on such thresholds should be done to validate the above speculation. Even more clear were the inter-individual differences in the peak detection. ERP waveforms of some participants contained two clear ERP components whilst other ERP waveforms contained none.

The most surprising result regarding the physiological data was the difficulty detecting the ERP components. The P1 and P3 components were found for some participants but not for others whilst other components could not be found at all. Compared to previous studies, approximately the same amount of trials and thus epochs were used for the ERP formation.

Although previous research visualized the grand-average ERP waveforms, they also performed their statistical analyses on the individual ERP waveforms. When averaging the EEG data of about 15 to 20 participants, a smoother ERP waveform will be obtained compared to averaging the EEG data of a single participant. This is because any noise left in the signal of a single participant will be averaged out when combining the signals of multiple participants. Unfortunately, when averaging the current EEG data over all participants, only for a few grand-average ERP waveforms some ERP components are visible. As an example, Appendix I, Figure I1 shows the grand-average frontal ERP waveform for both sleep conditions. Since the determination of the ERP components in the grand-average ERP waveforms did not improve compared to the participant specific ERP waveforms, this would not have been a solution for easier ERP component detection in the current study. Moreover, to our knowledge, similar pre-processing steps were performed as in previous studies. Therefore, the reason for the difficulty in detecting the ERP components remains a question.

As explained above, multiple reasons could have been the cause of not obtaining the expected results to the statistical tests on both the performance and physiological data. In future studies a sufficient number of participants, both typical and non-typical and from all generations, is important to obtain more conclusive results that are generalizable to the whole population. Further research on the effect of partial sleep restriction on the ANT accuracy, reaction time and attentional network scores as well as on the amplitude and latency of ERP components is needed in order to draw conclusions. Moreover, this further research is needed to conclude whether partial sleep restriction has indeed a different effect on ANT performance and ERP components than TSD has.

Exploratory investigation

Unfortunately, not enough useful data was present in the current study to further investigate the differences in the amplitude and latency of the five ERP components between the two experimental sleep conditions across the three different networks of the ANT. The Discussion section contains more insights on how to investigate this question in the future.

To explore whether the behavioral markers obtained with the ANT or the EEG-derived metrics are more sensitive to partial sleep restriction, the effect sizes obtained from both performance and physiological data were compared. Firstly, ANT accuracy might not be the most sensitive marker for sleep restriction as effect size was very small (d = 0.018). Secondly, ANT reaction time as well as the alerting effect might be more sensitive markers. The effect of sleep restriction on both reaction time and the alerting effects rendered medium effect sizes (d = 0.467 and d = 0.432, respectively), which suggest that the overall reaction time and the alerting component might be more sensitive markers for partial sleep restriction than the accuracy. The orienting and executive control effects are probably also less sensitive markers for partial sleep restriction as differences between the two sleep conditions showed only small to very small effect sizes (d = 0.164 and 0.036, respectively).

The most sensitive physiological metric might be the frontal P3 latency. Although this metric resulted in a statistically non-significant effect between the restricted and normal sleep condition, the effect size for sleep condition was large (η^2_G = 0.263). Similarly but less sensitive,

the frontal P3 amplitude might also be a sensitive physiological metric for sleep restriction, because this effect was close to reaching significance and had a medium effect size (η^2_G = 0.183). Regarding the parietal P1 amplitude and latency, no evidence was found for equivalence between the two sleep conditions. However, the parietal P1 amplitude or latency might not be the most sensitive markers for the effect of sleep partial restriction, because those markers have been implied to not differ as a function of sleep restriction or TSD in previous studies (Hoedlmoser et al., 2011; Stojanoski et al., 2019; Trujillo et al., 2009).

Since different statistical tests were used and thus different effect sizes were obtained for the performance and physiology data, a statistical comparison between the two sets of markers was not possible.

Limitations

Multiple limitations of the current study were identified and described below that could have impacted the results and the generalizability of those results.

Small sample size

The sample size is the largest limitation of this study. With only eight participants providing useful data to analyze the ANT and obtain ERP waveforms, both the statistical power as well as the generalizability of the current study to the whole population was expected to be low. Surprisingly, the effect sizes obtained are often medium, so trends found in the current study are expected to have societal relevance but need to be tested with a larger sample.

Insuperable data exclusion played a large role in this small sample size. Two participants had to be excluded because either their EEG or ANT data could not be used for analysis. Moreover, the initial sample size was already small (N=10). The exclusion of the data from the participant who was believed not to understand the incongruent ANT trials was especially unfortunate. Questions remain whether the misunderstanding of the task's instructions stemmed from the effect of sleep restriction or from the novelty of the task. For both sleep conditions, this participant had not only extremely low accuracies for the incongruent trials but also for the congruent and neutral trials. Moreover, reaction times for this participant were extremely high for both sleep conditions. Therefore, they could have been a very good example of the extreme inter-individual differences that are possible.

Participants

Besides the extremely small sample size used in the current study, there are more aspects of the participants that brought generalization limitations into the study. The participants included in the current study were mostly students enrolled in the Eindhoven University of Technology, as a result, the mean age was 22.6 years (SD = 4.8; range 19-32 years). A study by Williams and colleagues (2016) found younger adults (M = 21.6, SD = 3.0, range = 18-29) to respond significantly faster to ANT trials than older adults (M = 65.1, SD = 5.1, range = 60-76). Furthermore, they found the P3 at centro-parietal and fronto-central sites to occur later for the older adults compared to the younger adults. This prolonged latency shows the overall slowing of older adults. Regarding the P3 amplitude, Williams et al. (2016) found a decrease for older

adults for incongruent trials compared to congruent trials, whilst this effect was not found for younger adults. P3 amplitude reduction is expected to be caused by greater response inhibition (Groom & Cragg, 2015). Younger adults already determine their response before the P3 onset which is why their P3 amplitude was not modulated by the congruency of the stimulus. Older adults experience more difficulty inhibiting the information of the flanker arrows and selecting the correct response. Therefore, the P3 amplitude of older adults is modulated by the congruency of the stimulus (Williams et al., 2016).

Moreover, Reuter and colleagues (2019) studied six age groups (children: mean age of 9.32 year olds (SD = 0.65), young adults: mean age of 22.85 (SD = 2.50), early middle-aged adults: mean age of 42.62 (SD = 3.61), late middle-aged adults: mean age of 59.04 (SD = 2.39), older adults < 75: mean age of 71.93 (SD = 3.04) and older adults > 75: mean age of 78.16 (SD = 1.98)). All age groups performed a color flanker task while EEG was recorded. They found the young adults to have the quickest response speed and highest accuracy whilst the children and older adults performed worse on those areas. A similar u-shaped pattern was found for the P1, N1, N2 and P3 latencies with the shortest latencies occurring in the middle-aged groups. The P1 amplitude followed a u-shaped pattern over age whilst the N1 amplitude followed an inverted u-shaped pattern, both with the peak at the middle-aged group. Furthermore, they found age to shift the P3 amplitude from parietal to frontal areas. Lastly, they suggested the performance of the children to be regulated by cognitive processing speed whilst the performance of older adults was dependent on cognitive resources. These effects of age on both ANT performance and ERP component, lead to the question whether the results from the current study are generalizable individuals from different age categories, especially people older than 25 years old.

Furthermore, the current study excluded neuroatypical people from participation which prevents generalization to the neuroatypical population. Extensive research was done on the differences in ERP waveforms between neurotypical and neuroatypical people. People with attention-deficit hyperactivity disorder (ADHD), for example, are known to have differences in their ERP components. Downes and colleagues (2017) provided an overview of the effects of ADHD on the P2, N2 and P3 components and found effects on both latency and amplitude of those components. Moreover, Riggins and Scott (2020) reported multiple neurodevelopmental disorders (e.g. autism spectrum disorder, ADHD and language disorders) to have an effect on both the amplitude and latency of the P3. Unfortunately, these studies applied other cognitive tasks than the ANT and did not apply sleep restriction. In order for the results of the current study to be generalizable to the whole population, further research is needed on the effects of sleep restriction on ERP components as measured during the ANT on both neurotypical and non-neurotypical people.

Lastly, the possibility of a selection bias should be kept in mind upon interpretation of the results. Participants knew, before signing up for the study, that sleep restriction would be part of the current study. It could very well be that people who consider themselves to cope well with sleep loss signed up for the study whilst people who do not consider themselves to cope well with sleep loss refrained from participation.

Trigger alignment

When visually inspecting the ERP waveforms obtained in the current study, many signals showed a lot of very small and quickly fluctuating peaks instead of the desired smooth signal. Further research into the ANT, specifically the programming of the trigger onset time that was sent to the different devices used to monitor EEG responses showed a misalignment. This means that it is very probable that trigger onset times were delayed by 0 to 20 ms (depending on the trial). This lack of millisecond accuracy can unfortunately hinder the processing and detection of EEG activity. As explained in Appendix C, averaging EEG signals that are time-locked to the trigger results in the averaging out of noise and the peaks that are always at the same time after the trigger become more pronounced. However, when delays are incorporated in this process, the peaks might not align and instead form the small, quickly fluctuating peaks as seen in some of the ERP waveforms. This could be an explanation for not finding some ERP components in the ERP waveforms.

Possible application of current study

As explained before, quantification of the effects that sleep loss can have on attention in daily life is crucial for the development of interventions to support people suffering from sleep loss in their daily lives. Monitoring tools are needed to enable such quantification. Ideally people suffering from sleep loss would be able to have such a monitoring tool at home or, even better, can take it with them. Therefore, this monitoring tool would have to be small, light and easy to use. Furthermore, it would have to be able to measure some variable related to attention and use those measurement data to quantify on a scale how much attention someone has. The tool would then have to convert this attention scale to an accurate, reliable and understandable measure to the user. This last step is important for users to apply the information the monitoring tool collected to their daily lives. The tool could, for example, advise the user on whether or not they should drive a car or use heavy machinery.

Since the current study showed the frontal P3 ERP component to be the most sensitive marker of sleep restriction, this could be a great measure of attention that can be used for the monitoring tool. In order for ERP components to become apparent, a stimulus is needed to time-lock the ERP component to. Unfortunately, the ANT takes 20 minutes which might be too burdensome for people in their daily life. A shorter 10 minute version of the ANT exists (Weaver et al., 2013), but repetition of such tasks would still be burdensome. Furthermore, a practice effect might not have been found in the current study, but could become more apparent when the task is done repetitively. To our knowledge, a very short (e.g., 3 minutes) version of the ANT has not been developed yet and could be something for the future to develop. Once developed, this version of the ANT should be tested extensively to verify it measures the same elements as the original ANT does. A shorter ANT means less trials and thus less epochs for the formation of ERP waveforms. It is, therefore, important to verify whether ERP formation is possible and valid with the little amount of data obtained during the very short version of the ANT.

If a very short version of the ANT would be developed and ERP formation is possible, a wearable EEG device could be a good monitoring tool for attention in peoples' daily lives. A few wearable EEG devices are already on the market for health, education and entertainment purposes. NeuroSky is a company that developed such a device which could be further

developed to incorporate the desired functions needed to inform people about the status of their attentional capacities (NeuroSky, 2022). The currently developed EEG devices are only able to evaluate EEG frequency bands. Alterations to these currently developed devices could enable ERP analysis. A smartphone app could be used to administer the ANT and immediately report the status of the user's attentional capacities (e.g., advice on driving a car). Moreover, an algorithm could be used to obtain tailor made feedback for the user including initial variables like age (as this might affect the detection's location as well as the amplitude and latency of the ERP components measured).

Lastly and most importantly, a better understanding of the P3 component in relation to attention is needed in order for the translational step from the measured data to the attentional scale to be possible. Besides, an attentional scale should be developed with thresholds indicating how much attention is needed to still perform certain tasks safely (e.g. driving a car). This scale should be tailor made to match the user because inter-individual variability in attentional capacity as well as coping with sleep restriction are common.

Recommendations for future research

As explained before, the latency and amplitude of the ERP components might be different at different electrode sites. Since sleep restriction is expected to have an effect on both the amplitude and latency of ERP components, it is important to understand how these amplitudes and latencies behave at the different electrode sites. Unfortunately, the current study did not statistically compare the amplitudes and latencies between the electrode sites. For future research with a substantially larger sample size, it is recommended to add electrode site as a factor to the multi-level analyses on the ERP amplitude and latency. Firstly, for every ERP component, it should be known at which electrode sites those components can be obtained. Secondly, it needs to be investigated at which of those electrode sites the difference between the ERP components measured during the ANT under the two sleep conditions is the largest (e.g., at which electrode site differs the P3 latency the most between the sleep restriction and normal sleep condition?). Knowledge about how ERP components behave at specific electrode sites might also enable electrode selection for wearable devices in the future.

Similarly, research is needed to explore to what extent age as well as neurotypicality has an effect on ERP components during sleep restriction. An important question to be answered is whether ERP components from people with different ages react differently to sleep restriction. Therefore, in the case of a replication of the current study, people from different age categories and neuro-typicalities need to be included, and age as well as neurotypicality should be added as a factor to the statistical analysis to improve the generalizability of the results to the entire population and investigate moderations in sensitivity to sleep restriction as a function of age and neuro-typicality.

As explained in the introduction, the P3 amplitude and latency is dependent on task difficulty and thus differs for incongruent and congruent trials (Neuhaus et al., 2007). Similarly, the P2 amplitude increases with the complexity of visual stimuli (Pernet et al., 2003). Therefore, the effects of cue and stimulus type should in the future be added to the multilevel analysis on the amplitude and latency of the ERP components. This will enable exploration of the effects of cue and stimulus type and their interaction with sleep restriction.

As said before, ERP component detection was difficult. Although previous research showed ERP components were detected in EEG signals obtained during the ANT, research on ERP components in sleep restriction mostly used cognitive tasks like the PVT or Go/NoGo-task. The current study is, as explained before, part of a larger study which also included the PVT. To check whether the ERP component detection was difficult due to the ANT, an ERP analysis should be done on the EEG data obtained during this PVT. The ERP waveforms from the current study could then be compared to the ERP waveforms obtained from the PVT data. From this comparison it can be concluded whether ERP detection is easier on data obtained during the ANT or PVT. If, for example, the PVT results in clearer ERP component detection, the question remains why the detection of these components in the EEG data obtained during the ANT was difficult. These possible results could also mean that the ERP components obtained from the PVT are a better measure to quantify the effects of sleep restriction than the ERP components obtained from the ANT.

Krause and colleagues (2017) describe a dose-dependent effect of sleep restriction on attentional task performance. For future research, it would be interesting to know whether the dose-dependent effect of sleep restriction also applies to the ERP components. The current study only invited the participants to the lab after three consecutive nights of partial sleep restriction. Future studies should consider inviting the participants into the lab every day during both sleep conditions. Moreover, in those studies, the sleep restriction should be extended to more than three consecutive days to explore whether a possible threshold of sleep loss is present after which sleep restriction effects on the ERP components become apparent. Previous studies applying 40 hours of TSD showed, for example, a reduced N1 amplitude whilst less severe sleep restriction of 24 hours of TSD did not. Krause et al. (2017) also proposed the intra-individual differences to increase due to sleep restriction. The current study was not able to measure intra-individual differences in response to sleep restriction because the participants were never in the same condition twice. Therefore, a study in which the participants go through both sleep conditions twice or more times would enable intra-individual variability detection. In addition, this future study should investigate both the intra- and inter-individual variability in compensatory mechanisms. It is currently unknown whether the same participant is always dependent on the same compensatory mechanism or maybe these mechanisms show, due to sleep loss, variability as well.

Lastly, since most people with sleep problems are able to sleep a few hours per night instead of not sleeping at all for multiple consecutive nights, the results found in partial sleep restriction research have a higher ecological validity than the results from studies applying TSD. Therefore, more research is needed to discover whether the same effects as found by previous studies, using 24 hours of TSD, can also be found for partial sleep restriction. More importantly, for applications such as the monitoring tool previously described, further research is needed on people who actually struggle with sleep loss on a regular basis in their daily lives. People with sleep problems, instead of healthy participants, might get used to the consequences of sleep loss or learn to cope with those consequences. It could be that people with sleep problems, compared to healthy participants, have different compensatory mechanisms in place. Sleep loss

might thus have a different effect on their, compared to healthy participants, performance on cognitive tasks and on their ERP components.

Conclusion

The goal of the current study was to investigate the effects of sleep loss on brain activity as well as attention. The main research question was: "*What are the effects of 3 consecutive days of partial sleep restriction on the P1, N1, P2, N2 and P3 ERP components (measured during an Attention Network Test) compared to a normal sleep condition?*". Although no statistical significant differences between the two sleep conditions were found for the reaction times, accuracies and network scores, the overall reaction time and alerting effect provided indication for a medium effect of sleep restriction in the expected direction. Moreover, the ERP component detection was difficult and only a few P1 and P3 components were detected. The parietal P1 were not found to be equivalent in amplitude and latency between the two sleep conditions. Although no statistical significant difference between the two sleep conditions was found for the frontal P3 amplitude and latency, for both the amplitude and latency a trend in the expected direction was found based on, respectively, a medium and large effect size. Additionally, the comparison between the performance and physiological measures was difficult as the effect sizes could not be compared statistically. However, the current study provides a great framework in terms of method and statistical tests for future research, with more participants, to work with.

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Appendix

Appendix A - What is sleep and why do we need it?

Sleep is defined as a state of reduced reactivity during which one is inactive. Practically speaking, this means that, compared to wakefulness, one responds to a lesser extent to environmental stimuli, and motor activity as well as metabolism are reduced. Besides, sleep is always reversible which makes it distinct from coma or death (Abrams, 2015; Siegel, 2009). Since people need sleep for survival and health, something must happen in the human body during sleep which fulfills a vital function. What this exact function is, is yet unknown, but different theories exist as to why people need sleep.

The first of such theories is the energy conservation theory which states that people sleep to conserve energy (Webb, 1974). Abrams (2015) argues that this theory is in line with the evolutionary perspective of going into a state of lowered caloric needs when food was scarce to promote survival. Another theory is the restorative theory which argues that the body repairs itself during sleep from the damage sustained during wakefulness (such as death of neurons in the hippocampus, depletion of energy stores, oxidative stress and downregulation of receptors). This theory is partly based on the fact that the body produces hormones during wakefulness that release energy whilst the hormones produced during sleep are anabolic and stimulate the building-up aspect of metabolism (Weitzman et al., 1974). Lastly, the information processing theory states that learning and memory are promoted by sleep. Sleep would enable people to remember more and forget slower (Jenkins & Dallenbach, 1924). This theory argues that during wakefulness learning circuits become saturated and need to be restored to baseline levels (Abrams, 2015).

Although the exact reason for sleep is still unknown, it is known that during sleep multiple important processes happen inside the human body. Examples are the regulation of heart rate and blood pressure, the restoration of cognition and memory capacity as well as cellular repair and the regulation of immune defense (Liew & Aung, 2021). Furthermore, all three mentioned theories are in line with the synaptic homeostasis hypothesis from Tononi and Cirelli (2003). This hypothesis states that sleep is crucial to the restoration of the synaptic homeostasis which is brought out of balance due to synaptogenesis (synaptic development) and synaptic strengthening during wakefulness (Tononi & Cirelli, 2014).

Appendix B - Consequences of sleep deprivation

Physical & mental health

Besides sleep deprivation being a consequence of multiple diseases, it can also be the cause of many major health risks (Liew & Aung, 2021). Sleep apnea, for example, can cause sleep deprivation and can also immediately lead to resistant hypertension (Cowie, 2017). Chronic sleep loss could have serious health consequences like hypertension, diabetes, heart attack, and stroke (Institute of Medicine, 2006). Besides, it can lead to mental distress as well as depressive symptoms and even depression (CDC, 2005-2008; Institute of Medicine, 2006).

Another major consequence of sleep deprivation is obesity, for the very simple reason that when one is awake they can think about food more and eat more compared to being asleep. Furthermore, previous studies have shown that 4 hours of restricted sleep for two consecutive days already leads to decreased leptin levels (hormone that suppresses appetite) and increased ghrelin levels (hormone that promotes appetite) as well as increased hunger and appetite for calorie-dense food (Spiegel et al., 2004; Schmid et al., 2008). With obesity being both a consequence and a cause of sleep loss, a vicious circle is formed which makes it difficult to treat both sleep loss and obesity. The same effect can be seen for multiple other health conditions related to sleep deprivation.

Mood & emotions

Besides the health consequences described above, sleep deprivation could also lead to a decreased positive affect as well as higher anxiety levels. Furthermore, sleep deprived people reported to be more confused and irritable as well as having a worsened emotional regulation compared to normal sleep conditions (Baum et al., 2014; Lo et al., 2016; Talbot et al., 2010). Furthermore, Lee and her colleagues (2022) reported that the recognition of emotional expression in faces is impaired in people after a single night of TSD.
Previous studies have shown that sleep deprivation increases risk taking, impulsivity and reward sensitivity. An increase in reward sensitivity leads to an impaired reward discrimination accuracy, meaning people become less accurate in evaluating differences in reward values. These increases in consummatory and approach behavior are thought to be the result of altered dopamine signaling which is also associated with sleep deprivation (Krause et al., 2017).

Not surprisingly, research shows people who are restricted to sleep only a few hours per night report a higher subjective sleepiness and more fatigue compared to people who were allowed to sleep 8 or 9 hours per night (Cunningham et al., 2018; Lo et al., 2016). Likewise, Van Dongen and colleagues (2003) performed a study in which healthy participants were restricted to 8, 6 or 4 hours of sleep per night for 14 consecutive days, or to TSD for 3 consecutive days. They found subjective sleepiness as reported by the participants in the 4 or 6 hours of sleep conditions to increase over time, compared to the 8 hour sleep condition. Furthermore, subjective sleepiness increased acutely in the first few days of 4 or 6 hours of sleep and increased to a lesser extent in subsequent days. Moreover, participants in the TSD condition reported higher subjective sleepiness after 3 days than the participants in the 4 or 6 hour sleep condition reported after 14 days.

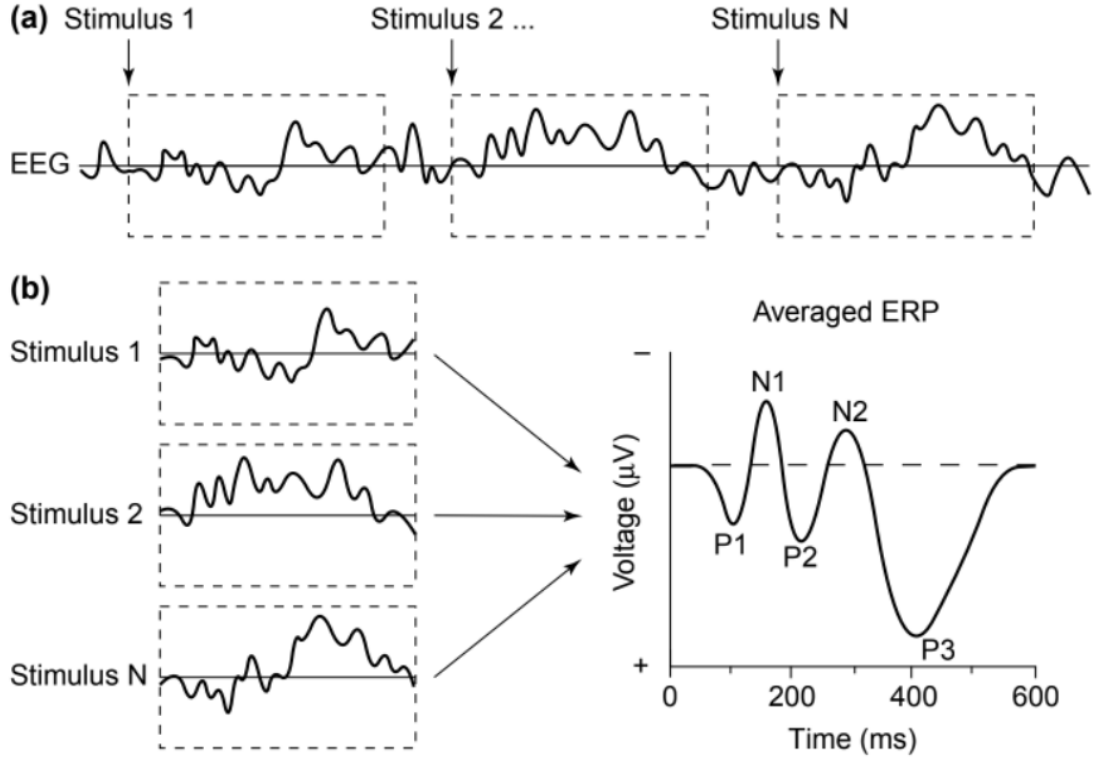
Appendix C - What are ERPs?

Due to pyramidal neurons in the brain firing simultaneously and thereby activating the next neurons, postsynaptic potentials are generated. These electrical potentials travel through the brain and skull to the scalp where they can be measured by electrodes of the EEG apparatus. Figure C1a shows how such measurement of a single electrode looks like. From this waveform, it is difficult to extract information that is time-locked to the stimuli. Therefore, the EEG waveform

is cut up in smaller pieces that are all time-locked to the stimuli (see Figure C1b). Subsequently, these segments are lined up in time and averaged, resulting in an event-related potential (ERP). By averaging over multiple time-locked waveforms, the brain activity unrelated to the stimulus will be averaged out and the remaining ERP waveform will contain only information that is consistently time-locked to the stimuli. The ERP waveform as seen in Figure C1b contains several positive and negative peaks, sometimes called components, indicated by a P and N, respectively. Furthermore a number is assigned to the peaks indicating the latency of the peak relative to the stimulus onset, e.g. P2 occurs 200ms after the stimulus onset which is set to 0ms. Furthermore, notice the reversed y-axis indicating the Voltage in μV running from positive to negative values.

Figure C1

"Extraction of the ERP waveform from the ongoing EEG. (a) Stimuli (1... N) are presented while the EEG is being recorded, but the specific response to each stimulus is too small to be seen in the much larger EEG. (b) To isolate the ERP from the ongoing EEG, the EEG segments following each stimulus are extracted and averaged together to create the averaged ERP waveform." Adapted from Luck et al., 2000.



Appendix D

The P1 component

The P1 component is a positive deflection occurring approximately 100 ms after visual stimulus onset or 50ms after auditory stimulus onset. This component is believed to reflect arousal levels and is associated with the suppression of unattended information (Key et al., 2005). Furthermore, the posterior P1 has been associated with alerting and orienting processes and is the earliest marker of visual attention (Williams et al., 2016). Most importantly, P1 amplitude is a marker of visual processing and attention whilst the P1 latency is a marker of encoding and processing speed (Galvao-Carmona et al., 2014; Reuter et al., 2019). When a visual stimulus is presented at an attended location, the P1 amplitude is increased compared to visual stimulus presentation at an unattended location. This indicates that heightened attention leads to enhanced visual processing (Talsma et al., 2005; Williams et al., 2016). This principle has been confirmed in the ANT; the P1 amplitude for spatial cue trials was increased compared to the no cue and center cue trials. It is thought that the increased P1 amplitude might be the reason for the improved performance on the spatial cue trial compared to the no and center cue trials (Galvao-Carmona et al., 2014). There are, however, studies finding the opposite effects in the P1 amplitude for attended versus unattended cue locations. Doallo et al. (2004), for example, found increased P1 amplitudes for invalid cues, meaning the stimulus was presented at an unattended location, compared to valid cues after which the stimulus was presented at the attended location.

The N1 component

The N1 component is a negative deflection about 100 ms after stimulus onset with a small amplitude relative to the background noise. This component is associated with sensory processing in primary sensory areas of visual, auditory and tactile stimuli (Boonstra et al., 2007; Gosselin et al., 2005). Some argue the N1 to be independent of attention or other cognitive processing (Näätänen, 1992), whilst others argue attention can in fact affect sensory processing and thus affect the N1 component (Campbell, & Colrain, 2002). It was, for example, found that asking people to attend to one ear resulted in an increased amplitude of their auditory N1 (Näätänen & Winkler, 1999; Woldorff & Hillyard, 1991). Similarly, the same effect on the N1 component was found upon administering a small dose of caffeine (Lorist et al., 1994a; Lorist et al., 1994b). These studies show attention either directly or through the mediation of caffeine has an effect on the N1 component. Furthermore, the N1 component is associated with selective attention, intentional discrimination processing and initial stimulus selection for later pattern recognition (Vogel & Luck, 2000). Lastly, visual stimuli elicit a smaller N1 amplitude and larger latency compared to auditory stimuli (Key et al., 2005).

The P2 component

The P2 component is a positive deflection around 130-250 ms after stimulus onset. This component reflects the perception of an object with respect to its shape and additional object information (Zhang et al., 2019). Besides, it is sometimes associated with sensation-seeking

behavior (Sur & Sinha, 2009). Therefore, stimulus type (more or less sensational) can have an effect on the P2 latency: an increase in the complexity of the stimulus (e.g. Asiatic characters vs. letters, for French participants) leads to an increased P2 latency (Pernet et al., 2003). This component is also associated with processes following the initial perception processing such as short-term memory and selective attention (Key et al., 2005; Tanovic et al., 2018). Lastly, the P2 component is sensitive to the working memory requirements of a task as well as (changes in) attention (Smith et al., 2002).

The N2 component

The N2 component is a negative deflection around 200-350 ms after stimulus onset (Zhang et al., 2019). This component has been associated with target selection and stimulus discrimination. Task type (e.g. semantic or physical tasks) and stimulus type (e.g. written words, human faces or objects) have been shown to cause differences in the N2 component. Furthermore, it is related to the detection of discrepancy between a certain stimulus and the expectation of the participant, but only whilst the participant pays attention to the stimulus (Key et al., 2005). Moreover, this component is associated with cognitive control, specifically in relation to successful inhibitory control (Downes et al., 2017). Similarly to other components, the N2 latency is a marker for encoding and processing speed (Reuter et al., 2019).
As opposed to the P2 component, an increasingly complex stimulus leads to a reduced N2 amplitude and has no effect on the N2 latency (Pernet et al., 2003). Lastly, an ERP component close to the N2 is the N170 which is a negative deflection around 156-189 ms after stimulus onset. Since the N170 reflects the visual processing of human faces, it will likely not interfere with the N2 as measured during the ANT (Key et al., 2005).

The P3 component

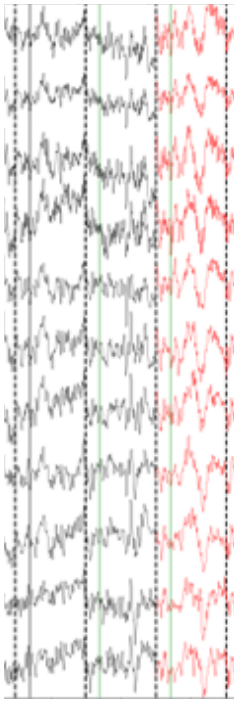
The P3 component (often called P300) is a positive deflection about 300 ms after stimulus onset. This is the most researched component to date and is mostly used to study the responses to unexpected auditory or visual stimuli (Key et al., 2005). Either surprising attended events or unattended events producing orienting are causing the P3 component (Pritchard, 1981). Typically, the oddball task is used to elicit this component. During this task, a target stimulus is presented infrequently, and therefore unexpectedly, in between distractor stimuli. The participant is instructed to only respond to the target stimulus (Boonstra et al., 2007; Sur & Sinha, 2009).
The P3 component is sometimes divided into subcomponents P3a and P3b, with P3a (sometimes called novel P3) preceding P3b (sometimes called target P3) in latency. Unexpected and infrequent novel stimuli elicit the P3a component which is associated with an attentional shift from one aspect of the stimulus environment to another. The P3a amplitude is positively correlated with attentional focus. Anticipated and infrequent target stimuli elicit the P3b component which is associated with the allocation of attentional resources during cognitive operations involved in updating working memory. Its amplitude is a measure for the amount of attentional resources allocated toward a stimulus. The P3b latency is believed to reflect stimulus detection- and evaluation-time, independent of the response selection and subsequent action (Boonstra et al., 2007; Broglio et al., 2009; Sur & Sinha, 2009; Thompson et al., 2020). It can

thus be concluded that the P3 component is affected by attention. Specifically, the P3 amplitude increases due to greater attention (Sur & Sinha, 2009). Furthermore, the P3 latency has been shown to relate to cognitive processing speed, more specifically the stimulus classification speed as a result of discriminating one event from another (Reuter et al., 2019; Sur & Sinha, 2009). Shorter P3 latencies are related to better performances, indicating it to be related to cognitive abilities (Key et al., 2005; Sur & Sinha, 2009).

Appendix E

Figure E1

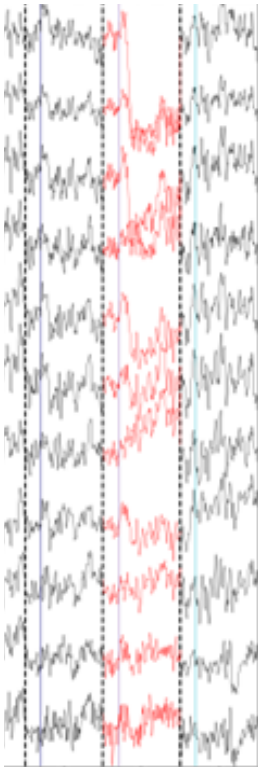
Example of Epoch for which all Electrodes Contain a Sudden Change in Signal Amplitude.



Note. The red epoch contains the sudden change in signal amplitude. Dashed vertical lines indicate epoch boundaries. Solid vertical lines indicate trigger onset times (colors indicate different trial types).

Figure E2

Example of Epoch Containing Signal Drift.



Note. The red epoch contains the signal drift. Dashed vertical lines indicate epoch boundaries. Solid vertical lines indicate trigger onset times (colors indicate different trial types).

Appendix F

Table F1

Mean Accuracy and Reaction Times for both Sleep Conditions for the Different Stimulus and Cue Types.

	Mean accuracy		Mean reaction time (ms)	
	NS	RS	NS	RS
Congruency				
Congruent	0.99 (0.11)	0.98 (0.15)	395 (88)	415 (99)
Incongruent	0.89 (0.31)	0.91 (0.29)	462 (89)	484 (108)
Neutral	0.98 (0.14)	0.98 (0.15)	379 (75)	397 (94)
Cue type				
No cue	0.97 (0.18)	0.96 (0.19)	442 (84)	464 (104)
Center cue	0.95 (0.22)	0.94 (0.23)	416 (100)	440 (112)
Double cue	0.94 (0.25)	0.96 (0.19)	401 (84)	417 (104)
Spatial cue	0.96 (0.19)	0.95 (0.23)	380 (83)	401 (96)

Note. Values indicated are means with standard deviations in parenthesis.

Figure F1

Mean Participant Accuracy to the Four Cue Types for the Three Stimulus Types

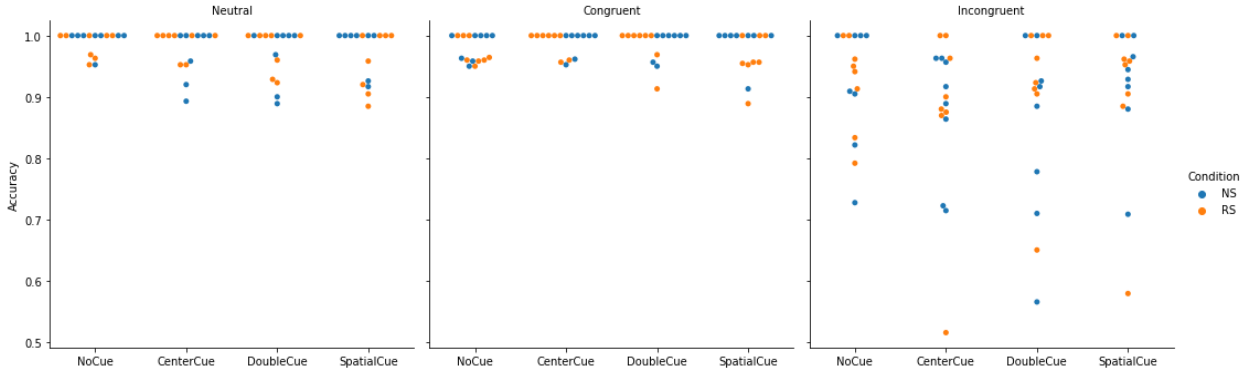


Table F2

Frontal P1 Amplitude and Latency for Participants 2 and 5.

	Amplitude (μV)				Latency (ms)			
	Participant 2		Participant 5		Participant 2		Participant 5	
Time	RS	NS	RS	NS	RS	NS	RS	NS
T1	3.393	2.521	3.138	3.594	99.00	92.50	104.00	96.75
T2	1.681	1.729	3.587	3.139	83.00	98.25	65.25	89.25

Note. T1 and T2 are respectively the ERP waveforms based on the first and second 144 epochs.

Table F3

Parietal P1 Amplitude and Latency for Participants 2, 3, 5 and 7.

Amplitude (μV)

	Normal sleep				Restricted sleep			
Time	Part 2	Part 3	Part 5	Part 7	Part 2	Part 3	Part 5	Part 7
T1	2.596	1.677	2.273	1.717	2.743	2.534	1.313	1.003
T2	3.429	2.642	2.764	1.738	1.895	3.495	1.983	0.994

Latency (ms)

	Normal sleep				Restricted sleep			
Time	Part 2	Part 3	Part 5	Part 7	Part 2	Part 3	Part 5	Part 7
T1	93.00	122.75	95.00	111.00	100.25	106.75	102.00	50.00
T2	97.00	106.75	105.25	78.50	94.75	90.75	86.75	50.00

Note. T1 and T2 are respectively the ERP waveforms based on the first and second 144 epochs.

Appendix G

A 2 (sleep condition: normal sleep, restricted sleep) × 2 (time: first 144 trials, second 144 trials) repeated measures ANOVA showed no significant main or interaction effects were found (see Table G1), also note the small effect sizes.

Table G1

Results Repeated Measures ANOVA on Parietal P1 amplitude.

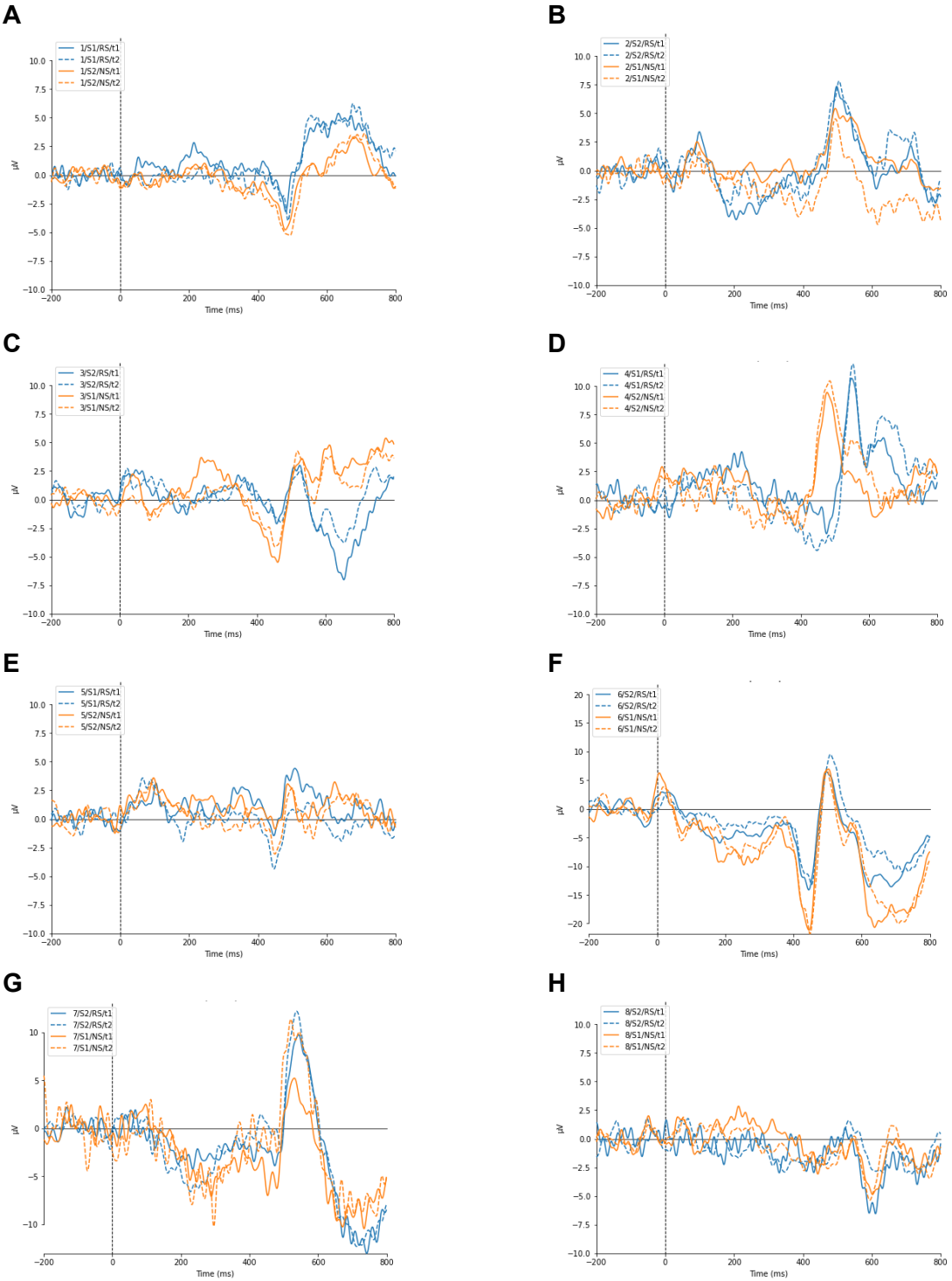
Factor	F-statistic	Numerator df	Denominator df	p-value	η^2_G
Sleep condition	0.782	1	3	0.442	0.064
Time	2.661	1	3	0.201	0.073
Sleep condition × Time	0.781	1	3	0.442	0.019

Note. df = degrees of freedom. η^2_G = generalized eta-squared.

Appendix H

Figure H1

Frontal ERP Waveforms for Participants 1-8 per Sleep Condition and per Time Group.

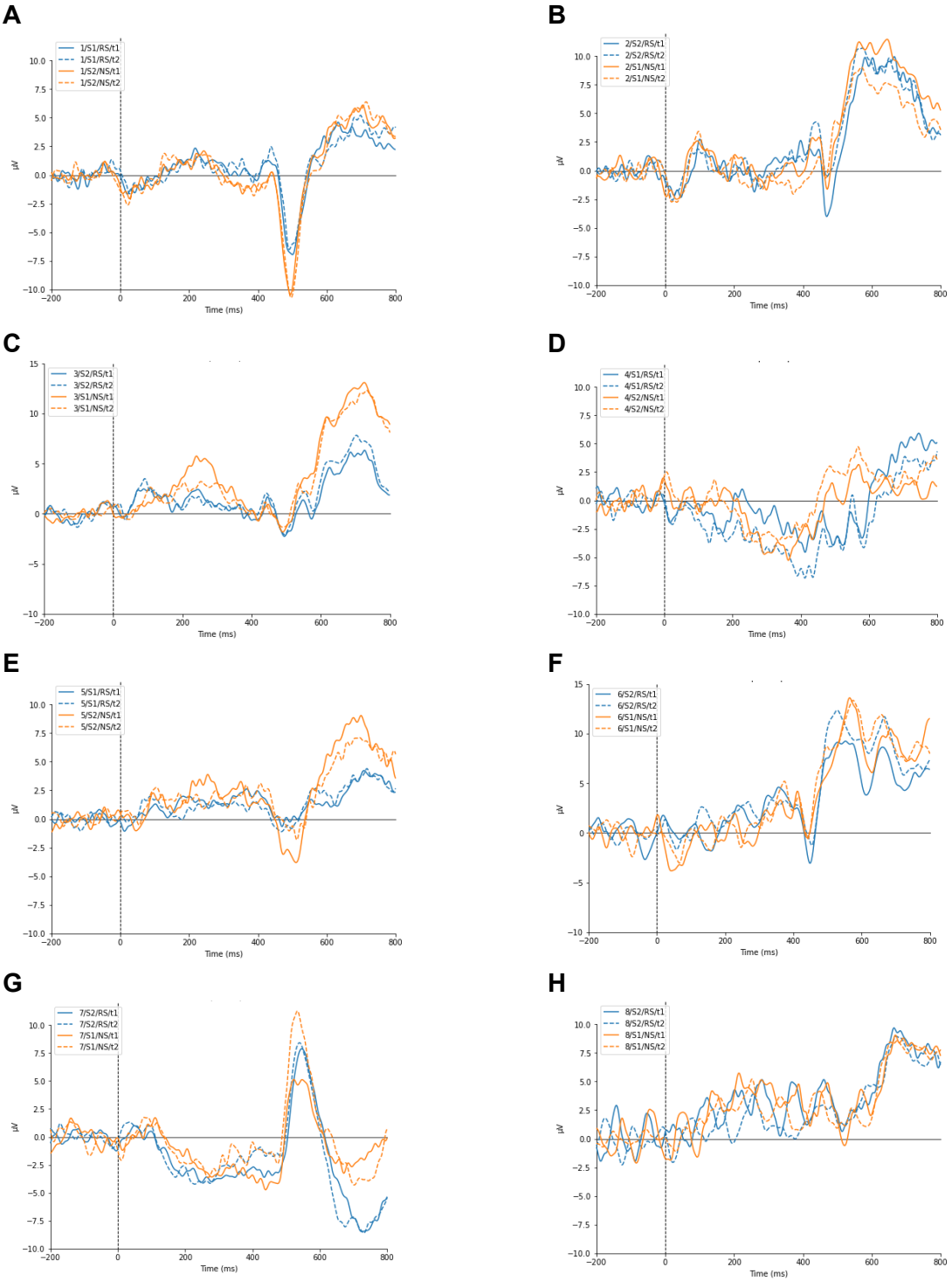


Note. **A** - **H** represent the frontal ERPs of, respectively, participants 1 - 8. S1 = first lab visit. S2 = second lab visit. NS = normal sleep. RS = restricted sleep. t1 = first 144 epochs. t2 = second

144 epochs. Y-axes for the plots regarding participants 6 and 7 are different from the other plots which have a y-axis running from -10 to 10 μV. Dashed vertical line represents stimulus onset time.

Figure H2

Parietal ERP Waveforms for Participants 1-8 per Sleep Condition and per Time Group.

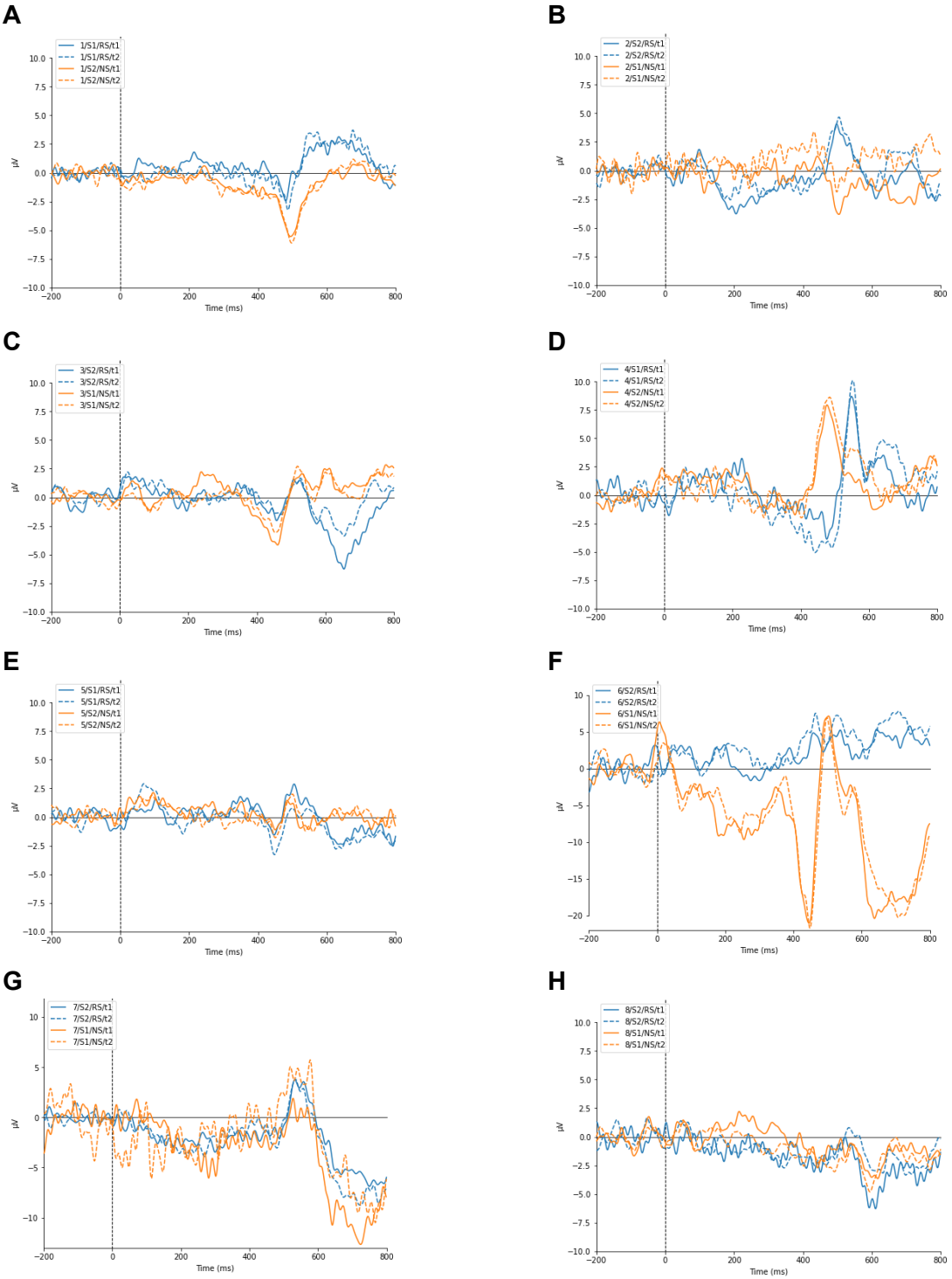


Note. **A** - **H** represent the parietal ERPs of, respectively, participants 1 - 8. S1 = first lab visit. S2 = second lab visit. NS = normal sleep. RS = restricted sleep. t1 = first 144 epochs. t2 = second 144 epochs. Y-axes for the plots regarding participants 3 and 6 are different from the other plots

which have a y-axis running from -10 to 10 μV. Dashed vertical line represents stimulus onset time.

Figure H3

Prefrontal ERP Waveforms for Participants 1-8 per Sleep Condition and per Time Group.

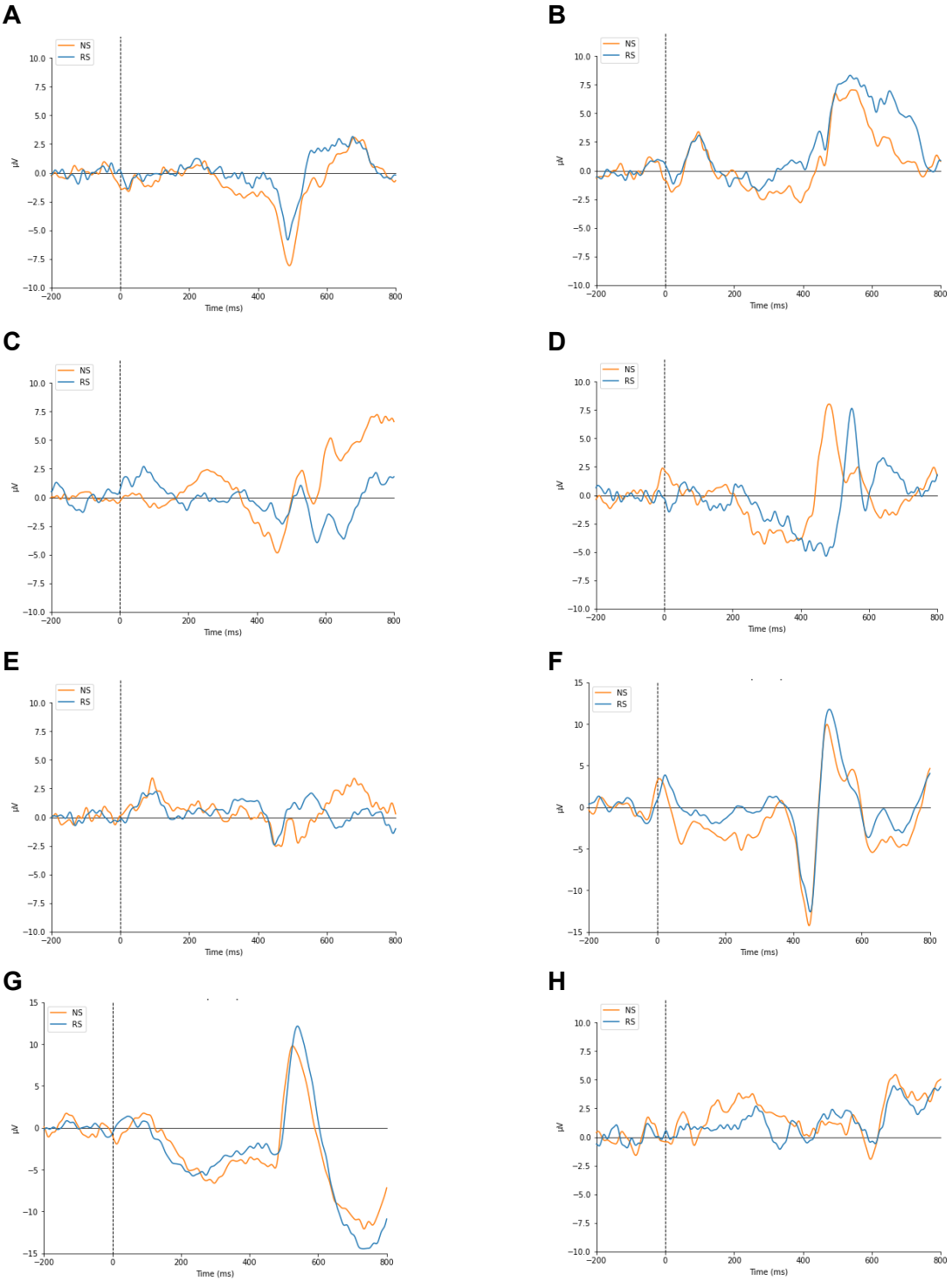


Note. **A** - **H** represent the prefrontal ERPs of, respectively, participants 1 - 8. S1 = first lab visit. S2 = second lab visit. NS = normal sleep. RS = restricted sleep. t1 = first 144 epochs. t2 = second 144 epochs. Y-axes for the plots regarding participants 6 and 7 are different from the

other plots which have a y-axis running from -10 to 10 μV. Dashed vertical line represents stimulus onset time.

Figure H4

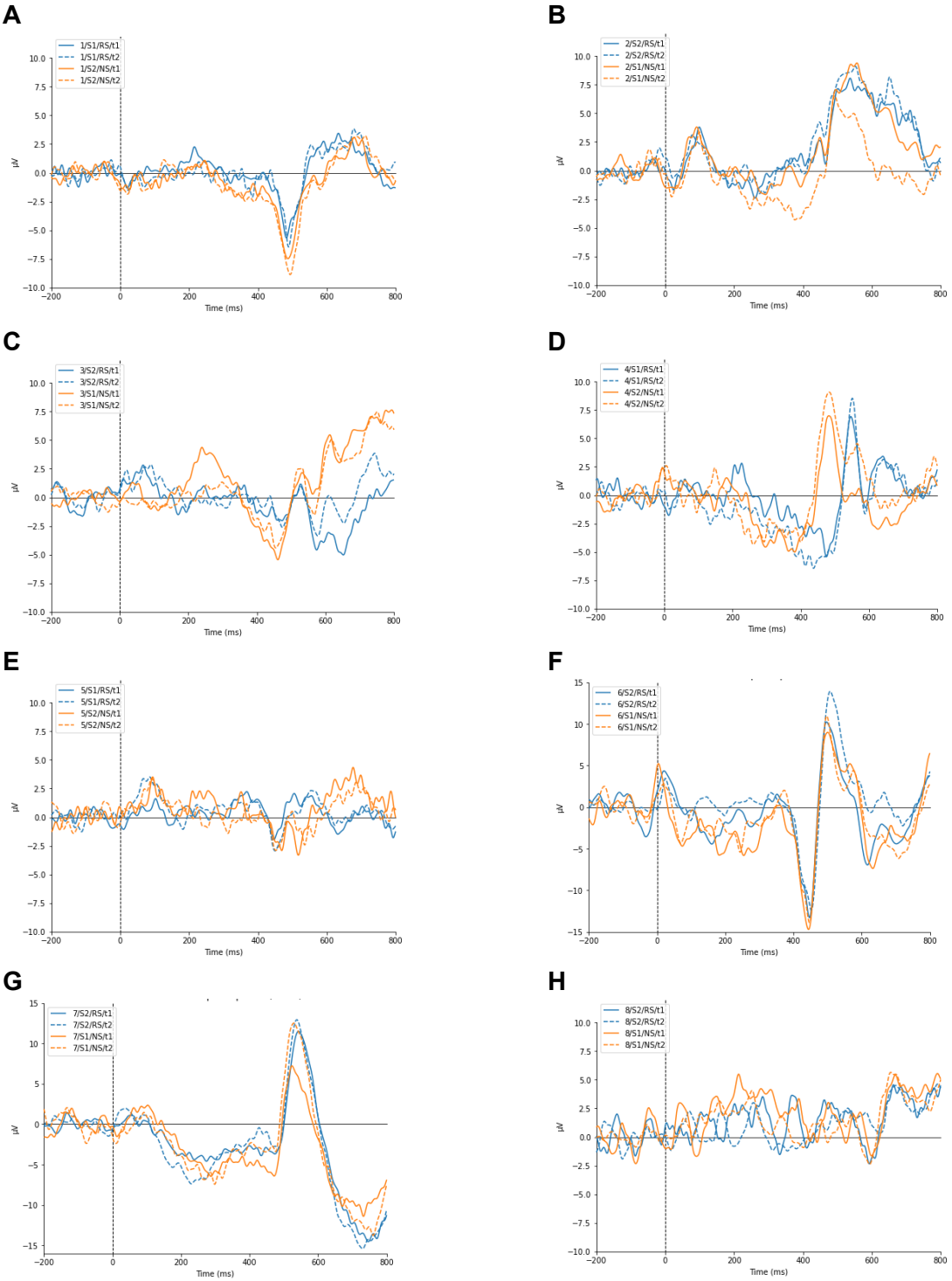
Central ERP Waveforms for Participant 1-8 for both Sleep Conditions.



Note. **A** - **H** represent the central ERPs of, respectively, participants 1 - 8. NS = normal sleep. RS = restricted sleep. Y-axes for the plots regarding participants 6 and 7 are different from the other plots which have a y-axis running from -10 to 10 μV. Dashed vertical line represents stimulus onset time.

Figure H5

Central ERP Waveforms for Participants 1-8 per Sleep Condition and per Time Group.



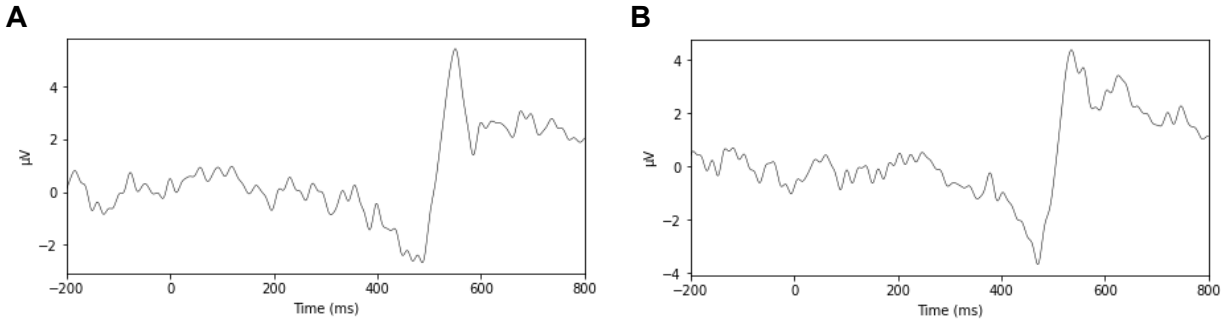
Note. **A** - **H** represent the central ERPs of, respectively, participants 1 - 8. S1 = first lab visit. S2 = second lab visit. NS = normal sleep. RS = restricted sleep. t1 = first 144 epochs. t2 = second 144 epochs. Y-axes for the plots regarding participants 6 and 7 are different from the other plots

which have a y-axis running from -10 to 10 μV. Dashed vertical line represents stimulus onset time.

Appendix I

Figure I1

Grand-average Frontal ERP Waveform for both Sleep Conditions.



Note. Grand-average frontal ERP waveforms for restricted sleep (**A**) and normal sleep (**B**). Waveform obtained by averaging over all eight participants.