HOW COGNITIVE FACTORS AFFECT SLEEP:
The impact of manipulated intention, instruction, and pre-sleep arousal on sleep quality in healthy sleepers

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Abstract

Sleep is important for health and well-being. Nevertheless, sleep is disturbed in about 20% of the population (e.g., by insomnia) with cognition (e.g., recurrent thoughts, rumination, or worry) making sleep harder. This dissertation therefore describes three cognitive models that illustrate the influence of cognition on sleep. The first model describes how negative cognitive activity leads to physical activation and to (emotional) stress. The second model adds a neurobiological component to the first in order to further substantiate this process. The last model tries to show this connection in healthy individuals and not only in patients. This is why the activation of "sleep/wake memories" and their embodiment is in the foreground. Additionally, a reactivation component during the night is included. The three studies attempt to substantiate these models and to show how changes in cognition before falling asleep influence nighttime sleep. This should lead to a better understanding of the relationship between cognition and sleep in healthy sleepers instead of patients.

The first study investigated the relationship between intention and sleep. Participants were instructed to improve or to worsen sleep intentionally. As expected, results confirmed that it is possible to sleep worse just by thinking about sleeping worse. Participants managed to increase their sleep onset latency, woke up more often, and decreased total sleep time. Furthermore, there was a misperception between objective and subjective measurements. This misperception is typical for patients with insomnia as well.

The second study aimed at looking at the relationship between pre-sleep (cognitive) arousal and its effects on sleep. Participants used social media intensively for 30 minutes before sleep, as previous studies have shown its arousing effects. Social media consumption was compared with a neutral control condition and a relaxing control condition, in which participants performed a progressive muscle relaxation session for 30 minutes. Results showed that social media consumption did not increase arousal and expected effects on sleep parameters were missing. Additionally, relaxation improved sleep despite the absence of a decreased arousal.

The aim of the third study was to explain the role of on-call instructions on sleep. Participants were instructed to respond to alarm sounds, which was compared to a neutral night. Furthermore, the study design introduced a sound-group with alarm sounds on both nights while the no-sound-group never got sounds. Results showed that the on-call instruction altered sleep quality, independently from the actual presentation of alarm sounds. Additionally, results showed a protective role of instruction on sleep on a neurophysiological level.

Taken together, these results confirm the relationship between cognition and sleep not only in patients with insomnia but in healthy sleepers as well. The three studies showed that the pre-sleep manipulation remained active during sleep and affected sleep quality. Out of the three models, the last model explained these results the best: the MemoSleep hypothesis states that pre-sleep cognitive activity is embodied through “sleep/wake memories” and (spontaneously) reactivated during the night. Thus, these “sleep/wake memories” affect sleep quality and recovery in the next morning. The results provided by this thesis provided initial evidence for the MemoSleep hypothesis. Future research should investigate this model and implement the targeted reactivation during the night not only with sounds but with the “sleep/wake memories” to confirm these preliminary data.

Zusammenfassung

Schlaf ist wichtig für die Gesundheit und das Wohlbefinden. Dennoch ist der Schlaf in ca. 20% der Bevölkerung gestört (z.B. durch Insomnie) mit Kognition (z.B. wiederkehrenden Gedanken, Ruminieren oder Sorgen), welche den Schlaf verunmöglichen. In dieser Dissertation werden daher drei kognitive Modelle beschrieben, welche den Einfluss von Kognition auf Schlaf verdeutlichen. Das erste Modell zeigt, dass negative kognitive Aktivität zu körperlicher Aktivierung und zu (emotionalem) Stress führt. Das zweite Modell ergänzt das erste noch um eine neurobiologische Komponente um die Prozesse weiter zu fundieren. Das letzte Modell versucht den Zusammenhang nicht nur bei Patient*innen aufzugeigen: Daher steht die Aktivierung von «Schlaf/Wach-Erinnerungen» und deren «Embodiment» im Vordergrund. Zusätzlich wird noch eine Reaktivierungskomponente während der Nacht eingebaut. Die drei Studien versuchen diese Modelle zu belegen und aufzuzeigen wie Veränderungen der Kognition vor dem Einschlafen den Nachtschlaf beeinflusst. Dadurch soll der Zusammenhang zwischen Kognition und Schlaf nicht nur bei Patient*innen besser verstanden werden.

Die erste Studie untersucht den Zusammenhang zwischen Wille und Schlaf. Dabei wurden den Versuchspersonen die Aufgabe gestellt, den Schlaf ohne weitere Hilfsmittel und allein durch Willenskraft zu verbessern oder zu verschlechtern. Erwartungsgemäss zeigte sich, dass eine Verschlechterung des Schlafs möglich war. Die Versuchspersonen schafften es die Einschlafzeit zu verlängern, häufiger Aufzuwachen und die gesamte Schlafzeit zu verringern. Dabei zeigte sich eine Fehlwahrnehmung zwischen der objektiven Messung mittels Polysomnographie und die subjektive Messung. Diese Inkongruenz ist ebenfalls typisch für Patient*innen.

Die zweite Studie sollte den Zusammenhang zwischen (kognitiver) Aktivierung vor dem Einschlafen auf den Schlaf zu zeigen. Dabei mussten die Versuchspersonen in den letzten 30 Minuten vor dem Einschlafen intensiv soziale Medien nutzen, da diverse Studien zeigen konnten, dass dies zu einer Aktivierung führt. Dies wurde mit einer neutralen und einer zweiten Kontrollbedingung verglichen, bei der die Versuchspersonen während 30 Minuten ein Entspannungs-training durchführten. Es zeigte sich, dass die Konsumation sozialer Medien zu keiner Aktivierung führte und erwartete Verschlechterungen des Schlafes ausblieben. Gleichzeitig zeigte sich eine Verbesserung des Schlafes durch Entspannungstraining trotz Ausbleiben einer Verringerung der Aktivierung.

Die dritte Studie wurde konzipiert, um die Rolle von Bereitschaftsdienst-Instruktionen auf den Schlaf zu klären. Die Instruktion während der Nacht auf Töne zu reagieren, wurde einer neutralen Nacht gegenübergestellt. Dabei wurde bei einer Gruppe in beiden Nächte Töne präsentiert während die zweite Gruppe nie Töne hörte. Die Studie zeigte, dass die Instruktion für Bereitschaftsdienst den Schlaf verändert und dies unabhängig von der tatsächlichen Präsentation von Tönen. Ebenfalls zeigte sich eine protektive Funktion der Instruktion auf Schlaf.

Insgesamt bestätigen den Zusammenhang zwischen Kognition und Schlaf auch bei gesunden Schläfer*innen. Die Resultate zeigen, dass die Manipulation oder Instruktion vor dem Schlaf, auch während dem Schlaf aktiv bleibt und die Schlafqualität beeinflusst. Dieser Zusammenhang kann am besten durch das dritte Modell erklärt werden, da die MemoSleep-Hypothese besagt, dass die kognitive Aktivität vor dem Einschlafen in unserem Körper zum Ausdruck gebracht (z.B. durch veränderten Herzschlag) und durch «Schlaf/Wach»-Erinnerungen abgespeichert werden. Diese körperliche Veränderung wird durch eine (spontane) Reaktivierung der Erinnerungen während der Nacht aufrechterhalten und beeinflusst dadurch die Schlafqualität sowie das Wohlbefinden am nächsten Morgen. Die vorliegenden Resultate geben einen ersten Beleg und die MemoSleep-Hypothese sollte in ergänzenden Studien noch weiter überprüft werden.

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List of Abbreviations

AASM	American Academy of Sleep Medicine	Professional society for the medical subspecialty of sleep medicine
ARAS	Ascending Reticular Activating System	Important for sleep-wake regulation and mainly active during wakefulness
CBT-I	Cognitive-Behavioral Therapy for Insomnia	Psychotherapeutic treatment developed to help patients with insomnia
ECG	Electrocardiogramm	Measuring heart rate
EEG	Electroencepahlogram	Measuring brain activity with electrophysiological potential
EMG	Electromyogram	Measuring muscle activity and muscle tone
EOG	Electrooculogram	Measuring eye movements
ERP	Event-Related Potential	Electrophysiological response to a stimulus (e.g. specific sensory, cognitive, or motor event)
FoMO	Fear of Missing Out	A person is afraid of missing out on something going on in the social bubble
FoMS	Fear of Missing a Sound	A person is afraid of missing out on a sound during an on call-night (for example not waking up when the alarm rings)
HR	Heart Rate	Speed of the heartbeat (measured by the number of beats per minute (bpm))
HRV	Heart Rate Variability	Physiological phenomenon of variation in the time interval between heartbeats
NREM	Non-Rapid Eye Movement	Non-REM sleep (sleep stages N1, N2, N3)
NWAK	Numbers of awakening	Number of awakening / wake reaction from sleep
PAL	Pair Associated Learning task	Episodic memory task with semantically associated word pairs
PFC	Pre-Frontal Cortex	Part of the cerebral cortex which covers the front part of the frontal lobe
PMR	Progressive Muscle Relaxation	Relaxation technique
PNS	Parasympathetic Nervous System	The parasympathetic system is responsible for activities that occur when the body is at rest

PSG	Polysomnography	Gold-standard to measure sleep objectively (containing EEG, EOG, EMG, ECG, and Respiration)
PSQI	Pittsburgh Sleep Quality Index	A self-report questionnaire that assesses sleep quality over the last four weeks
PTSD	Post-Traumatic Stress Disorder	A mental disorder that can develop after exposure to a traumatic event
PVT	Psychomotor Vigilance Test	Test to measure the effect of sleepiness on vigilance
REM	Rapid Eye Movement	REM sleep
SD	Standard Deviation	Measure of the amount of variation of a set of values
SEM	Standard Error of Means	Standard deviation of a statistic (i.e. sample mean) sampling distribution
SFA-R	Schlaf-Fragebogen A, revised	Questionnaire to rate sleep quality subjectively in the morning
SNS	Sympathetic Nervous System	The sympathetic nervous system is responsible to stimulate the body's fight or flight response.
SOL	Sleep Onset Latency	Time it takes a person to fall asleep
SWA	Slow Wave Activity	Oscillatory activity in a frequency of 0.5 – 4.5 Hz
SWS	Slow Wave Sleep	Period of sleep where the EEG activity is characterised by slow waves with a frequency range of 0.5–4.5 Hz
TST	Total Sleep Time	Time from light out in the evening to light on in the morning (including all sleep stages and waketime)
WASO	Wake After Sleep Onset	Time a person spends awake after turning of the light
VLPO	VentroLateral PreOptic area	Area of the hypothalamus involved in the sleep-wake regulation and mainly active during sleep

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1 Introduction

Most of us have probably experienced the feeling of lying in the bed and thinking about an upcoming event. This event might be a positive one, such as going on vacation the next day, or a negative one like an exam or an oral presentation. We cannot stop thinking about what will happen tomorrow. Have we thought about everything? Did we miss anything? Thinking constantly and in detail about things that have happened – or not – in the past is called rumination, which is defined as "the act of thinking deeply about something" (Oxford Advanced Learner's Dictionary, 2021). Rumination is associated with sleep disorders such as insomnia, and reported by the most insomnia patients (Pillai & Drake, 2015). In the course of this conceptual introduction, key models, research gaps, and prospects will be illustrated. The following chapter will first explain the cognitive model of insomnia (Harvey, 2002, see chapter 1.2.1), which describes that rumination and worry prevent patients from sleeping. Secondly, the hyperarousal model of insomnia (Riemann et al., 2010, see chapter 1.2.2) will be described, explaining that rumination and worrying about sleep creates a hyperarousal on a physiological level, thereby causing changes on a neurophysiological level and leading to insomnia. So far, most studies and models are based on patients with insomnia. To close this important research gap, the last chapter of this introduction describes the MemoSleep hypothesis (Rasch, 2016, see chapter 1.2.3), which explains how pre-sleep cognition and embodied sleep/wake memories might affect sleep in patients as well as in healthy sleepers.

The following sections lay out the theoretical background on which the papers of this dissertation will be based on. In the beginning, this thesis will provide an overview on sleep, as humans spend about one third of their lifetime in this brain state. Afterwards it will describe in detail the three models mentioned previously in order to explain the relationship between cognition and sleep. Thereafter, it will outline several methods to actively affect pre-sleep cognition (i.e. with social media) and that affect ongoing processes during sleep (i.e. with a pre-sleep on call-instruction). Against this background, three main hypotheses will be constructed for the gaping holes in research, which will be investigated in the following three manuscripts.

1.1 Sleep

First, this section starts with a description of sleep. Sleep is defined as a state of reduced consciousness with unresponsiveness to external stimuli as well as perceptive disengagement and physiological inactivity (Carskadon & Demet, 2011). Sleep is regulated by complex neurobiological mechanisms providing the shift between sleep and wakefulness with a certain periodicity (Borbély & Achermann, 1999). Based on the Borbély's (1982) two-process model there are two individual processes regulating the shift between wakefulness and sleep (see Figure 1). A first process is the drive to sleep (Process S) which represents sleep homeostasis. During process S sleep pressure accumulates during the day up until the point in which an individual is required to sleep, thereby translating into an increase of slow wave sleep (SWS) during the night. After falling asleep, the pressure to sleep decreases and a person wakes up, when it reaches its minima. The second process of the two-process model is the circadian rhythm (Process C), which is regulated by the inner clock. Almost all body functions (e.g. body temperature) and cells follow a circadian pattern of approximately 24 hours. At the same time, Process C calculates the optimal time for a person to sleep. As the circadian rhythm differs between people, this Process C differs too. Following Borbély (1982), in most people the minimum is around 4 am, time at which a person is the most tired, whereas the climax is around 4 pm. Figure 1 shows the interaction of this two processes.

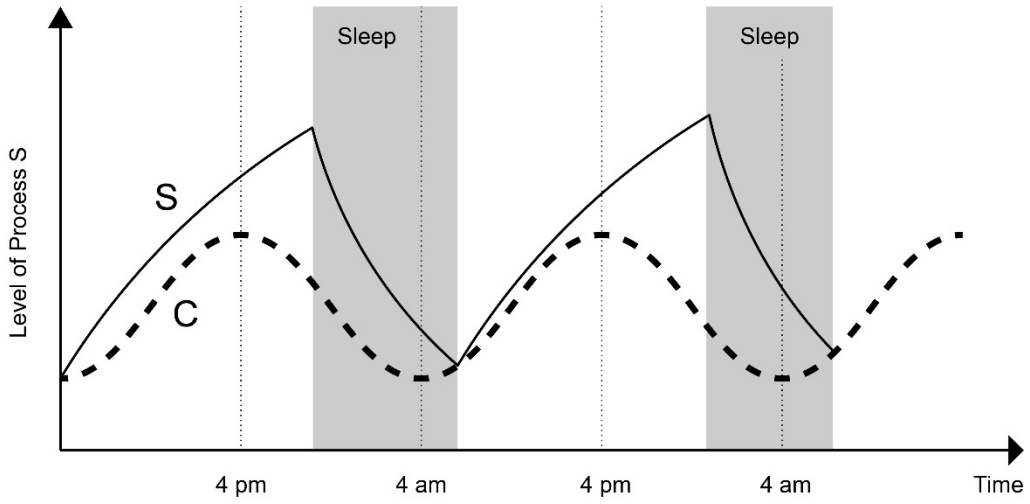

Figure 1. Two-Process Model of Sleep Homeostasis (adapted from Borbély (1982)). The model shows the two processes based on the model of Borbély: Process S (solid line) and Process C (negative function; dashed curve). Process S represents the drive to sleep and increases during the day until sleep onset, where it starts decreasing (grey bars). This is coupled with the amount of slow wave sleep. Meanwhile Process C cycles from its maximum (approximately 4 am) to its minimum (approximately 4 pm, when people are most tired) and represents the circadian rhythm.

1.1.1 Sleep Architecture

As mentioned above, sleep is characterized by its reduced consciousness while the brain remains very active during sleep and dynamically alternates between different sleep stages in a cyclic pattern (Rasch & Born, 2013). Sleep is typically separated in two major phases: Rapid-Eye-Movement (REM) and Non-REM (NREM) sleep. The latter can be decomposed into three stages: NREM sleep stage 1 (N1), NREM sleep stage 2 (N2) and NREM sleep stage 3 (N3) (Iber et al., 2007). Following the American Association of Sleep Medicine's (AASM, Iber et al., 2007) classification, N1 is characterized as a transition stage between wakefulness and sleep and covers about 5-10 % of a person's regular night sleep (total sleep time, TST). During N1 the EEG activity slows down: from alpha (8-13 Hz) down to a mixed frequency (predominantly between 4-7 Hz) while the EOG shows slow rolling eye movements. N2 sleep covers 45-55% of TST and is thereby the predominant sleep stage (Kirsch, 2014). K-complexes and sleep spindles characterize N2 sleep. K-complexes are defined as a "well-delineated negative sharp wave immediately followed by a positive component […] with total duration ≥ 0.5 seconds" (Iber et al., 2007, p. 26). Secondly, sleep spindles are characterized by fast EEG frequency bursts between 11-16 Hz with at least a 0.5-second duration. The third NREM sleep stage is N3, which is the deepest sleep stage. Therefore, this sleep stage is also known as deep sleep or slow wave sleep (SWS). It contains slow waves of frequencies between 0.5-4.5 Hz with a high amplitude of > 75 μV. N3 covers about 15-25 % of TST. The remaining 20-25 % of TST are covered by REM sleep. As its name already posits, REM sleep is characterized by rapid eye movements: "conjugate, irregular sharply peaked eye movements with an initial deflection usually lasting < 500ms" (Iber et al., 2007, p. 27) visible in the EOG. The EMG displays the lowest muscle tone (atonia) compared to the other sleep stages. Moreover, similar to wakefulness, brain activity in REM shows low amplitudes and mixed frequency EEG including fast theta activity (4-8 Hz), explaining the "paradoxical sleep" term by which it is also known (Peigneux, Laureys, Fuchs, et al., 2001). The term of so-called dream sleep is often associated with REM sleep and remains incorrect, as dreams do not only occur during REM although they appear most emotional, intense, and vivid in this stage (Hobson, 2005; Kirsch, 2014; Stephan et al., 2021). Sleep stages follow a cyclic pattern of approximately 90 minutes (Feinberg & Floyd, 1979), which is pictured in a so called hypnogram (see Figure 2).

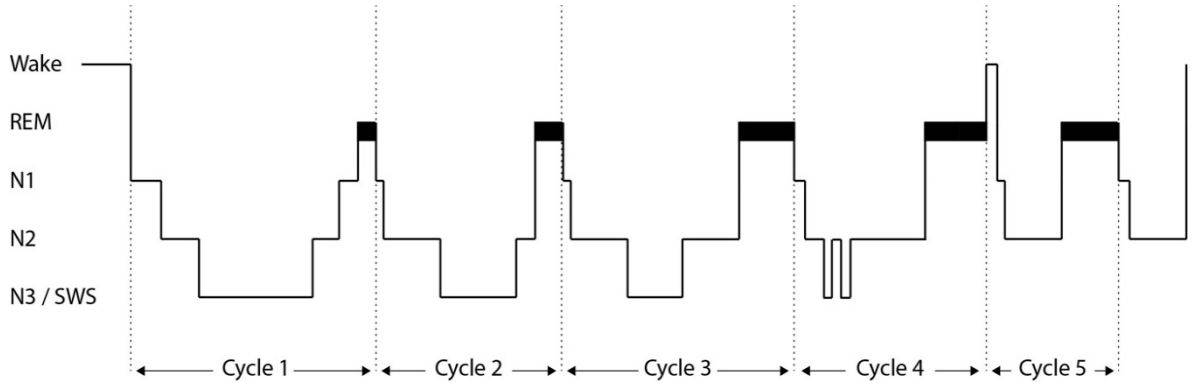

Figure 2. Hypnogram of a night of sleep. A hypnogram displays the distribution of all sleep stages (N1-3, REM) as well as the wake periods (WASO) over one night. The y-axis displays the different stages while the x-axis represents the time from going to bed (very left) to waking up in the morning (very right). This typical hypnogram shows the cyclic alteration between NREM and REM sleep (Feinberg & Floyd, 1979), and the predominance of NREM (especially N3/SWS) in the first half of a night and the dominance of REM sleep in the second half.

1.1.2 Functions of Sleep

Aside from reduced consciousness, sleep is a very interesting and important brain state. So far, we know about sleep architecture, which is a characteristic of sleep. The aim of this section is to deepen the knowledge about its functions. Research has shown, that sleep is important for different bodily functions. For example, beneficial metabolic processes take place during sleep. One of these processes is the cleaning of metabolic waste products in the brain via cerebrospinal fluid (Xie et al., 2013). Following this line, a recent study showed that cleaning metabolic waste is even associated with the low oscillatory frequencies involved in memory consolidation and neural computation (Fultz et al., 2019). In addition, it has been shown that during non-rapid eye movement sleep (NREM) in particular, slow oscillations are involved in neural computation and support memory consolidation (Diekelmann & Born, 2010; Levenstein et al., 2017; Rasch et al., 2007). Moreover, sleep not only affects metabolic clearance or memory consolidation positively, it also helps to strengthen our immune system. A review by Besedovsky and colleagues (2012) concluded beneficial effects of sleep on the expression of T-cells[1]. They established that sleep plays an important role in the adaptive immune response as it helps to create an immunological memory, especially with respect to the role of slow wave sleep (SWS) as it promotes beneficial effects on immune system.

[1] T-cells are one type of the lymphocytes (white blood cells). These cells are important in the adaptive immune response (Murphy & Weaver, 2016)

One important function of sleep is its role in memory consolidation (Rasch & Born, 2013). Research on memory and sleep dates back a long time. Already in the 17th century, Ebbinghaus presented a beneficial effect of sleep on memory by showing reduced forgetting after sleep (Ebbinghaus, 1885). Almost 40 years later, in the first experimental study, Jenkins and Dallenbach (1924) showed that sleep affects learning and memory consolidation. They showed that participants remembered more nonsense syllables when they were allowed to sleep for two hours compared to a wake-group. This was the starting point for various studies showing beneficial effects of sleep on memory consolidation (e.g. Andrillon et al., 2017; Barrett & Ekstrand, 1972; Diekelmann & Born, 2010; Drosopoulos et al., 2007; Fowler et al., 1973; Peigneux, Laureys, Delbeuck, et al., 2001; Rasch & Born, 2013).

A next function of sleep is to help process emotions. Palmer and Alfano (2017) showed that sleep loss increases experience of negative emotion. REM sleep in particular is linked to emotions and emotion processing as well as its regulation. It has been shown that the affective and emotional brain homeostasis is supported by REM sleep (Goldstein & Walker, 2014). This homeostasis prepares an organism for the next day providing optimal emotional and social functioning. Van der Hem and Walker (2009) showed that, during REM sleep, emotion-related brain regions are activated, which in turn successfully reactivate emotional events. They call this "sleep to forget and sleep to remember" (Van Der Helm & Walker, 2009, p. 14), as the emotional tone is forgotten overnight whereas the targeted memory of an emotional episode is remembered. REM sleep strengthens the declarative memory-component of emotions and weakens the affective tone at the same time. Thus, sleep helps to process, reactivate, and regulate emotions. This is especially important for the development of post-traumatic stress disorder (PTSD) as patients often suffer from nightmares. In these patients it seems that the separation of affective tone and emotional memory components does not work (Van Der Helm & Walker, 2009). The reason for this lack of separation is still unknown, but it has been shown that sleep after a trauma, REM sleep is protective against the development of a trauma in healthy sleepers (Kleim et al., 2016).

Sleep is not only associated to PTSD but also with various mental disorders. For example acute sleep deprivation increases symptoms of anxiety and depression (Babson et al., 2010). This relationship is shown to be bidirectional: sleep quality is reduced in patients suffering from mental disorders and on the other hand treating the disturbed sleep (e.g. insomnia) can reduce symptoms of mental illness (Freeman et al., 2020; Wulff et al., 2010). One example of mental illness where better sleep quality prevents the severity of symptoms is

schizophrenia. Studies have shown that delusions and psychotic experiences decrease when sleep disruption is reduced (Freeman et al., 2009; Myers et al., 2011)

Additionally to psychological health, a recent meta-analysis (Schrimpf et al., 2015) confirmed helpful effects of sleep on physiological health as well. Especially for pain perception, the authors confirmed an increase in the severity of symptoms after sleep deprivation. They showed that sleep deprivation increased subjectively and objectively reported pain therefore implying that reduced sleep quality increases pain in chronic patients but that enhancing it could lead to a decrease in the severity of pain perception (Bonvanie et al., 2016; Kundermann et al., 2004; Lautenbacher et al., 2006). Another beneficial effect of sleep is its effect on Alzheimer's disease[2]. From studies with patients is known that disturbed sleep is associated with the development of dementia or cognitive diseases in general (Ohayon & Vecchierini, 2005; Shi et al., 2018). Typical biomarkers of Alzheimer's diseases increased after a night of sleep deprivation in memory-associated brain regions in healthy participants (Shokri-Kojori et al., 2018). Although there are no studies with patients, the results might indicate promising new treatment options for Alzheimer's disease based on enhanced sleep quality of Alzheimer's disease (Wunderlin et al., 2020).

To sum everything up, sleep is a reduced state of consciousness with a wide range of distinct activity in the EEG. It can be separated into different sleep stages following a cyclic pattern over the night, known as sleep architecture. Sleep has various functions and is beneficial in many ways. We have seen that sleep is important for different body functions, memory consolidation, or health. Aim of this thesis is to explain the relationship between sleep and cognitive functions. We thereby define cognition as "any directedness in one's thoughts or behaviors, whether or not this involves conscious decision making" (APA Dictionary of Psychology, 2021). This means cognitive functions that are involved in the will of a person to plan or to act out a plan – with or without its awareness. To see this relationship between sleep and cognition, in the next sections of this thesis would like to explain the three models (cognitive model of insomnia (Harvey, 2002), hyperarousal model of insomnia (Riemann et al., 2010), and MemoSleep hypothesis (Rasch, 2016)) in more detail. These models help to explain what is known – or not – about cognitive functions and their influences on sleep and vice versa.

[2] Alzheimer's diseases is a neurodegenerative disease where patients start to forget to remember events, occasions and / or person. On a neurophysiological level it is associated with growing amount of beta amyloid plaques and increasing neurofibrillary tangles, as well as the loss of neuronal connections (Deardorff & Grossberg, 2019)

1.2 Cognition and Sleep

So far, one study looked at the interrelationship between cognitive factors (e.g. willpower, intention, or instructions) and sleep in healthy sleepers. Schmidt and colleagues (2018) showed that in perfectionistic individuals, concerns and doubts were positively associated with the severity of insomnia symptoms, although participants did not report any diagnosed insomnia. The study further showed a positive correlation between those two cognitive arousing factors with negative emotions and thoughts (e.g. guilt, regret, and shame): the more participants reported concerns and doubts, the more they reported negative emotions and thoughts at bedtime, which is in line with symptoms of insomnia. This leads us to the question, how is cognition involved in insomnia or sleep in general?

1.2.1 Cognitive Model of Insomnia

One explanation for this connection between cognition (e.g. rumination) and insomnia is given by the *cognitive model of insomnia* (Harvey, 2002, see Figure 3). The bases for this first model are cognitive models of anxiety disorders as well as the finding that insomnia patients worry and ruminate excessively about sleep-related concerns (Harvey, 2002; Morin, 2004). The model explains how negatively toned cognitions about sleep lead to insomnia. Patients with insomnia worry excessively about their sleep (e.g. getting enough sleep) and its consequences for daytime functioning and health. On a physiological level, rumination and worry lead to an enhanced activity of the sympathetic nervous system (SNS) and thereby create an arousal. On a cognitive level, these cognitive processes lead to emotional distress and a state of anxiety (Harvey, 2002; Harvey et al., 2005). This anxious state transfers attention selectively towards sleep, sleep monitoring, and other sleep related threat cues. Patients start to monitor internal processes (e.g. body sensations) and their environment with a *sleep threat filter*, which only allows negative aspects of sleep to enter thoughts and provides additional reason for rumination and worry. Another aspect of this sleep threat filter is its selective attention, which initially leads to a distorted perception of a sleep deficit and after a certain time to a real deficit of sleep and insomnia. A patient with a subjective sleep deficit worries even more about her/his sleep, which triggers the negatively toned cognitive activity and thereby worsens the arousal and distress even more. Exacerbating processes like erroneous beliefs about sleep (e.g. “When I haven’t slept well, I must take the day easy” (Harvey, 2002, p. 879)) triggers cognitive activity and boosts physiological arousal. Additionally, an insomnia patient implements safety

behaviours, which should help to deal with the negative cognitive activity but are often maladaptive coping strategies (e.g. excessive time in bed, daytime naps).

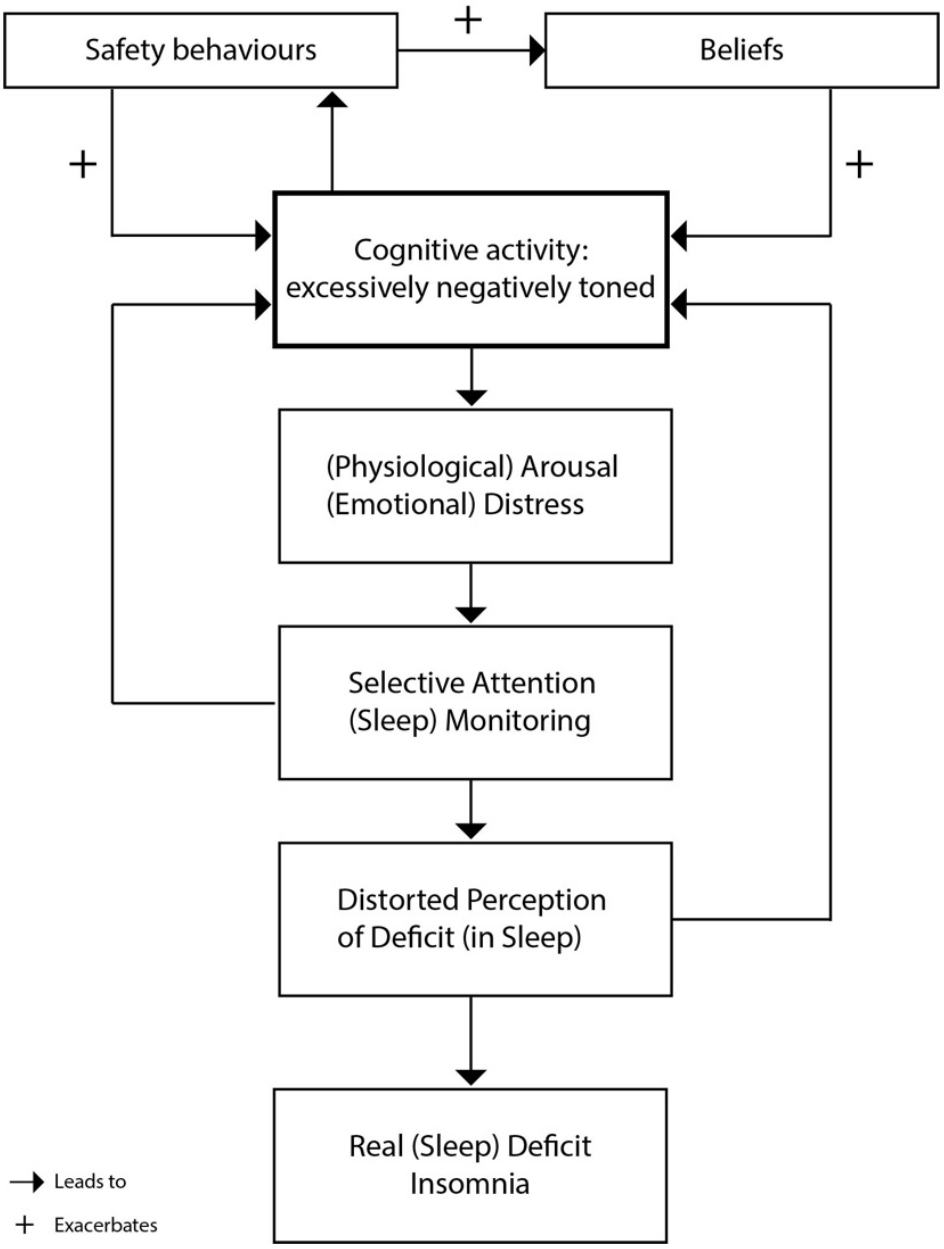

Figure 3. Cognitive Model of Insomnia adapted from Harvey (2002). The entry point of the model is characterized by negatively toned cognitive activity about getting enough sleep and its daytime consequences. This rumination creates an autonomic reaction (arousal) as a result of the body's fight-or-flight-reaction coupled with (emotional) distress leading to a state of anxiety. This state shifts attention towards sleep-related cues and thoughts (i.e. indicators of not functioning well after a night of less sleep) and supports the negatively toned cognition. The selective attention towards sleep supports the perception of a sleep deficit although this must not be the case and supports the negative cognition even more. Finally, this leads to real sleep deficits and insomnia. Additionally, patients adopt safety behaviours to cope with the excessive cognitive activity which exacerbate them even more and supports erroneous beliefs.

To sum it up, the cognitive model of insomnia explains how negative toned cognition leads to insomnia by creating physiological arousal and emotional distress. Patients shift their attention towards sleep, its monitoring and its threats. Additionally, maladaptive safety behaviours and beliefs trigger the negative cognitive activity even more and thereby force the development and conservation of insomnia. Although this is a good model, giving a first explanation about the connection between sleep and cognition for patients suffering from insomnia, there are still remaining questions. Is there an effect of cognition on the neurophysiological level? Is there a difference of its effect on the long run? What are the cognitive processes behind a chronification of insomnia? These questions might find answers in the hyperarousal model of insomnia (Riemann et al., 2010), which will be explained in the next section.

1.2.2 Hyperarousal Model of Insomnia

Adapted from the cognitive model of insomnia (Harvey, 2002), Riemann and colleagues (2010) introduced the *hyperarousal model of insomnia* as another explanation for how cognition affects sleep. In addition to the cognitive model of insomnia, sleep is not only linked to changes on a cognitive-behavioral level, but also to changes on a neurobiological level. The hyperarousal model of insomnia (Riemann et al., 2010, see Figure 4) illustrates how a cognitive-behavioral hyperarousal together with a neurobiological cascade can lead to chronic insomnia. On a cognitive level the model is close to the cognitive model of insomnia: psychosocial stress leads to rumination and worrying. This is the case in an acute insomnia (< 90 days) where patients show sleep related problems such as enhanced sleep onset latency (SOL), increased time spent awake after sleep onset (WASO) and a reduced total sleep time (TST). If this continues for months, individuals enter the stage of a subchronic insomnia (3-6 months). Here individuals have more and more sleep related worries, they shift their attention towards sleep and sleep related thoughts. They worry during the day (and night) about sleep loss and its consequences for the next day's functioning. Moreover, they increasingly adopt negative behaviours. For example, patients stay in bed longer than needed for sleep (prolonged time in bed) or they engage in naps during the day to compensate for their loss of sleep during the night. This negative behaviour is maladaptive for the treatment of insomnia and enhances the possibility to develop a chronic insomnia, when participants condition themselves to these negative behaviours. Parallel to the cognitive-behavioral level, on a neurobiological level, stress, excessive rumination, worry, and inadequate coping increases the level of stress

hormones (e.g. cortisol) and excitatory neurotransmitters (e.g. monoamine). This is the human body's adaptive way to cope with such situations. At the same time, levels of inhibitory neurotransmitters (e.g. adenosine) and serotonin decrease. Consequently, there are acute changes of the sleep-wake regulation showing more activation in the ascending reticular activating system (ARAS, which is mainly active during wakefulness) and/or less activation in the ventrolateral preoptic area of the hypothalamus (VLPO, which is mainly active during sleep). This leads to acute changes of the sleep wake regulation creating a cortical hyperarousal. If this hyperarousal continues for months, it leads to a dysregulation in the two processes of sleep homeostasis (Borbély, 1982) and a chronification of the insomnia. Chronic insomnia patients suffer of chronic (cortical) hyperarousal, which is present day and night. This is associated with changes in the autonomic nervous system like increased heart rate or enhanced cortisol levels (Spiegelhalder et al., 2011; Vgontzas et al., 2001). Further the previously acute changes in the sleep-wake regulation (e.g. enhanced activity ARAS and/or decreased activity in the VLPO) are now chronic. Together with the cognitive behavioral changes, this leads to insomnia as well as to comorbid psychopathologies (e.g. anxiety or depression).

To conclude, the hyperarousal model of insomnia postulates that cognition directly and indirectly affects sleep quality negatively thereby initially leading to an acute insomnia, then subchronic, and finally a chronic state of insomnia (Riemann et al., 2010; Wicklow & Espie, 2000). Moreover, it explains how the obsession about sleep-associated thoughts (cognition) affect sleep quality negatively on a long run and creates a chronic hyperarousal on both a cognitive and especially on a physiological level (autonomic hyperarousal). In the next section, this thesis will give a possible explanation for the relationship between cognition and sleep in healthy sleepers instead of insomnia patients by introducing the MemoSleep Hypothesis.

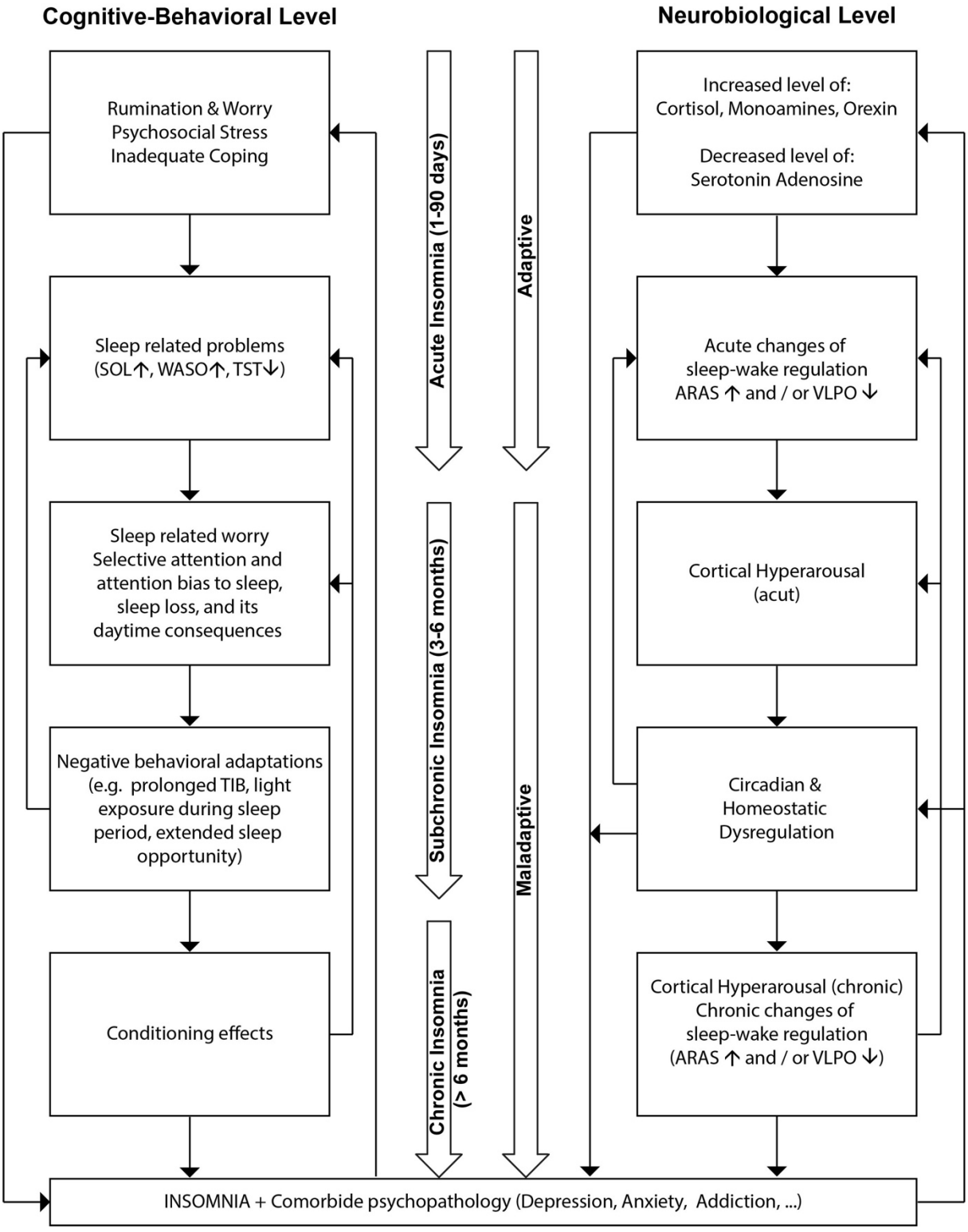

Figure 4. Hyperarousal Model of Insomnia adapted from Riemann et al. (2010). The model shows two different Levels which interfere with each other and finally lead to insomnia. First, on a cognitive-behavioral level, Rumination leads to sleep related problems (prolonged sleep onset latency (SOL) and wake time after sleep onset (WASO), shorter total sleep time (TST). The insomnia becomes subchronic before it gets to a chronic level due to conditioning effects of maladaptive cognition and behavior. Secondly this model implements a neurobiological level, which explains the changes in the brain metabolism, in the ascending reticular activating system (ARAS) as well as in the sleep inducing activation in the ventrolateral preoptic area of he hypothalamus (VLPO), and finally creating a cortical hyperarousal leading to dysregulation of the circadian and homeostatic systems.

1.2.3 Memories-of-Sleep (MemoSleep) Hypothesis

Based on the two previous models, we have seen that cognition affects sleep negatively. Rumination, worrying and focussing on sleep are some of the maintaining characteristics in the development and chronification of insomnia. Although this is an important topic, as the prevalence of sleep disorders has highly increased in our society (Statista, 2021), and a certain importance has been given to how cognition affects sleep in healthy sleepers. Therefore aim of this section is to introduce the *Memories-of-Sleep (MemoSleep) hypothesis* (Rasch, 2016, see Figure 5). This hypothesis attempts to explain how cognition affects sleep for not only insomnia patients but also in healthy sleepers.

The MemoSleep hypothesis is based on three aspects: the first is the pre-sleep activation of wake or sleep-related cognition, which – as a second aspect – is embodied as *wake or sleep memories*. Assuming that on the one hand the activated pre-sleep cognitions are wake-related (wake memories) they lead to an increased arousal and somatic tension. On the other hand, in case of pre-sleep activated sleep related memories referred as sleep memories, the arousal decreases, and the person finds him- or her-self in a state of relaxation. As a third aspect of this hypothesis, these sleep or wake memories are reactivated spontaneously during the night or while sleeping, see Figure 5. This spontaneous reactivation depends on the degree of pre-sleep activation, which in turn affects the state of pre-sleep arousal, thereby also influencing sleep quality as well as the sensation of recovery after getting up. In the case of sleep memories, sleep quality will be enhanced whereas in case of wake memories the sleep quality decreases. Therefore, the hypothesis explains why we sleep badly the night before going on vacation (positive wake memory) or the night before an exam (negative wake memory). For example, thinking about an exam (wake-related cognition) will induce a negative pre-sleep state thereby activating bodily systems (embody the wake-related cognition) that increases arousal and somatic tension. This is visible for example with an increased heart rate, EMG activity, breathing rate, and cortisol level. During sleep this embodied state of “exam” (including cognitive and somatic arousal) is reactivated and therefore decreases SWS, sleep quality and reduces recovery after sleep. Positive wake-related cognitions (e.g. vacation) lead to the same bodily function and thereby have the same consequences during the night as negative wake-related cognitions. At the same time, the MemoSleep hypothesis provides an idea to why pre-sleep relaxation techniques (e.g. progressive muscle relaxation (PMR) (McCloughan et al., 2014; Simeit et al., 2004) or hypnosis help to enhance sleep quality (positive sleep memory, see e.g. Cordi et al., 2014) or why thinking about snoring (negative sleep memory) does not affect sleep quality negatively. Here, PMR or a sleep-inducing hypnosis (sleep-related cognition) will

induce a positive pre-sleep state, embody the sleep-related memories that decreases arousal and somatic tension thereby decreasing the activity of physiological parameters (e.g. heart or breathing rate, cortisol level or EMG activity). During sleep this embodied state of "relaxation" is reactivated and therefore SWS, sleep quality and the post-sleep recovery will increase. Negative sleep-related cognitions (e.g. thinking about snoring) are embodied the same way as the positive ones and thereby create the same bodily states (decreased arousal and somatic tension), which are reactivated and imply the same consequences during the night and the next morning (better sleep quality and recovery and decreased arousal).

Concretely, the MemoSleep hypothesis explains the relationship between sleep and cognition in healthy sleepers, but it can also be applied in case of insomnia. For example, an insomnia patient activates wake memories before going to sleep (e.g. rumination about sleep threats). These wake memories are already embodied and lead to an enhanced arousal and somatic tension. During the night, these wake memories are reactivated spontaneously, and the somatic tension is kept high until next morning, where patients do not feel recovered at all. Here, the MemoSleep hypothesis can explain why rumination or worry lead to worse sleep.

Even though the MemoSleep hypothesis explains how cognition might affect sleep in healthy sleepers, there is a lack of studies confirming this hypothesis. Despite the fact that the two previously discussed models confirm that mental activity and thoughts can disturb sleep in insomnia patients, the underlying mechanisms are not fully understood. Although the MemoSleep hypothesis provides an initial idea of how mental activity might disturb sleep, it is not clear to what degree we can influence healthy sleep simply with the power of our will. So far, no studies looking at the effect of willpower on sleep have been conducted. This research gap will be filled with the first manuscript that will be discussed in the next sections of this thesis. In this project, we were interested in whether the intention to change sleep actually affects sleep. This was achieved by giving the participants the instruction to willingly affect sleep quality.

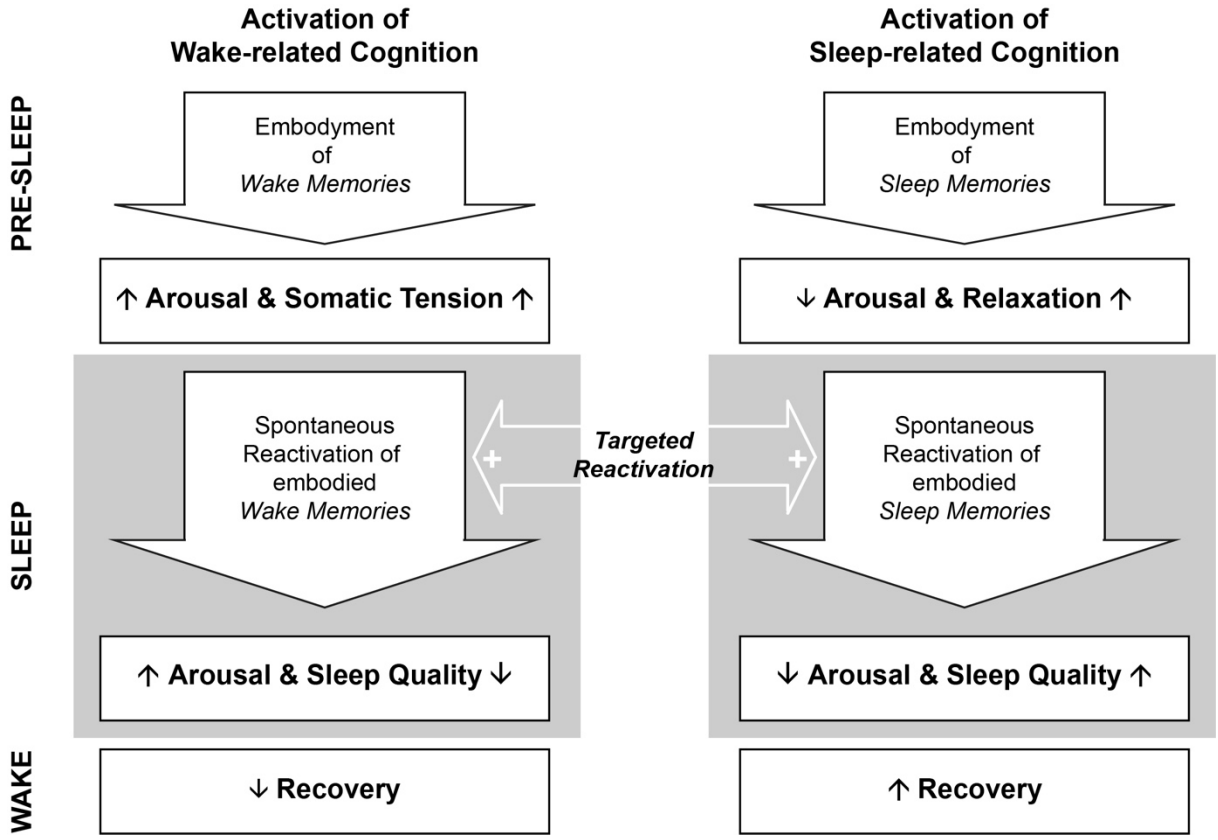

Figure 5. Model of the MemoSleep Hypothesis. The MemoSleep hypothesis (Rasch, 2016) explains how pre-sleep activation of wake-related cognition leads to decreased sleep quality and recovery whereas pre-sleep activation of sleep-related cognition enhances sleep quality and recovery. Before sleep a person activates wake / sleep memories which are embodied. This leads to an increased arousal and somatic tension in the case of wake memories or the opposite by embodied sleep memories. While sleeping, these wake / sleep memories are reactivated spontaneously. Reactivated wake memories thereby lead to an increased arousal while sleeping and decreased sleep quality showing more (NWAK) , longer time awake (WASO), and less deep sleep (SWS). Further, in the morning the person feels less recovered. For reactivated sleep memories the effects are the opposite: less NWAK and WASO, more deep sleep and in the morning better recovery. MemoSleep introduces the possibility to reactivate the wake / sleep memory while sleeping actively. This targeted memory reactivation boosts the effects of the spontaneous reactivation.

Taken together, the MemoSleep hypothesis provides an overall explanation for the relationship between cognition and sleep. By introducing the embodied sleep/wake memories and their spontaneous reactivation during the night, it is possible to apply the MemoSleep hypothesis to healthy, and not exclusively in insomniac, sleepers. Important in this hypothesis is the cognitive activation before sleep (pre-sleep activation). Therefore, the next section will introduce two aspects affecting pre-sleep cognition. First this thesis would like to introduce social media as an arousing factor (see chapter 1.3), which increases cognitive activity before sleep. It has been shown that social media use affects sleep negatively due to its cognitively arousing characteristics (Johansson et al., 2016; Mauri et al., 2011; Van den Bulck, 2003). As

a second aspect, this thesis would like to introduce a setting for on call-work (see chapter 1.4), which will reactivate the embodied wake memories during the night. This setting recreates on call-duty in a laboratory setting and participant hear sounds during the night and have to react as fast as possible. On call-work (and its arbitrary created laboratory setting) thereby figures as an example for ongoing cognitive processes during sleep. Both aspects affect pre-sleep cognition (e.g. wake-memories) and thereby are assumed to affect sleep negatively.

1.3 Social Media

The previous sections have demonstrated the importance of cognition on sleep and have highlighted the importance of sleep for psychological and physiological health as well as for different bodily functions or memory consolidation. Meanwhile, three models about the mechanism underlying this relationship have been developed, discussed and provided with evidence. The following paragraphs will give a short overview of social media affecting cognitive processes. Some might wonder, why social media? Due to its presence, social media affects different aspects of our life (Carter et al., 2016; Vogels, 2019; Woods & Scott, 2016). Indeed, because of its omnipresence there is always the possibility to be provided with new (positive or negative) information (Abel et al., 2016; Anderson & Jiang, 2018; Przybylski et al., 2013), which is cognitively stimulating and arousing (Mauri et al., 2011). This is highly relevant with respect to the relationship between cognitive functions and sleep: cognitively stimulating and arousing processes that are implicated in social media and which remain active day and night should therefore affect sleep as well, which is in line with the described models. Therefore, the next sections will give a background on social media and its interrelationship with cognition and sleep.

To start I would like to answer the question related to the definition of social media. Social media are “forms of media that allow people to communicate and share information using the internet or mobile phones” (Cambridge Dictionary, 2021). It is a form of communication, which allows us to stay connected with people around the world as it is based on the internet. What exactly does that mean? Social media are web-based services witch allow a person to create an open or semi-open profile as well as a list with contacts. It is possible to share content with others and stay connected with friends and acquaintances (Boyd & Ellison, 2007). Most activity is based on posting self-created content such as photos and videos or to read about and follow others concerning their opinions, questions and answers, personal

information, or knowledge (Pagani et al., 2010). Indeed, social media affects various aspects of our daily life and wellbeing (Carter et al., 2016; Woods & Scott, 2016). Especially for young people, social media is very important and 81% of teens report that it provides a connection to their peers (Anderson & Jiang, 2018). Although social media has a positive part to play in many aspects of our life, it has its negativity as well. One of the most important aspects is the so-called fear of missing out – or short FoMO (Vorderer et al., 2016). FoMO explains the phenomenon by which a person is afraid of missing out on something going on in their own social bubble which creates a need to reach out for its (social) news. It explains that a person needs to stay in touch with the social environment all the time to know what their own social environment is doing (Abel et al., 2016; Przybylski et al., 2013). Although FoMO is not a new phenomenon, with the expansion of contacts to the virtual world it grew in importance (Abel et al., 2016; Wortham, 2011). Thanks to new technologies, social media has become more portable and is available wherever we are, including the bedroom (Cain & Gradisar, 2010; Exelmans & Van den Bulck, 2016). In their study, Woods and Scott (2016) showed that using social media regularly during day-time and at night is associated with poor sleep quality. They report that a bigger involvement in social media during the day and at night led to poorer sleep quality. Just having a smartphone in the bedroom already leads to a worse sleep quality with prolonged SOL, WASO, and reduced TST (Johansson et al., 2016). One important factor of having a smartphone in the bedroom is that it is available while lying in bed before falling asleep. This creates a state of increased arousal or hyperarousal leading to impairment in sleep quality (Della Monica et al., 2018; Mauri et al., 2011; Van den Bulck, 2003; Woods & Scott, 2016), which is also what happens in the case of patients by the hyperarousal model of insomnia (Riemann et al., 2010). In general, we can see that sleep is affected by social media. Participants with high social media usage show reduced sleep quality (Levenson et al., 2016; Li et al., 2015), shorter TST (Chahal et al., 2013; Espinoza & Juvonen, 2011) and prolonged SOL (Arora et al., 2014).

In relation to sleep, the aspect of FoMO is important to consider. FoMO is a negative cognitive activity leading to distress and (physiological) arousal. This is in line with the cognitive model of insomnia (Harvey, 2002). It seems reasonable to assume that a person suffering from FoMO changes their habits during the day and night. For example, Adams and colleagues (2017) showed that FoMO leads to prolonged SOL, interruptions during the night, and reduced TST. FoMO is also one of the reasons why social media enhances the numbers of wake reactions during the night (Arora et al., 2014). Worrying about missing out on something in the social environment might cognitively arouse the person and thereby affects sleep

negatively. The second manuscript that this thesis will discuss contributes more evidence towards this field of research.

In summary, social media consumption affects subjective sleep in different ways. Although the fact of having a smartphone in the bedroom is not only associated with social media, it should be regarded as the main contributor since it is the connecting factor which can trigger FoMO. In addition, by creating an arousal, social media affects sleep directly leading to prolonged SOL, unstable sleep (more awakenings), or reduced TST. Most studies have looked at subjective sleep ratings and have concluded that social media consumption before and during the night affects sleep negatively. More studies including objective sleep measurements are required to provide information about the relationship between social media, arousal and sleep. So far, we have looked at the pre-sleep cognitive states. The next section will focus on the ongoing cognitive processes during the night. This is illustrated by looking at the pre-sleep instruction of being woken up during the night and how on call-duty affects sleep.

1.4 On Call-Work

As explained before, the MemoSleep hypothesis posits that pre-sleep cognitive activation affects sleep quality due to an embodiment of sleep/wake memories (Rasch, 2016). What if these wake memories were reactivated during the night? Is this targeted reactivation affecting sleep quality and the state of arousal as expected? An on call-setting is an example for a targeted reactivation with the embodied wake memory “alarm”, “work”, or “get up”. Are these wake memories reactivated during the night? Do they help to sustain the arousal and do they decrease sleep quality (e.g. lighter sleep, more awakenings, and more waketime)?

Indeed, the main characteristic of on call-duty is that a person might be woken up during the night by an alarm or a specific sound (e.g. alarm sound or a baby’s cry). Similar to the FoMO in the social media environment, in an on call-setting a person fears to miss a sound (FoMS), for example not waking up when the alarm rings. The FoMS is as well explained with the cognitive model of insomnia (Harvey, 2002, see chapter 1.3) or with the hyperarousal model of insomnia (Riemann et al., 2010). The latter explains how acute insomnia symptoms relates to worrying and stress (e.g. FoMS) with a person’s incapacity to sleep through the night due to the interrupted night-sleep (Hall et al., 2017). On call-duty means to be alert during the night while sleeping and being conscious of the environment, which thereby might create a

hyperarousal. This phenomenon is known for example in parents (Cottrell & Khan, 2005; McCann et al., 2015), health personnel (Gaba & Howard, 2002; Smithers, 1995), or firefighters (Finnerty, 1977; Paterson et al., 2016). In this thesis, the focus is on on call-duty in a working environment.

If somebody mentions that she or he is allowed to sleep during worktime, the first reaction is a positive one. Is it not a dream to get a salary while sleeping? In reality, this dream comes with severe side effects such as physical and mental health problems (Appleton et al., 1998; Balch et al., 2010; Chambers & Belcher, 1994; Chambers & Campbell, 1996; Rout et al., 1996). Further, on call lowers mood and well-being, as well as increasing tension, frustration and disturbances in family-life (Imbernon et al., 1993; Rankin et al., 1987). Noteworthy, the negative effects of on call-work on well-being appear regardless if an employee is called in or not during their shift (Bamberg et al., 2012).

As on call-work takes place during the night and while sleeping, the effects on sleep must be even more relevant. Indeed, more than half of employees working on call occasionally claim difficulties falling asleep, and 6% cannot fall asleep on a regular basis (Smithers, 1995). Only one third of the employees in this study reported that they always slept through the night when they were on call compared to almost three fourths when not being on call. The same study also reported that after the night on call, employees felt more tired, lacked concentration and were not able to perform their work routine. It seems that knowing to be on call for this night, affects sleep. This is in line with the MemoSleep hypothesis. Considering the task “wake up when called” as a wake memory which creates a pre-sleep arousal. This wake memory is reactivated during the night spontaneously and thereby maintains the arousal and affects sleep quality as well as recovery. This is confirmed by the results of Smithers (1995) as employees were not fit enough for social activities due to nightshifts on call. This tiredness the day after on call-duty was reported also by ship engineers (Torsvall & Åkerstedt, 1988). In addition to tiredness, a shortened TST and reduced sleep quality in an on call-night was reported. Objectively sleep quantity was shorter in on call-nights, participants showed less REM sleep and SWS. Furthermore, the EEG power density during on call-nights was reduced – especially in the first cycle. Interestingly, these effects were shown before any alarm went off. This means that only the thought of being woken up provoked reduced sleep quality. It seems that there are ongoing cognitive processes during on call-nights. The third manuscript in the thesis will contribute more evidence in this regard. There is a lack of studies in a controlled laboratory setting as most of the studies are field studies looking at actual on call work with no controlled reactivation of wake memories during the night. Field studies showed that the anticipation (or

intention) of an alarm sound waking someone up prevented workers to develop normal sleep patterns during the night regardless of being called or not (Torsvall & Åkerstedt, 1988).

To sum it up, on call-work affects our life both in the day and in the night negatively. During daytime, employees working on call claim negative affections in their social life and more often report mental and physiological disorders. At night they suffer from problems such as difficulties in falling asleep and report negative sleep quality, which is measurable on an objective level with less SWS and REM sleep during a night on call. Worse sleep because of on call-work can be explained using the MemoSleep hypothesis as the pre-sleep activated instruction figures as an embodied wake memory of "waking up", which is reactivated during the night. Having outlined the relationship between sleep and cognition as well as cognitively enhancing factors in more detail, the following last section will prepare for the three manuscripts by shortly summarizing the respective hypotheses.

2 Aims and Hypotheses

So far, we have seen that there are effects of cognition on sleep. Especially pre-sleep rumination affects sleep quality negatively. The aim of this thesis is to find out how different pre-sleep manipulations and instructions affect sleep and if there is an effect of ongoing processes during the night. This leads to the overall hypothesis that pre-sleep cognitive states affect sleep. We assume that the instruction given right before sleep will stay active during the night. Therefore, a created pre-sleep arousal will continue during the night and will lead to participants waking up more often and more easily during the night.

In the first manuscript we created a baseline to see what positive and negative cognitions are able to do on someone's sleep. The fact that the intention to sleep worse might decrease sleep quality seems logical but has not been confirmed experimentally yet. Due to the two models for insomnia, which assumed that negative cognition (e.g. rumination, worry, or psychosocial stress) lead to sleep related problems and insomnia, we therefore hypothesized that participants would be able to sleep worse, but not better than normal.

In the second manuscript, we investigated the effects of social media consumption on sleep. Social media has proven to worsen sleep quality as well as to increase arousal. In the study, we therefore compared the introduced pre-sleep social media condition with a neutral baseline and a progressive muscle relaxation (PMR) instruction. We expected that pre-sleep social media consumption would induce a physiological arousal and thereby reduce subjective and objective sleep quality. Further we expected an arousing effect of social media on sleep, which comes with prolonging SOL and thereby affects sleep quality negatively.

The aim of the third manuscript was to investigate the relationship between the instruction of on call-work and sleep. It has been proved that on call duty reduces sleep quality. Most studies were conducted in a working environment and based on subjective ratings. Here we aim to confirm the relationship experimentally in a controlled setting. We therefore simulated an on call-setting for participants in the sleep laboratory. Our expectations were that the instruction to be on call would affect sleep quality negatively. Furthermore, we hypothesized that the presentation of sounds might affect participants unconsciously and that alarm sounds disturbed sleep on a neurobiological level even if participants might not wake up.

Over all, the aim of this thesis is to look how cognitive factors affect our sleep. Therefore, different cognitive factors are considered in relation to the three different models. At the end, this thesis would like to discuss which of the introduced models above can explain the best, how and what cognitive factors affect sleep the most.

3 Manuscripts

3.1 Healthy sleepers can worsen their sleep by wanting to do so: the effects of intention on objective and subjective sleep parameters[3]

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[3] A similar version of this manuscript has been published in "Nature and Science of Sleep"

Abstract

Purpose: Sleep is regulated by homeostatic and circadian factors. In addition, psychological factors have a strong modulatory impact on our sleep, but the exact underlying mechanisms are still largely unknown. Here we examined the role of intentions on subjective and objective sleep parameters. Young healthy sleepers were instructed to voluntarily either worsen or improve their sleep. We predicted that participants would be capable of worsening, but not improving, their sleep compared to a regular sleep condition. In addition, we predicted that the instruction to alter sleep would lead to a higher discrepancy between subjective and objective sleep variables.

Participants and methods: Twenty-two healthy students participated in one adaptation and three experimental nights. Polysomnography and subjective sleep parameters were measured during all four nights. Participants were instructed to sleep regularly ("neutral"), better ("good") or worse ("bad") than normal, in a counterbalanced order.

Results: The instruction to sleep "bad" increased objective sleep onset latency and the number of awaking's during the night. The effects were stronger on subjective sleep variables, resulting in a higher sleep misperception in the "bad" condition as compared to the other two conditions. The instruction to sleep "good" did not improve sleep nor did it affect sleep misperception.

Conclusion: We conclude that intention is sufficient to impair (but not improve) subjective and objective sleep quality and to increase sleep misperception in healthy young sleepers. Our results have important implications for the understanding of the impact of psychological factors on our sleep.

Keywords

Cognition, Sleep, Sleep Quality, Intention, Sleep Misperception

Plain language summary

Sleep is characterized by a reduction in consciousness. Therefore, sleep is seen often as a biological state that cannot be influenced by our intention. Here we study how that healthy sleepers are capable of objectively worsening (but not improving) their sleep simply by wanting to do so. In addition, participants strongly overestimate their induced sleep disturbances, as typically seen in insomnia patients. Our results show that our pre-sleep intentions and goals to manipulate sleep can affect ongoing sleep processes and influence evaluations of our sleep quality. Our results suggest that psychological process continue to be active during the state of sleep and can influence biological sleep regulation and maintenance in spite of the reductions in consciousness.

Introduction

Sleep is a natural and reversible state of reduced consciousness which is regulated by complex neurobiological mechanisms and homeostatic processes (Borbély & Achermann, 1999). In the case of disturbed sleep, a wide range of different sleep-inducing drugs are available which are known to help fall asleep as well as to maintain it (Greenblatt & Shader, 1978; Hindmarch et al., 2005; Holbrook et al., 2001; Roehrs & Roth, 2001). Based on the increased understanding of sleep-regulating neurobiology, new drugs have been developed and are available to allow a better preservation of the natural sleep architecture (Brunner et al., 1991; Lader, 1992), in particular with respect to the restorative stage of slow wave sleep (SWS) (Aeschbach et al., 1994; Dijk, 2009).

In addition to the neurobiological aspects of sleep, psychological factors play a critical role in sleep onset and maintenance. According to the cognitive models of insomnia (Harvey, 2002; Harvey et al., 2005), insomnia patients tend to strongly worry about their sleep and about the negative consequences of not getting enough sleep. This worry might increase attention for cues that pose a threat to their sleep and increase non-functional safety behaviors. Together with false beliefs about sleep, excessive sleep anxiety is presumably a causal factor for sleep disturbances. In addition, patients with insomnia can have a strong misperception of their actual sleep quality resulting in a large subjective-objective discrepancy of sleep parameters such as sleep onset latency (SOL), number of awakenings from sleep (NWAK) and time spent awake at night (WASO) (Rezaie et al., 2018). According to the hyperarousal model of insomnia (Riemann et al., 2010), cognitive-psychological factors act in concert with biological arousal processes to inhibit sleep. Consequently, relaxation of somatic tension and reduction of worries, together with cognitive therapy on sleep beliefs, are key aspects of cognitive-behavioral therapy for insomnia (CBT-I) (Riemann et al., 2017). CBT-I uses behavioral strategies such as psychoeducation, stimulus control, sleep restriction, and paradoxical intervention (Riemann et al., 2017; Riemann & Perlis, 2009).

While it seems clear from insomnia research that our mental activity and thoughts can disturb our sleep, the basic mechanisms are not fully understood. Importantly, it is not clear to what extent we can manipulate healthy sleep intentionally. Intentions are a part of our executive functions, mainly involving neuronal networks in the prefrontal cortex (Haynes et al., 2007; Momennejad & Haynes, 2012), which can have a modulatory top-down influence on the basic neurophysiological functioning of our brain. Sleep, when considered as a state of strongly reduced consciousness, typically appears to be beyond our cognitive control. Therefore, it is important to examine whether, and to what extend, healthy participants can voluntarily influence their sleep. Furthermore, it is highly relevant to explore the possible mechanisms underlying the influence of voluntary intentions on sleep. For example, participants might be able to alter their biological or somatic arousal state voluntarily before sleep, which in turn might affect subsequent sleep. Alternatively, the possible effects of intention on sleep might depend on mental activity per se, independently from somatic pre-sleep arousal. In addition, our

intention might influence the discrepancy between subjective and objective sleep parameters often seen in insomnia patients.

To test the effect of intentions on sleep, a number of young healthy participants spent three nights in the sleep laboratory and were instructed to sleep "as bad as possible", "as good as possible" or "as usual", in a counterbalanced order. They were instructed to manipulate sleep latency, the number and duration of awakenings, and sleep depth, according to the respective condition. They received no instructions regarding how they should achieve these manipulations. Sleep was recorded using a polysomnography setup. As we expect participants to be able to manipulate sleep intentionally, we hypothesized that participants would be able to sleep worse when asked to do so, as indicated by subjective and objective sleep parameters. However, because nighttime sleep in young healthy participants is already close to optimal, we do not expect intentions to make sleep better in our study, thus the condition to sleep "as good as possible" will be invariant across sleep parameters compared to the control condition. We also expect that effects of intentions on sleep will be larger for subjective as compared to objective sleep parameters, resulting in a higher subjective-objective sleep discrepancy. Finally, we will examine the association between somatic arousal before and after sleep (as measured by electrocardiography (ECG)) to test whether sleep changes induced by intention could be solely explained by changes in pre-sleep arousal or not.

Materials and methods

Participants

Twenty-four young healthy students (15 women) with a mean age of 22.3 ± 3.0 [SD] participated in the study. Two participants were excluded due the detachment of electrodes during the night. Participants were recruited through an advertisement and a newsletter for students. They were not on any night shifts and were asked to keep a regular sleep rhythm. Participants were no extreme chronotypes for morningness or eveningness. Based on the German translations of the Morningness-Eveningness Questionnaire (D-MEQ; Griefahn et al., 2001) four participants were moderate morningness and four participants were moderate eveningness types. Participants reported that they did not suffer from any known sleep disorders, however they did report a high subjective sleep quality. (Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989) 3.95 ± 1.68 [SD]). They were instructed to abstain from alcoholic and caffeinated beverages on experimental days as well as the day before the test days. The local ethics committee of the University of Fribourg approved the study and all subjects gave written informed consent prior to participating. This is in accordance with the Declatation of Helsinki. Subjects received either university course credits or financial compensation of at least 180 CHF for participating in the experiment. In case of an early drop out payment was provided proportionately.

Experimental Design and Procedures

The within-subject design included one adaptation and three experimental nights in the sleep laboratory at the University of Fribourg with the different conditions: "bad", "good" and "neutral" sleep. The procedure of the three experimental conditions was identical with the exception of the instructions given to the participants prior to sleep (see below). The experimenter and the subject were blind towards the condition until the very moment before sleeping. The order of the conditions was randomly assigned and balanced between the subjects.

Participants arrived at the sleep lab and filled in some standardized questionnaires before the installation of the polysomnography (PSG including electroencephalogram (EEG), electromyogram (EMG), electrooculogram (EOG), electrocardiogram (ECG)). They then completed a memory task where they learned 40 semantically associated word pairs (paired associated learning (PAL) task (Rasch et al., 2006)) before going to bed, after which they were instructed to lay in bed and the instructions for the night were given to them ("Sleep as well / badly / neutrally as possible"). Eight hours after lights out, the experimenter woke the participants up. Directly after getting up, participants filled in standardized questionnaires (concerning subjective sleep rating, mood), and completed a psychomotor vigilance test (PVT), as well as the morning-recall of the PAL. The session ended with the removal of the PSG (see Figure 1).

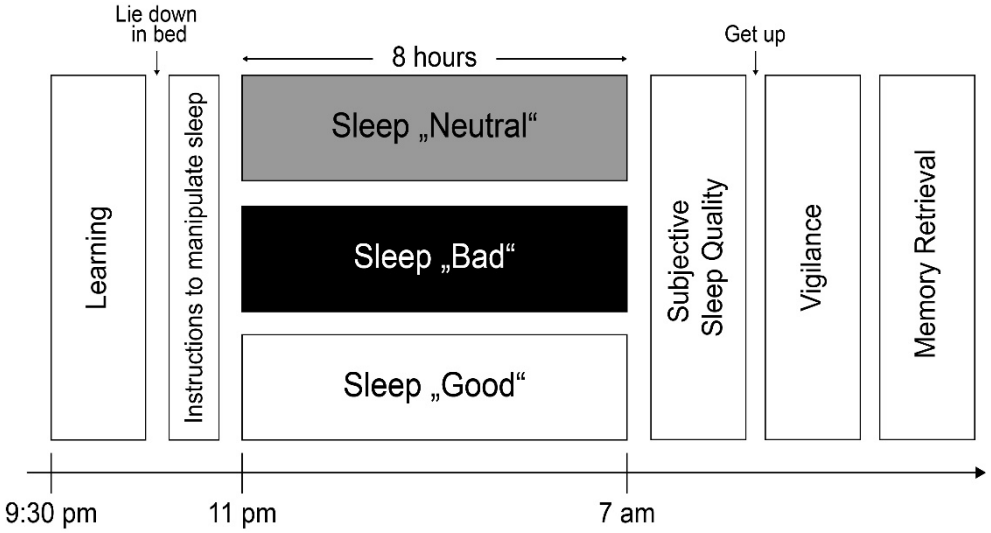

Figure 1. Procedure and experimental design. Participants spent one adaptation night and three experimental nights in the sleep lab for a total of four nights, each separated by one week. Directly before going to sleep and when lying in bed, participants received the instructions to sleep worse than normal (sleep "bad"), better then normal (sleep "good") or to sleep as usual (sleep "neutral"). In the "bad" condition, they were asked to willingly decrease sleep quality by falling asleep later, waking up more often, staying awake longer during the night and sleeping less deep. In the "good" condition, participants were instructed to decrease sleep quality by falling asleep quicker, staying asleep and sleeping as deeply as possible. No instructions were given regarding how participants could achieve these goals. Participants had to stay in bed in the dark and were not allowed to get up during the night. Sleep was recorded using a polysomnography setup. Before the instructions, participants filled out questionnaires and performed a word-pair learning task. After sleep participants filled out questionnaires, performed a psycho-vigilance task (PVT) and retrieved word-pairs learned before sleep.

Material

Instructions

Instructions were given orally when participants were lying in bed and were ready to sleep. The instructions were as follows:

- "Bad" sleep: participants were instructed to sleep as bad as possible. They should try to fall asleep as late as possible (ideally, they should not fall asleep at all), wake up during the night (and if so, stay awake), sleep less time, and sleep only lightly (enhance the light sleep).
- "Good" sleep: participants were instructed to sleep as well as possible. They were instructed to fall asleep quickly (ideally directly after the experimenter turned off the light), not wake up during the night (if so, they should try to fall back asleep fast), sleep as long as possible (ideally the full 8 hours) and sleep as deeply as possible.
- "Neutral" sleep: in the neutral night, participants were given the following instructions: "This is your neutral night. Sleep as regularly as possible."

For all three conditions, participants were instructed to stay in bed in the darkness. They were not allowed to get up, read, or access their mobile phones. No additional instructions were given on how to reach "good" vs. "bad" sleep. The participants were told that if they were able to sleep better/worse as indicated by the PSG measurements, they would be rewarded with an additional 25 CHF for each experimental session. However, at the end of experiment, participants received the additional 25 CHF based on their subjective achievements.

Questionnaires

During the first night, participants filled out a general questionnaire for health and personal information (for example related to Body Mass Index, handedness, sex, experience in a sleep laboratory, or sleep habits) where the Edinburgh Inventory for Handedness (Oldfield, 1971), the Pittsburgh Sleep Questionnaire Inventory(Buysse et al., 1989) and a questionnaire for chronotype (Griefahn et al., 2001) were used. Additionally, in each session, participants filled out a general questionnaire with information regarding their sleep the previous night as well as general health, such as consumption of alcohol, caffeine, drugs, or nicotine. Lastly, they filled out a questionnaire relative to their mood (Steyer et al., 1997). In the morning, while still lying in bed, participants were asked to rate their subjective sleep quality (Görtelmeyer, 2011), and again, their mood (Steyer et al., 1997). After the last night they performed the Harvard Group Scale of Hypnotic Susceptibility (high suggestibility ≥ 7) (Bongartz, 1985).

Memory measurement

Episodic memory was tested with a paired-associated learning (PAL) task (Rasch et al., 2006). Participants learned a list of 40 semantically related word pairs. Each trial started with the first word of a pair which was presented for 3000ms, followed by a 500ms blank interval which separated the single trials. The words were presented in black font on a white screen via E-Prime (Psychology Software Tools, Pittsburgh). Each pair was presented only once while the order was kept constant. Immediately after learning, participants were confronted with a cued recall test. Here, they had to come up with the corresponding word when the first word was displayed. During the recall test the word pairs were presented for an infinite amount of time, or until the participant pressed enter, then the correct answer was presented straight away for 1000ms followed by a 500ms blank interval which, again, separated the single trials. A second recall test followed. In this second recall test the participants did not receive any correction or confirmation of the correct word pair; right after filling in the answer, a 500ms blank interval followed. During recall the order of the word pairs differed from the learning phase but was kept constant across subjects. A third recall phase took place in the morning. Performance was measured as the percentage of words recalled in the morning relative to the amount of words remembered immediately after learning. Response time was not restricted.

Psychomotor Vigilance Test (PVT)

In the morning participants performed a psychomotor vigilance test (PVT) which is designed to measure the effect of sleepiness on vigilance (Dinges & Powell, 1985). Subjects were asked to press the space key with their non-dominant hand as soon as they recognized the millisecond counter on the screen, which appeared at random intervals. After the keypress the reaction time in milliseconds was shown for 1s.

Polysomnographic recordings

To measure sleep, EEG, EMG, EOG and ECG were used. EEG was recorded using 10 single gold-cap-electrodes following the 10-20-EEG-system with a sampling rate of 500 Hz. Impedances were kept below 5 kΩ. Following the AASM guidelines, electrodes were referenced against Cz during recording and then re-referenced to the mastoids when doing offline analyses. Data was preprocessed with Brain Vision Analyzer 2.0 (Brain Products GmbH, Gilching), filtering the data by 0.3 – 35 Hz following the guidelines suggested by the American Association of Sleep (Iber et al., 2007). Two independent sleep scorers visually scored sleep in 30s periods based on derivations F4, C4, O4, EOG (left and right), and EMG. Stages N1–N3, REM sleep, and WASO were scored following the AASM-guidelines (Iber et al., 2007). Additionally data were analyzed with an automated sleep scoring algorithm (The SIESTA Group Schlafanalyse GmbH, Vienna). These analyses provide more insight on microstructural parameters such as arousals and stage shifts.

Analysis of the EEG data

For sleep analysis we used the SleepTrip toolbox (Weber, 2019) for Matlab (Mathworks, Natick). Data were preprocessed with Brain Vision Analyzer 2.0 (Brain Products GmbH, Gilching) before spectral analysis. We calculated the average power of oscillatory activity in different frequency bands: slow wave activity (SWA) (0.5 – 4.5 Hz), theta activity (4.5 – 8 Hz), alpha activity (8 – 11 Hz), slow spindles (11-13 Hz), fast spindles (13-15 Hz) and beta activity (15-30 Hz). In addition, we calculated the ratio between SWA and beta activity as this measure is associated with objective sleep quality (Cordi et al., 2019; Krystal, 2008; Maes et al., 2014). Data from lights off in the evening to lights on in the morning were analyzed, segmented for NREM sleep (N2 and N3 sleep) and REM sleep.

Analysis of Sleep Cycles

A cycle analysis was performed using Matlab (Mathworks, Natick). The first cycle started with sleep onset. A cycle ends when a REM episode is not followed by another REM episode for 15 minutes. For each cycle we calculated the amount of N1, N2, N3, REM, and WASO as well as the duration of each cycle.

Analysis of ECG

ECG (electrocardiogram) analysis was carried out using Kubios HRV Premium 3.2.0 (Kubios Oy, Kuopio). Therefore, the ECG signal for pre-sleep (lights off to the first stage of N1) and the whole night (lights off to lights on) was exported in EDF+-format using BrainVisionAnalyzer Version 2.0 (Brain Products GmbH, Gilching). Kubios HRV Premium offers an automatic artifact correction based on RR series (the interval between two R-signals) to eliminate ectopic beats and artifacts in unfiltered data (Tarvainen et al., 2019). Afterwards data were analyzed in a time and frequency domain and subsequently used for the calculation of the mean heart rate (mean HR measured in beats per minutes) which served as an index for physiological arousal (Kogler et al., 2015). Furthermore, Kubios provides an index to analyze the tone of the parasympathetic nervous system (PNS) and sympathetic nervous system (SNS). We used the PNS- and SNS-indexes to evaluate an appropriate measure for physiological stress. The PNS-index is a PNS tone index based on mean RR whereas the SNS-index is a SNS tone index to evaluate stress which is based on Mean HR (Tarvainen et al., 2019).

Statistical Analysis

According to our experimental manipulation before sleep (see section “Instruction”), our main outcome variables were subjective and objective sleep quality, SOL, WASO, NWAK and sleep depth. For each of these main outcome variables, a separate repeated measures analysis of variance (ANOVA) with the within-subject factor condition (“bad”, “good”, and “neutral”) was calculated. In a separate analysis, we used a 2x3 Analysis of Variance with the within-subject factors “type of parameter” (subjective vs. objective) and condition (“bad”, “good”, and “neutral”) for the five main outcome

variables. In addition, we report Pearson's correlation coefficient (r) for the strength of association between these objective and subjective sleep parameters.

In exploratory analyses we analyzed further sleep parameters, vigilance, sleep-associated memory consolidation, and heart rate variability (HRV) using an ANOVA for repeated measures with the within-subject factor condition ("bad", "good", "neutral"). For oscillatory power during NREM and REM sleep, we used a 3x2x3 ANOVA for repeated measurements using the factors "condition" (bad, good, neutral), "hemisphere" (left, right) and "topography" (frontal, central, parietal). And we report Pearson's correlation coefficient (r) for the strength of association between sleep parameters and vigilance.

Post-hoc paired t-tests with a Bonferroni correction were conducted in case of significant main effects or interaction effects. We set the level of significance to $p \leq 0.05$ and reported effect sizes ($\eta 2$) only for significant data. Data were analyzed using IBM SPSS Statistics 25 (IBM Corp., Armonk) and R Studio (RStudio Team, Boston). Results are presented as means ± standard errors of the mean (SEM).

Data Availability

Datasets analyzed during the current study are available online on https://osf.io/asz94/.

Results

Subjective Sleep Parameters

According to the instructions, our primary outcome variables were subjective sleep quality, subjective SOL, subjective WASO, subjective NWAK, and subjective sleep depth. All subjective variables were measured with the SFA-R questionnaire (Görtelmeyer, 2011).

In accordance with our hypothesis, participants were able to subjectively worsen, but not to improve their sleep. In the "bad" sleep condition, participants rated their sleep quality in the morning as worse (16.23 ± 0.98, scale from 7 to 35, higher values indicating better sleep quality) compared to the "good" (25.05 ± 1.20), and "neutral" conditions (24.55 ± 1.11) (repeated measures ANOVA with the factor condition ("bad", "good", "neutral"), $F(2, 20) = 24.55$, $p < 0.001$, $\square 2 = 0.71$, see Figure 2a, Table 1). Post-hoc pairwise comparisons confirmed that with the instruction to sleep "bad" participants rated their sleep quality significantly worse as compared to the "neutral" and "good" conditions (both $p < 0.001$). "Good" and "neutral" conditions did not differ significantly ($p > 0.50$).

We observed a similar pattern for SOL, WASO, NWAK and sleep depth (see Figure 2b-e, for post-hoc pairwise comparisons).

Objective Sleep Parameters

As was reported for subjective sleep parameters, on the objective level participants were able to worsen but not to improve their sleep by simply wanting to do so. Similar to the subjective ratings, our primary outcome variables for objective sleep parameters were sleep efficiency (taken as objective measure of sleep quality), SOL, WASO, NWAK and amount of SWS in minutes (taken as objective measure of sleep depth).

As predicted and in line with the subjective measures of sleep quality, participants who were instructed to sleep "bad" showed a reduced sleep efficiency with 93.67 ± 0.97% compared to 97.45 ± 0.57% in the "neutral" condition and 96.91 ± 0.66% in the "good" condition ($F(2,42) = 12.69$, $p < 0.001$, $\eta^2 = 0.377$). Post-hoc pairwise comparisons confirmed that there was a significant reduction of sleep efficiency in the "bad" condition compared to both the "neutral" ($p < 0.001$) and "good" ($p = 0.004$) conditions. The latter two conditions did not differ ($p > 0.20$, see Figure 2f and Table 1).

Table 1. Sleep parameters for the three conditions (subjective and objective)

	Bad	Good	Neutral	
	M ± SEM	M ± SEM	M ± SEM	F-Test
Subjective Sleep Parameters				
Sleep Quality	20.28 ± 0.592	24.833 ± 0.77	24.547 ± 0.61	**24.253*****
SOL [min]	52.27 ± 3.51	17.64 ± 3.44	14.02 ± 2.97	**53.988*****
WASO [min]	39.55 ± 8.04	9.09 ± 2.04	10.55 ± 3.66	**11.273*****
NWAK	3.41 ± 0.18	2.16 ± 0.23	2.05 ± 0.21	**20.573*****
Sleep Depth	2.14 ± 0.15	3.59 ± 0.17	3.73 ± 0.19	**47.513*****
Objective Sleep Parameters				
Sleep Efficiency [%]	93.67 ± 0.97	96.91 ± 0.66	97.45 ± 0.57	**12.09*****
SOL [min]	29.75 ± 4.46	14.77 ± 3.14	12.21 ± 2.75	**12.864*****
WASO [min]	13.84 ± 2.28	15.25 ± 4.12	9.89 ± 2.341	1.126
NWAK	8.05 ± 0.87	5.68 ± 0.95	4.73 ± 0.65	**6.98****
NWAK + Movements	16.46 ± 1.60	10.68 ± 1.20	11.41 ± 1.11	**10.34*****
Duration per Awakening [min]	1.63 ± 0.38	1.59 ± 0.47	1.23 ± 0.30	0.40
N1 [min]	34.89 ± 3.96	39.52 ± 3.15	33.61 ± 2.53	1.68
N2 [min]	211.73 ± 6.20	231.61 ± 7.32	234.80 ± 6.49	**4.57***
N3 [min]	82.18 ± 6.05	89.09 ± 6.89	84.80 ± 5.61	1.05
REM [min]	97.89 ± 4.28	88.30 ± 4.70	100.02 ± 4.25	3.05
Move [min]	3.16 ± 0.81	2.21 ± 0.61	2.55 ± 0.61	1.60
N1 [%]	7.83 ± 0.87	8.47 ± 0.67	7.20 ± 0.53	2.02
N2 [%]	47.71 ± 1.14	49.69 ± 1.53	50.37 ± 1.35	1.46
N3 [%]	18.46 ± 1.29	19.11 ± 1.50	18.24 ± 1.25	0.35
REM [%]	22.11 ± 0.92	18.92 ± 0.97	21.43 ± 0.88	**4.77***

	Bad	Good	Neutral	
	M ± SEM	M ± SEM	M ± SEM	F-Test
Move [%]	0.71 ± 0.18	0.46 ± 0.13	0.54 ± 0.13	2.28
WASO [%]	3.16 ± 0.54	3.35 ± 0.92	2.13 ± 0.51	2.55
TST [min]	443.68 ± 97.86	465.98 ± 101.70	466.00 ± 102.66	**8.98*** **
SWS Latency [min]	16.77 ± 5.97	17.00 ± 4.80	17.00 ± 5.33	0.01
REM Latency [min]	78.34 ± 1.71	97.64 ± 33.37	82.93 ± 2.03	2.14
Memory				
Encoding (evening)	32.77 ± 1.06	32.86 ± 1.05	31.18 ± 0.88	2.362
Recall (morning)	31.86 ± 1.08	31.64 ± 1.21	30.05 ± 0.97	2.698
Consolidation [%]	97.22 ± 0.97	95.98 ± 1.34	96.40 ± 1.53	0.508
Vigilance (Morning)				
Recaction Time (RT)	368.12 ± 12.14	332.42 ± 7.52	342.48 ± 7.79	**7.994*** **
Reactions	77 ± 0.55	76.27 ± 0.66	77.36 ± 0.58	0.834
Errors	1.5 ± 8.92	12 ± 0.38	1.27 ± 0.40	1.409

Notes: Subjective parameters are based on subjective ratings in the SF-A-R(Görtelmeyer, 2011). Objective values are based on polysomnographic recordings. Non-rapid eye movement (NREM)-sleep, stage 1, 2, and 3 sleep (N1, N2, N3), rapid eye movement sleep (REM), waketime after sleep onset (WASO), total sleep time (TST), sleep onset latency (SOL), slow wave sleep latency (SWS latency), REM sleep latency (REM latency) are all measured in minutes [min] and the percentages indicate parietal percentage of TST [%]. For memory, numbers indicate absolute or relative values of correctly recalled words that were presented in the evening (learning phase with first recall) and in the morning (retrieval phase with second recall). Consolidation refers to the difference in performance between learning and retrieval phases. For vigilance, the reaction time (RT), the number of reactions, and amount of errors during the 10 minutes of the psychomotor vigilance task (PVT) were measured.
Values are means ± standard error of mean (SEM). * indicates $p \leq 0.05$ and ** indicates $p \leq 0.01$
*** indicates $p \leq 0.001$

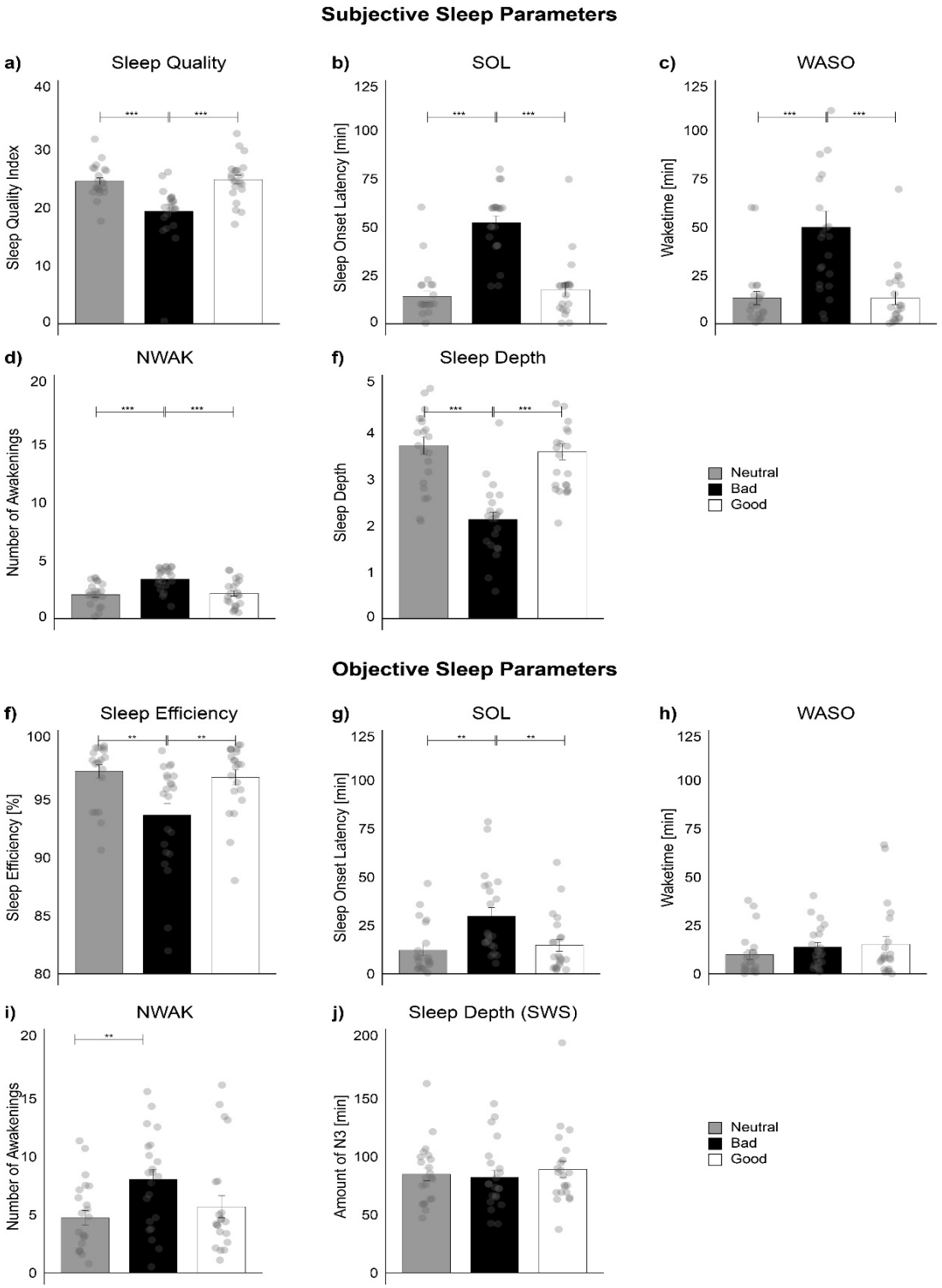

Figure 2. Effects of instructions on subjective and objective sleep parameters. On the subjective level, after being instructed to sleep "bad" (black bars), participants reported (a) to sleep worse (sleep quality), (b) took more time to fall asleep (sleep onset latency (SOL)), (c) spent more time awake after sleep onset (WASO), (d) woke up more often (NWAK) and (e) slept less deep (sleep depth) as compared to the night with the instruction to sleep good (white bars) and as compared to the night without specific instructions ("neutral", grey bars). The instruction to sleep "good" (white bars) did not alter subjective evaluations of sleep. For objective sleep parameters, the instruction to sleep "bad" (f) reduced sleep efficiency as an objective measurement for sleep quality, (g) extended SOL and (i) increased the NWAK during the night as compared to the night without instructions ("neutral"). No effect was observed for WASO (h) and the time spent in SWS (j). Again, no effect occurred after the instruction to sleep "good". Means ± standard errors of the mean are indicated. Significant pair-wise comparisons from post-hoc tests are indicated by **: $p \leq 0.01$. ***: $p \leq 0.001$.

We observed the same pattern for objective SOL, as people took roughly 15 minutes longer to fall asleep when they were asked to sleep "bad" (see Figure 2g and Table 1). In addition, they increased their NWAK: in the "bad" sleep condition participants awoke 8.05 ± 0.87 times, whereas they woke up only 4.73 ± 0.65 times in the "neutral" condition and 5.68 ± 0.95 times in the "good" condition ($F(2,42) = 6.98$, $p = 0.002$, $\eta 2 = 0.249$ (see Figure 2i). This difference was even more pronounced when movements were included in the number of awakenings ($F(2,42) = 10.34$, $p < 0.001$, $\eta 2 = 0.330$, see Table 1).

In contrast to our expectations, instructions were not able to influence the objective time spent awake (WASO, $F(2,42) = 1.03$, $p > 0.30$, see Figure 2h, Table 1) or the objective depth of sleep (sleep stage N3 $F(2,42) = 1.05$, $p = 0.358$, see Figure 2j, Table 1).

The exploratory analysis of total sleep time also showed a reduced sleep time in the "bad" condition ($F(2,42) = 8.98$, $p = 0.001$, $\eta 2 = 0.30$, see Table 1). Note that differences in SOL directly affect total sleep time in our study, as all participants were awakened after exactly eight hours of time in bed. After correcting for differences in SOL, total sleep time did not differ between conditions ($F(2,42) = 1.39$, $p > 0.20$). Explorative analysis on other sleep parameters did not reveal any significant differences for N1, movement time, SWS latency and REM latency sleep (all $p > 0.10$). In contrast, time spent in stage N2 sleep was significantly shorter in the "bad" condition (211.73 ± 6.20 minutes) as compared to the "neutral" (234.80 ± 6.49 minutes) and "good" (231.61 ± 7.32 minutes, $F(2,42) = 4.57$, $p = 0.016$, $\eta 2 = 0.18$) conditions. However, this effect was, again, fully explained by an increase in SOL of 20 min the "bad" condition ($p < 0.60$, after correction for SOL). Interestingly, REM sleep appeared to be shorter in the "good" condition (88.30 ± 4.70 minutes) as compared to "neutral" (100.02 ± 4.25 minutes) and "bad" conditions (97.89 ± 4.28 minutes). The effect only reached a statistical trend ($p = 0.058$), but was statistically significant when considering the percentage of REM sleep relative to total sleep length ($F(2,42) = 4.77$, $p = 0.014$, $\eta 2 = 0.19$). This effect cannot be explained by SOL as the effect was still present after correcting for differences in SOL between the conditions ($F(2.42) = 7.63$, $p = 0.001$, $\eta 2 = 0.27$; see Table 1 for an overview).

We also considered microstructural parameters such an arousal index and the amount of stage shifts between the different sleep stages. Results of the arousal index analysis are shown in Table 2. Additionally we analyzed the stage shifts between different sleep stages and showed a significant effect of the condition on the shifts from any sleep stage to wake ($F(2, 40) = 5.24$, $p = 0.010$, $\eta 2 = 0.21$) but not between the sleep stages. Post-hoc comparison confirmed significantly more stage shifts from any sleep stage to wake in the "bad" (13.76 ± 2.18) compared to the "neutral" (10.10 ± 2.84) condition. The other conditions did not differ (all $p > 0.27$).

Table 2. Arousals and stage shifts

	Neutral	Bad	Good	
	M ± SEM	M ± SEM	M ± SEM	F-Test
Arousal				
Arousals in TST	159.10 ± 28.37	159.19 ± 25.10	167.95 ± 28.80	1.81
Arousals in NREM	141.57 ± 24.79	139.48 ± 23.24	153.10 ± 29.69	2.86
Arousals in REM	17.52 ± 7.42	16.71 ± 5.46	14.86 ± 3.71	1.67
Arousal Index (TST)	20.86 ± 3.65	21.86 ± 3.51	22.17 ± 3.66	1.18
Arousal Index NREM	24.07 ± 3.63	25.21 ± 3.72	25.37 ± 3.97	1
Arousal Index REM	10.4 ± 3.69	10.51 ± 2.92	9.90 ± 2.41	0.26
Stage shifts from any sleep stage to				
Wake	10.10 ± 2.84	13.76 ± 2.18	10.90 ± 1.96	5.24**
N1	42.52 ± 7.64	43.86 ± 7.42	45.71 ± 9.17	1.18
N2	55.48 ± 10.91	54.86 ± 9.60	57.38 ± 13.09	0.45
N3	19.67 ± 6.11	18.10 ± 2.84	18.76 ± 5.67	0.44
REM	7.76 ± 1.31	7.86 ± 1.53	6.71 ± 0.87	1.33

Notes: Analysis with an automatic sleep scoring algorithm (The SIESTA Group Schlafanalyse GmbH, Vienna) providing the number of arousals, an arousal index and stage shifts. Number of arousals as well as the arousal index were analyzed for total sleep time (TST), non-rapid eye movement as well as rapid eye movement sleep (REM). Further stage shifts from any sleep stage to wake and to NREM sleep, stage 1, 2, and 3 sleep (N1, N2, N3) are shown. Values are means ± standard error of mean (SEM). ** indicates $p \leq 0.01$

We also analyzed whether the type of instruction before sleep influenced the cyclic structure of sleep. The amount of cycles was of 4.36 ± 0.12 over all participants and in each condition ("bad": 4.36 ± 0.19; "good": 4.36 ± 0.23; "neutral": 4.36 ± 0.19). It did not differ for the three conditions ($F(2,20) = 0$, $p = 1.00$). Also, the mean duration of the first four cycles was not affected by the different instructions before sleep (for further information see supplementary material Table S1).

Effect of pre-sleep instructions on oscillatory power during NREM and REM sleep.

In addition to sleep architecture, we also analyzed the effects of intentions on oscillatory power in different frequency bands (slow-wave activity (SWA), theta, alpha, slow spindle, fast spindle and beta band). We observed no significant main effects of the factor condition (bad, good, neutral) on oscillatory power during NREM sleep (all $p > 0.10$). Furthermore, no significant interaction was found for the factor "condition" with the factors "hemisphere" (left, right), "topography" (frontal, central, parietal), or both (all $p \geq 0.085$). Moreover, regarding the ratio between power in the SWA band and beta band, which is considered an indicator of objective sleep quality, no effects nor interactions with the factor condition were observed ($p \geq 0.085$). Also, during REM sleep we did not find any main effects or interactions with the factor condition ($p > 0.27$, for an overview see supplementary material Table S2).

Comparing objective and subjective sleep parameters

Participants generally overestimated the time it took them to fall asleep as well as the time they spent awake during the night. The degree of overestimation was largest in the "bad" condition: participants reported a SOL of 52.27 ± 3.51 min, whereas they actually fell asleep after 29.75 ± 4.46 min, resulting in an overestimation of ca. 23 min. In contrast, they only overestimated SOL by 5 and 2 min in the "good" and "neutral" conditions respectively (main effect subjective vs. objective SOL: $F(20, 1) = 54.08$, $p < 0.001$, $\eta 2 = 0.73$; interaction with condition: $F(19, 2) = 11.62$, $p = 0.001$, $\eta 2 = 0.55$). Similarly, in the "bad" condition participants reported having spent 39.55 ± 8.04 min WASO, whereas the actual time spent awake was of 13.84 ± 6.18 min (overestimation of ca. 26 min). In contrast, overestimation of WASO was only 5 min in the "good" condition and less than 1 min in the "neutral" condition (main effect subject vs. objective WASO: $F(20,2) = 7.75$, $p = 0.003$, $\eta 2 = 0.44$; interaction with condition, $F(20,2) = 4.83$, $p = 0.019$, $\eta 2 = 0.33$). Thus, the degree of overestimation was larger in the "bad" condition as compared to the "good" and "neutral" conditions (see Table 1).

In spite of the high overestimation of SOL in the "bad" condition, subjective estimates of SOL were still positively and significantly correlated with the objective measure of sleep onset (Pearson's $r = 0.58$, $p = 0.004$; see Table 3). Also, in the "good" condition, subjective and objective SOL were moderately to highly correlated ($r = 0.64$, $p = 0.001$). Only in the "neutral" condition no significant or positive correlation was observed between these two measurements ($r = 0.23$, $p > 0.30$). WASO was not significantly correlated in any of the three conditions ("bad": $r = 0.14$, $p > 0.50$, "good": $r = 0.31$, $p > 0.17$, "neutral": $r = 0.27$, $p > 0.23$). In addition, objective NWAK was negatively correlated with subjective sleep quality ($r = -0.56$, $p = 0.004$) and sleep depth ($r = -0.65$, $p \leq 0.001$) in the "neutral" condition (see Table 3).

Table 3. Correlation between objective and subjective sleep parameters

			Objective Sleep Parameters				
			Sleep Efficency	SOL	WASO	NWAK	Depth (N3)
Subjective Sleep Parameters	Bad	Sleep Quality	-0.059	0.067	-0.203	-0.108	0.05
		SOL	-0.58**	0.58**	0.19	-0.09	0.183
		WASO	0.115	-0.13	0.142	0	0.099
		NWAK	0.376	-0.36	0.222	0.24	-0.256
		Depth	0.02	-0.015	-0.274	-0.35	0.24
	Good	Sleep Quality	0.192	-0.189	-0.183	-0.35	-0.051
		SOL	-0.64**	0.64**	0.07	0.22	-0.284
		WASO	-0.162	0.16	0.305	0.079	0.028
		NWAK	-0.283	0.28	0.294	0.197	0.066
		Depth	0.23	-0.229	0.039	-0.413	0.124
	Neutral	Sleep Quality	0.221	-0.221	-0.257	-0.555**	0.209
		SOL	-0.233	0.232	0.1	0.357	0.04
		WASO	-0.181	0.176	0.266	0.221	-0.101
		NWAK	-0.26	0.258	0.485*	0.256	-0.242
		Depth	0.341	-0.34	-0.109	-0.653**	0.094

Notes: Analysis with an automatic sleep scoring algorithm (The SIESTA Group Schlafanalyse GmbH, Vienna) providing the number of arousals, an arousal index and stage shifts. Number of arousals as well as the arousal index were analyzed for total sleep time (TST), non-rapid eye movement as well as rapid eye movement sleep (REM). Further stage shifts from any sleep stage to wake and to NREM sleep, stage 1, 2, and 3 sleep (N1, N2, N3) are shown. Values are means ± standard error of mean (SEM). ** indicates $p \leq 0.01$

Memory consolidation and Vigilance

In our study, we also tested whether the instructions to sleep "good", "neutral" or "bad" could affect memory consolidation during sleep and vigilance the next morning. Pre-sleep instructions did not affect overnight memory formation differentially ($F(2, 18) = 0.508$, $p > 0.60$). However, the pre-sleep instruction to sleep "bad" resulted in slower reaction times in the Psychomotor Vigilance Test (PVT) directly after waking up. Two participants were excluded because of the high amount of errors (> 3 SD). Vigilance (as indicated by reaction time) was significantly lower after sleep in the "bad" condition (368.12 ± 12.14 milliseconds) as compared to the "neutral" (342.48 ± 7.79 milliseconds) and "good" conditions (332.42 ± 7.52 milliseconds, $F(2,42) = 7.99$; $p = 0.001$, $\eta 2 = 0.25$ see Figure 3, for post-hoc tests). In an exploratory analysis, we examined whether the decreased vigilance after the "bad" night, as compared to the "neutral" night, could be explained by differences in subjective or objective sleep parameters. Thus, we correlated the difference in reaction time on the PVT task between "bad" and "neutral" conditions, with the difference in sleep parameters between the "bad" and "neutral" conditions. Interestingly, neither any objective nor any subjective sleep parameters correlated with the decrease in

reaction time in the "bad" condition (corrected for "neutral") (all $p > 0.10$; see Table 4). When calculating the difference between "good" and "neutral" conditions, we observed several correlations with subjective but not objective sleep parameters: the difference in reaction time correlated with differences in subjective ratings of sleep quality ($r = -0.627$, $p = 0.002$) and with the feeling after waking-up ($r = -0.572$, $p = 0.005$) between the two conditions. As positive differences in sleep quality ratings (e.g. better sleep quality in "good" vs. "neutral" conditions) are predictive for a more negative difference in reaction times (e.g. fast reaction time in "good" vs. "neutral" conditions), negative correlations are to be expected.

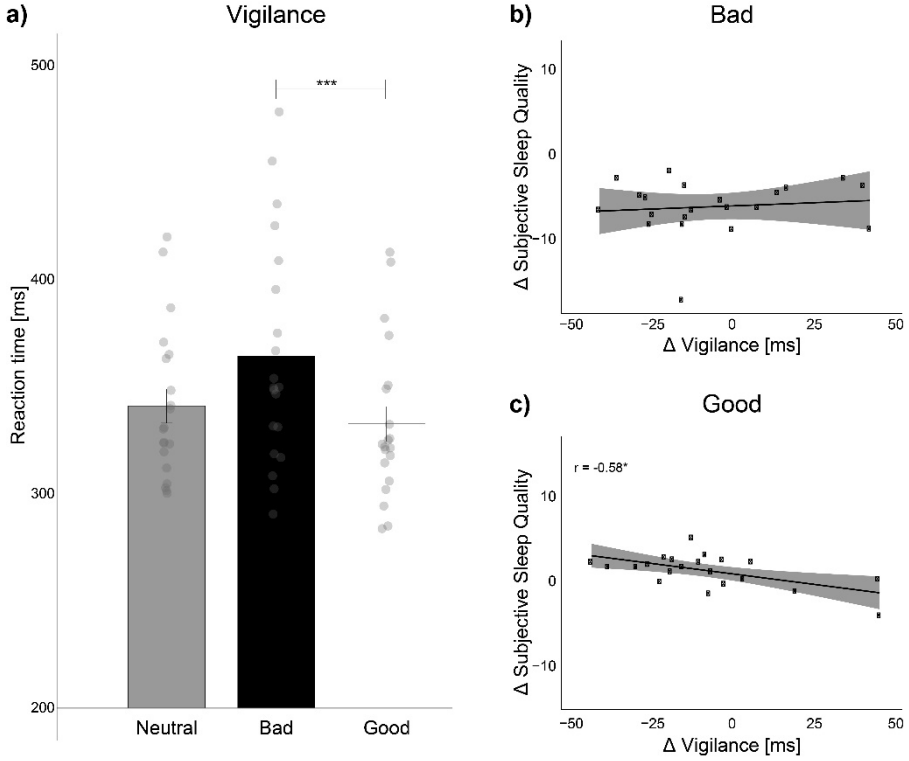

Figure 3. Effects of the instructions to change sleep quality on morning vigilance.(a) The instruction to sleep "bad" increased reaction in the psychomotor vigilance test (PVT) as compared to the "good" and "neutral" night (main effect condition $p = 0.001$). Post-hoc tests revealed a significant difference between "bad" and "good" instructions ($p = 0.034$) but not between "bad" and "neutral" ($p = 0.11$) or "good" and "neutral" ($p > 0.46$). Means ± standard errors of the mean are indicated. (b) The overall increase in reaction time in the PVT in the "bad" condition (corrected for performance in the "neutral" condition) was not correlated with any of our main subjective or objective sleep parameters (see Table 3). (c) In the "good" condition, the change in reaction (corrected for the neutral condition) correlated negatively with the change in subjective sleep between good and neutral sleep conditions. The direction of the correlation were expected, as better sleep correlated with faster reaction times and better vigilance. * indicates $p \leq 0.05$.*** indicates $p \leq 0.001$

Table 4. Correlation of Vigilance with subjective and objective sleep parameters

Vigilance	Δ Bad	Δ Good	Δ Bad	Δ Good
	Subjective Sleep		Objective Sleep	
Δ Sleep Quality	0.092	-0.627***	0.030	-0.008
Δ SOL	-0.047	-0.289	0.009	0.009
Δ WASO	-0.065	0.133	-0.034	0.309
Δ NWAK	0.081	0.169	-0.044	0.413**
Δ Sleep Depth	-0.058	-0.194	0.311	0.201

Notes. Correlations were calculated separately for the "good" and "bad" conditions. All parameters are corrected for the values obtained in the neutral condition (Δ Bad: "bad" – "neutral" and Δ Good: "good" – "neutral", respectively). ** indicates $p \leq 0.01$. *** indicates $p \leq 0.001$.

Heart rate and heart rate variability before sleep

As the instruction to sleep "bad" had a clear negative impact on subjective and objective sleep parameters, we examined whether the difference in physiological arousal before falling asleep could explain these changes. First, we analyzed average heart rate before falling asleep (from lights off to the first sign of sleep (N1)). Surprisingly, participants had the lowest heart rate before sleep in the "bad" condition (65.99 bpm ± 1.05) as compared to the other conditions ("good" = 69.73 ± 1.34; "neutral" = 67.16 ± 1.21). This decrease in heart rate reached a statistical trend ($F(2,39) = 2.52$, $p = 0.097$, $\eta 2 = 0.03$). Regarding heart rate variability, neither of the indexes for activity of the sympathetic tone (SNS index) nor for the parasympathetic tone (PNS index) were affected by the pre-sleep instructions (both $p > 0.30$). No significant correlations between heart rate / heart rate variability and any subjective or objective sleep parameters were observed (all $p > 0.05$). However, one exception to this is found in the differences in heart rate between the "good" and "neutral" conditions as they were positively correlated with the difference in objective WASO ($r = 0.453$, $p = 0.017$, see Table 5).

Table 5. Correlation of Heart Rate with subjective and objective sleep parameters

Heart Ratr [bpm]	Δ Bad	Δ Good	Δ Bad	Δ Good
	Subjective Sleep		Objective Sleep	
Δ Sleep Quality	0.193	0.013	0.012	0.283
Δ SOL	0.029	-0.262	0.011	-0.278
Δ WASO	-0.112	-0.304	0.245	0.453*
Δ NWAK	-0.017	0.154	0.085	0.244
Δ Sleep Depth	0.085	0.029	-0.037	0.313

Notes. Correlations were calculated separately for the "good" and "bad" conditions. All parameters are corrected for the values obtained in the neutral condition (Δ Bad: "bad" – "neutral" and Δ Good: "good" – "neutral"). * indicates $p \leq 0.05$.

Subjective reports how participants manipulated their sleep

Participants reported different methods in order to manipulate their sleep according to the pre-sleep instructions. To subjectively affect their sleep in a negative way, most participants reported trying to keep their eyes open (7 reports), to do exercises with fingers, legs or arms (2 reports) or to lie in an uncomfortable sleep position (4 reports). With regard to mental strategies that were implemented to sleep "bad", participants reported generally activating their thinking (12 reports), thinking about their planning and their to-do-list for the next day / week (8 reports) and thinking about negative / sad events (6 reports). One participant mentioned that he got upset about not falling asleep and another one recited a poem.

To affect sleep positively, participants mentioned using relaxation techniques such as breathing techniques, meditation and mind clearing ("do not think at all"), as well as muscle relaxation (11 times) and finding a comfortable and relaxing position to sleep in. In addition, participants mentioned thinking about positive memories and positive situations six times. Another strategy used by various participants was the active generation of sleep-related thoughts (e.g. "fall asleep fast", "sleep as usual" (as they knew they were healthy sleepers) or "fall asleep").

Discussion

Our results support the previously mentioned prediction that intention can decrease sleep quality but not improve it in healthy young participants. On the subjective level, all of our five primary outcome measures (sleep quality, SOL, WASO, NWAK, and sleep depth) show this pattern of results. On the level of objective sleep parameters, three out of five primary outcome values (SE, SOL, and NWAK) also support this pattern of results, whereas no effects of voluntary sleep manipulation were observed for WASO and the amount of SWS.

As we examined sleep in young healthy participants, their sleep during the night was already very good without having to provide them with any instructions: on average they had a high sleep efficiency (over 97%), they fell asleep after 12 minutes and had only 2% WASO during the total of the 8 hours of sleep. Average sleep stage duration and sleep stage distribution also was in the normal range. As this sleep pattern is close to optimal sleep, there is probably not much room for improvement of sleep simply by wanting to do so. To achieve good sleep, participants reported using breathing or relaxation techniques as well as thinking positive thoughts. Still, these techniques did not produce any further improvement in sleep. However, most of these techniques need a certain degree of training, a reason which may help explain why our physiological parameters of sleep showed no changes (Morin et al., 1999). For example, oscillatory power during the night – and particularly SWA, which is an important measure of the homeostatic component of sleep – was not altered by our experimental manipulation, which might be interpreted as evidence that the sleeping brain is regulated mainly in a physiological manner (Allada et al., 2017; Finelli et al., 2000). However, as sleep was close to optimal, our null finding

does not imply that these techniques or intentions in general, are ineffective in improving sleep. We can only conclude that intention cannot further improve very good sleep in healthy young participants. In patients with insomnia, relaxation techniques have been shown to improve some aspects of sleep (Kahn et al., 1968; Means et al., 2000; Nicassio & Bootzin, 1974). In addition, the anticipation of receiving a treatment or a medication thinking that it will help sleep (known as the "placebo" effect) can improve subjective and also some objective sleep variables in persons with disturbed sleep (Colagiuri et al., 2012; Neukirch & Colagiuri, 2015; Winkler & Rief, 2015). Also in healthy sleeper, sleep improvements (extension of SWS and increases of SWA) are possible for example, using music (Cordi et al., 2019; Lai & Good, 2006), hypnotic suggestions (Cordi et al., 2014, 2020), or rocking bed movements (Bayer et al., 1993)).

Some participants also reported that they generated sleep related thoughts (e.g. "fall asleep fast"), which is in line with a paradoxical negative rebound (Rezaie et al., 2018). Previous studies have suggested that wanting to fall asleep, prolongs sleep latency and impairs sleep quality (Espie & Wicklow, 1999; Schmidt et al., 2018; Wicklow & Espie, 2000), as the intention to fall asleep fast can generate cognitive arousal which impairs sleep. Conversely, insomnia patients can profit from the paradoxical instruction "to try to stay awake" (Morin et al., 1999). In our study we did not find any evidence for a strong impairing effect of the intention to sleep "good" on sleep in young healthy participants on our primary outcome measure.

However, in our exploratory analysis, during the "sleep good" night the percentage of REM sleep was significantly reduced and occurred descriptively (but not significantly) later, as compared to the other two conditions. The exact role of REM sleep for "good" vs. "bad" sleep is not clear. From research in depressive participants, an earlier onset of REM sleep and more REM sleep has been associated with stronger depressive symptoms, and a reduction in REM sleep has been suspected to be beneficial for treatment outcomes (Habukawa et al., 2018; Pesonen et al., 2019; Steiger & Kimura, 2010; Steiger & Pawlowski, 2019; Wichniak et al., 2000). However, whether less REM sleep is beneficial for emotional regulation in healthy participants is not clear. As participants were also not instructed to manipulate REM sleep, the reason and impact of the reduction of REM sleep after the instruction to sleep "good" remains elusive but may be interpreted as an impairment on sleep.

In contrast to the "good" condition, the instruction to sleep "bad" clearly impaired sleep quality both on the level of subjective but also objective sleep parameters. Thus, healthy participants are able to impair their sleep intentionally. Participants were particularly capable of extending the time it took them to fall asleep. Importantly, they were not allowed to get up, but they had to lie in bed in the darkness without any distraction (e.g. reading, video, social media, etc.). Thus, they had to stay awake only using their intention as a way of affecting their sleep. As for the strategies used, they reported keeping their eyes open, actively generating (negative) thoughts and planning activities for the next day. Objectively, by using these strategies they managed to stay awake 17 minutes longer on average, compared to the "neutral" night. While this is a robust and reliable extension of SOL by intention, the additional time of

17 minutes is also not very much, and a SOL of 30 minutes in total is probably still in the normal range. Thus, in healthy sleepers, sleep pressure is strong enough to override intentions and induce sleep within 30 – 45 minutes, although some subjects were able to stay awake for 75 minutes.

In addition to SOL, participants instructed to sleep "bad" were capable of increasing the NWAK as well as the number of shifts from any sleep stage to wake. On the other hand, they were not capable of increasing WASO. This finding is particularly important as it shows that the intention to sleep "bad" was still active while the participant was sleeping, resulting in an increased drive to wake up from the sleep state but not to stay awake as much as possible. The fact that one's brain can automatically and gradually prepare to wake up because one was asked to wake up as much as possible during the night, falls in line with an individuals' ability to program their awakenings at a scheduled time (such as in the case of an alarm). This is indicated by increases in plasma concentration of adrenocorticotropin during the sleep period before the expected awakening, although sleep stages remained unchanged in this study (Born et al., 1999). Thus, in spite of the strongly reduced consciousness during sleep, our intentions and goals, which are activated during wakefulness before going to sleep, remain active during the sleeping state and can influence the sleep state itself. In both the "bad" and "good" conditions, sleep should be affected by our intention. Intentions are part of our executive functions, which are mainly located in the prefrontal cortex (PFC). While the neurobiological mechanisms of intentions are not completely clear, it is widely assumed that they are capable of modulating activity and sensitivity in other brain areas (top-down-regulation). For example, the intention to execute motor movements typically alters motor-related brain activity before the actual initiation of the movement (Jeannerod, 1994; Wairagkar et al., 2018). Intentions and plans "to do something" in the future are typically conceptualized as prospective memories (Barner et al., 2017, 2019; Fabbri et al., 2014). In addition, intentions can remain active over long periods (e.g. "go to the post office tomorrow at 3 pm"). These "prospective memories" rely on prefrontal brain areas, but also involve other memory-related brain areas, in particular the parietal lobe (e.g. precuneus; Burgess et al., 2011). Thus, we assume that the formation of the intention "to sleep worse" or "to sleep better" - based on the PFC - will have top-down influences on the balance between wake-promoting arousal systems and sleep-promoting GABAergic inhibitory systems of our brain (i.e., flip flop switch model of sleep and wake promoting brain regions; Saper et al., 2001). The top-down influence of the intention will be most influential during the phase of falling asleep, because consciousness is still present during this period. However, together with reduced consciousness during the sleep period, the intention might remain active and have a top-down influence on sleep-maintaining systems. Similar to other prospective memories, this continued influence during sleep might involve memory-related brain areas and mechanisms. Our result suggest that the prospective memory system maintains its function also during the sleep state. The intentions set before sleep are continuously maintained and activated in our prospective memory system throughout sleep. Alternatively, one could speculate that future plans set before sleep are stored as memory representations in the brain. As memories have been shown to be reactivated during certain sleep stages (Rasch & Born, 2013), it might

be possible that future plans are also repeatedly and spontaneously reactivated during sleep. For declarative types of memory, evidence indicates that information acquired before sleep is spontaneously and repeatedly reactivated during SWS and possibly also during NREM sleep stage 2 (Rasch & Born, 2013). On a conceptual level, the instruction to sleep "bad" could also be regarded as a new memory acquired before sleep, which might then be reactivated during sleep alongside other memories. When reactivated, this plan to sleep "bad" and "wake up during the night" could affect the ongoing sleep process, thereby resulting in an increased number of awakenings during sleep as well as more shifts from any sleep stage to wake. However, this explanation requires further empirical support.

Generally, our results show that the instructions to sleep "bad" do not only affect SOL, but also ongoing sleep processes after sleep onset. These findings are in line with the result that the instruction of an early wake up time in the morning induces a preparation of a wake response 2-3 hours before the actual time of awakening (Born et al., 1999). As follows, this is consonance with anecdotal reports that people tend to wake up before their alarm clock or hours before an important wake up time (e.g. due to catching a flight).

The effects caused by the instruction to sleep "bad" cannot be explained by increases in physiological arousal before or during sleep. It might have been possible that the participants extended their sleep onset by voluntarily increasing their physiological arousal level while lying in bed. However, heart rate analysis during the period falling asleep provided no support for this claim. In fact, heart rate even tended to be lower in the "bad" condition as compared to the other two conditions. Thus, a psychological explanation for the decreases in sleep quality is more likely.

The decreased sleep quality after the instruction to sleep "bad" affected behavioral performance in the morning and decreased measures of vigilance. Interestingly, the individual difference in decreased vigilance after the "bad" night could not be explained neither by differences in subjective nor objective sleep parameters. Thus, it remains unclear which sleep parameter exactly contributes to the reduction in vigilance. The significant correlation in the "good" condition raises the suspicion that reaction time in the PVT might be particularly influenced by the subjective evaluation of one's own sleep quality. However, as this correlation is not present in the "bad" condition, the increase in reaction time cannot be explained by participants' impression that they have slept worse.

SOL, WASO and NWAK were generally overestimated in the subjective reports as compared to the objective parameters, but these parameters on the subjective level correlated positively with the same parameters on an objective level, with small to medium effect sizes. Interestingly, the instruction to sleep "bad" caused a higher subjective-objective discrepancy as compared to the "good" and "neutral" condition. As scientists are aware of the effects of a reward related task (i.e. receiving money as compensation for good performance), one could speculate that participants rated their sleep worse in the "bad" condition because they wanted to fulfill the expectations of the experimenter who asked them to sleep "bad". However, this kind of demand characteristic should equally affect subjective and objective ratings in the "good" condition. Here, no evidence of the same participants fulfilling the experimenter's

expectations were detectable in the data. Therefore, we consider the role of demand characteristics for the subjective sleep ratings as minor. Especially because we showed that differences exist between objective and subjective sleep parameters. A higher discrepancy between objective and subjective sleep parameters is an important symptom in subtypes of insomnia patients (Manconi et al., 2010; Rezaie et al., 2018), and a decrease of this discrepancy is related to improvements in insomnia symptoms (Crönlein et al., 2020; Mitchell et al., 2019). A recent pilot study suggests that the subjective opinion of one's own sleep length and quality can have an even stronger impact on feelings of tiredness and impaired cognitive performance the next day when measured as objective sleep parameters (Rahman et al., 2020). Our results show that the simple instruction to sleep "bad" can strongly increase the discrepancy between objective and subjective sleep parameters, while the same person shows a much lower discrepancy when he or she expects his or her sleep to be normal or even good. This finding highlights the possibility that our perception of sleep largely depends on our psychological goals and intentions related to our sleep, and less on changes of objective parameters during sleep (see e.g. Riemann et al., 2012).

While our study clearly shows that intentions are capable of influencing sleep in healthy participants, objective changes in sleep parameters, in particular, remain rather small. For example, we did not observe any effects of the sleep instructions on power in the SWA band, which we already know has been implicated in the process of homeostatic regulation of sleep. This result shows that a major part of objective sleep is regulated by physiological mechanisms that are beyond our intentional control. Our results also suggest that intentional processes cannot explain previous reports on the extension of SWS and the increase in SWA by means of more subconscious interventions (e.g. placebo effects (Fratello et al., 2005), music (Cordi et al., 2019; Lai & Good, 2005), hypnotic suggestions (Cordi et al., 2014)). Thus, the sole instruction of sleeping "good", "bad" or "neutral" is not enough to find large effects on objective sleep architecture.

Conclusion

In sum, here we have shown that intentions can impair, but not improve healthy sleep. Thus, bad sleep can be the result of our intention to sleep "bad". Furthermore, the intention to sleep "bad" increases the discrepancy between subjective and objective sleep parameters, resulting in a stronger overestimation of the SOL, WASO and NWAK as compared to the other two instructions. Furthermore, participants were also capable of extending their sleep onset and increasing their NWAK on an objective level. Thus, the goal to sleep "bad" was still active during the sleep state and resulted in a disturbance of the sleep process, suggesting that goals and intentions are relevant for maintaining a stable sleep state. While the simple instruction to sleep "good" was not enough to further improve sleep, recent studies conducted in our laboratory show that the amount of SWS can be extended by using more subconscious techniques, such as hypnotic suggestions (Cordi et al., 2014, 2020). Taken together, our intentions, thoughts and imagination are important influencing factors of our subjective and objective sleep quality.

Limitations

Although our study design was already complex, we are aware that three nights are not sufficient to verify all relevant points of our hypothesis. Among others, one limitation regards our study design: we only used three experimental nights. In particular, an additional night without any instructions would have been important to estimate whether our "neutral" instruction could also alter, or at least influence, sleep. As a second point, the criteria of absence of sleep disorders in our recruiting process (only healthy sleepers are recruited to participate in this non-clinical trial) was only confirmed by the PSQI (Buysse et al., 1989). This questionnaire is used as an instrument to assess self-reported sleep quality and does not allow to identify sleep disturbances or insomnia based on proper diagnostic criteria. Additional tests for sleep disorders were not conducted nor was sleep history assessed throughout our participants. However, the very high sleep efficiency renders sleep disorders in our sample rather unlikely. Another limitation of our study is that participants may have reached a ceiling effect in different sleep parameters, such as sleep efficiency, resulting in high values in all conditions. Therefore, discrimination between participants in the different conditions was unfeasible, thus the expected improvement when trying to sleep better than normal was not possible. The lack of changes in different sleep parameters may be due to a partially sleep-restricted population resulting from the instruction to get up at 7 am on the day of the experiment. For higher clinical relevance, future studies should systematically examine the influence of intentions on sleep in participants with sleep disturbances.

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Supplementary Material

Table S1. Sleep Cycles for conditions

	Bad	Good	Neutral	
	M ± SEM	M ± SEM	M ± SEM	F-Test
Cycle 1	n = 22			
Duration	90.59 ± 8.11	101.93 ± 10.41	98.92 ± 10.30	0.479
N1 [%]	9.72 ± 3.14	8.35 ± 1.83	11.59 ± 4.85	0.353
N2 [%]	29.60 ± 3.44	34.89 ± 3.13	31.12 ± 3.50	0.940
N3 [%]	42.87 ± 5.03	43.28 ± 4.98	46.90 ± 4.88	0.282
REM [%]	14.25 ± 2.85	11.12 ± 2.37	9.06 ± 1.06	1.682
Wake [%]	2.90 ± 1.10	2.03 ± 0.83	1.03 ± 0.33	1.787
N1 [min]	6.52 ± 1.28	7.57 ± 1.28	6.64 ± 1.42	0.290
N2 [min]	29.66 ± 4.79	36.41 ± 5.64	35.66 ± 6.76	0.571
N3 [min]	39.11 ± 4.75	44.11 ± 5.94	45.98 ± 4.96	0.619
REM [min]	12.25 ± 2.15	11.11 ± 2.59	9.02 ± 1.40	0.717
Wake [min]	2.32 ± 0.67	2.39 ± 0.90	1.09 ± 0.38	1.659
Cycle 2	n = 22			
Duration	101.73 ± 6.11	103.52 ± 6.76	103.64 ± 5.38	0.038
N1 [%]	4.36 ± 0.83	5.84 ± 0.73	5.77 ± 1.10	1.033
N2 [%]	37.22 ± 3.45	42.14 ± 3.40	41.81 ± 2.92	1.157
N3 [%]	36.46 ± 4.18	31.34 ± 4.26	30.73 ± 4.75	0.819
REM [%]	19.12 ± 1.97	19.18 ± 1.80	19.86 ± 1.71	0.071
Wake [%]	2.01 ± 0.63	0.91 ± 0.42	1.36 ± 0.81	0.857
N1 [min]	4.02 ± 0.60	6.34 ± 0.08	6.52 ± 1.53	1.939
N2 [min]	37.52 ± 3.64	44.32 ± 4.98	44.32 ± 4.19	1.216
N3 [min]	37.02 ± 5.00	31.43 ± 4.69	29.39 ± 4.50	1.140
REM [min]	20.18 ± 2.40	19.75 ± 2.12	21.11 ± 2.09	0.129
Wake [min]	2.16 ± 0.71	1.16 ± 0.66	1.77 ± 1.14	0.388
Cycle 3	n = 22			
Duration	102.05 ± 7.94	111.75 ± 6.09	113.93 ± 4.79	1.168
N1 [%]	10.86 ± 3.33	7.58 ± 0.97	7.16 ± 1.05	1.054
N2 [%]	43.03 ± 3.48	53.41 ± 2.21	49.30 ± 3.95	3.079
N3 [%]	19.00 ± 4.21	12.53 ± 2.18	16.54 ± 3.97	0.946
REM [%]	21.13 ± 2.22	22.20 ± 2.32	24.37 ± 2.22	0.635
Wake [%]	5.07 ± 1.54	3.79 ± 1.77	1.85 ± 0.61	1.377
N1 [min]	8.02 ± 1.70	8.48 ± 1.29	8.48 ± 1.42	0.038
N2 [min]	46.36 ± 5.12	59.36 ± 3.63	55.89 ± 4.67	3.068**
N3 [min]	19.11 ± 4.05	12.55 ± 2.12	18.07 ± 4.10	1.178
REM [min]	22.86 ± 3.00	25.32 ± 3.13	28.34 ± 2.99	0.928
Wake [min]	4.70 ± 1.43	5.52 ± 2.94	2.30 ± 0.80	0.827

Cycle 4	n = 15			
Duration	100.97 ± 5.77	98.20 ± 7.93	97.43 ± 6.18	0.196
N1 [%]	8.26 ± 1.54	9.85 ± 2.05	7.19 ± 1.12	1.428
N2 [%]	50.65 ± 3.41	47.71 ± 4.75	51.95 ± 3.68	0.318
N3 [%]	6.60 ± 2.20	19.29 ± 5.15	15.00 ± 4.57	5.151**
REM [%]	31.42 ± 3.18	21.67 ± 2.35	24.15 ± 2.44	3.141*
Wake [%]	1.72 ± 0.69	0.67 ± 0.30	0.78 ± 0.52	1.114
N1 [min]	8.03 ± 1.69	8.23 ± 1.37	6.80 ± 1.03	0.645
N2 [min]	51.70 ± 3.99	44.76 ± 5.58	49.97 ± 4.15	1.032
N3 [min]	7.17 ± 2.52	20.87 ± 5.95	14.20 ± 3.74	5.844**
REM [min]	31.03 ± 3.14	22.77 ± 3.05	24.57 ± 3.11	2.365
Wake [min]	1.60 ± 0.69	0.73 ± 0.34	1.00 ± 0.73	0.526
Cycle 5	n = 5			
Duration	85.75 ± 16.75	69.50 ± 24.50	78.25 ± 4.75	0.67
N1 [%]	8.32 ± 3.41	12.85 ± 2.71	6.84 ± 1.54	1.39
N2 [%]	55.30 ± 7.44	50.92 ± 5.91	55.05 ± 8.98	0.09
N3 [%]	16.22 ± 7.99	7.67 ± 5.36	-	1.95
REM [%]	18.02 ± 4.44	24.27 ± 3.29	37.03 ± 8.66	2.73
Wake [%]	1.21 ± 1.09	3.65 ± 2.46	0.30 ± 0.30	1.10
N1 [min]	7.10 ± 3.76	9.90 ± 2.52	5.70 ± 1.69	0.62
N2 [min]	41.20 ± 6.11	39.40 ± 6.22	41.60 ± 8.85	0.04
N3 [min]	11.70 ± 5.49	6.70 ± 4.47	-	4.55
REM [min]	14.30 ± 4.97	19.00 ± 3.14	28.40 ± 6.79	1.89
Wake [min]	1.10 ± 0.98	2.80 ± 1.71	0.30 ± 0.30	1.09

Notes. Cycle analysis of sleep stages: Non-rapid eye movement (NREM)-sleep, stage 1, 2, and 3 sleep (N1, N2, N3), rapid eye movement sleep (REM), waketime after sleep onset (WASO), total sleep time (TST), sleep onset latency (SOL), slow wave sleep latency (SWS latency), REM sleep latency (REM latency) in minutes [min] and in parietal percentage of TST [%]. M and SEM are used to represent mean and standard error of means. * indicates $p \leq 0.05$. ** indicates $p \leq 0.01$ *** indicates $p \leq 0.001$.

Table S2. Power bands during sleep

		dfNum	dfDen	F
Slow Oscillations (SO)	**[0.5 - 1.5 Hz]**			
NREM	Hemisphere	1	15	4.62*
	Topography	1.15	17.26	49.23
	Condition	1.66	24.93	0.39
	Topography x Hemisphere	2.37	35.54	1.78
	Topography x Condition	2.88	43.26	2.06
	Hemisphere x Condition	1.46	21.88	0.46
	Topography x Hemisphere x Condition	4.36	65.4	1.44
REM	Hemisphere	1	15	2.01
	Topography	2.51	37.72	5.16**
	Condition	1.37	20.55	0.36
	Topography x Hemisphere	1.63	24.47	1.79
	Topography x Condition	2	30.05	2.01
	Hemisphere x Condition	1.8	26.93	1.03
	Topography x Hemisphere x Condition	2.24	33.54	1.44
Slow Wave Activity (SWA)	**[0.5 - 4.5 Hz]**			
NREM	Hemisphere	1	15	3.47
	Topography	1.15	17.2	47.53***
	Condition	1.71	25.59	0.44
	Topography x Hemisphere	2.48	37.19	2.35
	Topography x Condition	2.98	44.68	2.1
	Hemisphere x Condition	1.55	23.2	0.68
	Topography x Hemisphere x Condition	4.29	64.32	1.45
REM	Hemisphere	1	15	0.51
	Topography	2.37	35.48	4.05*
	Condition	1.82	27.34	0.26
	Topography x Hemisphere	1.65	24.75	1.9
	Topography x Condition	1.88	28.14	2.05
	Hemisphere x Condition	1.8	26.95	1.67
	Topography x Hemisphere x Condition	1.93	28.9	1.31
Delta	**[1.5 - 4.5 Hz]**			
NREM	Hemisphere	1	15	0.53
	Topography	1.24	18.55	36.34***
	Condition	1.95	29.25	0.57
	Topography x Hemisphere	2.66	39.92	3.45*
	Topography x Condition	3.16	47.37	2.12
	Hemisphere x Condition	1.87	28.09	1.3
	Topography x Hemisphere x Condition	3.22	48.29	1.18

REM	Hemisphere	1	15	0.28
	Topography	1.45	21.73	2.55
	Condition	1.52	22.77	0.28
	Topography x Hemisphere	1.23	18.43	1.43
	Topography x Condition	1.24	18.54	1.58
	Hemisphere x Condition	1.22	18.34	1.88
	Topography x Hemisphere x Condition	1.24	18.53	1.06
Theta	**[4.5 - 8 Hz]**			
NREM	Hemisphere	1	15	0.1
	Topography	2.06	30.87	16.14***
	Condition	1.39	20.81	0.31
	Topography x Hemisphere	1.42	21.32	2.16
	Topography x Condition	1.63	24.48	1.37
	Hemisphere x Condition	1.22	18.23	1.4
	Topography x Hemisphere x Condition	1.28	19.24	1.46
REM	Hemisphere	1	15	1.01
	Topography	1.15	17.19	1.5
	Condition	1.21	18.13	0.36
	Topography x Hemisphere	1.11	16.63	1.1
	Topography x Condition	1.07	16.02	1.19
	Hemisphere x Condition	1.05	15.81	1.62
	Topography x Hemisphere x Condition	1.1	16.44	1.04
Alpha	**[8 - 11 Hz]**			
NREM	Hemisphere	1	15	0.25
	Topography	1.91	28.68	28.21***
	Condition	1.88	28.19	0.51
	Topography x Hemisphere	2.19	32.9	3.12
	Topography x Condition	3.04	45.6	1.27
	Hemisphere x Condition	1.93	28.94	2.07
	Topography x Hemisphere x Condition	2.5	37.54	2.5
REM	Hemisphere	1	15	2.85
	Topography	1.99	29.87	3.65*
	Condition	1.76	26.42	0.44
	Topography x Hemisphere	1.45	21.81	1.39
	Topography x Condition	1.49	22.32	1.05
	Hemisphere x Condition	1.18	17.74	1.16
	Topography x Hemisphere x Condition	1.3	19.46	1.93

Sigma	**[11 - 15 Hz]**			
NREM	Hemisphere	1	15	0.29
	Topography	2.03	30.44	59.63***
	Condition	1.36	20.47	0.98
	Topography x Hemisphere	2.3	34.55	3.96*
	Topography x Condition	3.21	48.17	1.21
	Hemisphere x Condition	1.97	29.62	1.9
	Topography x Hemisphere x Condition	2.28	34.19	2.29
REM	Hemisphere	1	15	0.58
	Topography	1.76	26.33	2.21
	Condition	1.96	29.47	0.36
	Topography x Hemisphere	1.46	21.86	1.32
	Topography x Condition	1.35	20.2	1.4
	Hemisphere x Condition	1.25	18.8	0.69
	Topography x Hemisphere x Condition	1.29	19.4	1.43
Beta	**[15 - 30 Hz]**			
NREM	Hemisphere	1	15	3.05
	Topography	2.46	36.94	24.49***
	Condition	1.8	27.01	0.78
	Topography x Hemisphere	2.29	34.31	2.79
	Topography x Condition	2.91	43.58	1.41
	Hemisphere x Condition	1.98	29.69	0.2
	Topography x Hemisphere x Condition	2.22	33.28	1.63
REM	Hemisphere	1	15	0.12
	Topography	2.18	32.74	6.85**
	Condition	1.89	28.31	1.26
	Topography x Hemisphere	2.16	32.44	0.72
	Topography x Condition	3.15	47.31	0.82
	Hemisphere x Condition	1.97	29.62	0.11
	Topography x Hemisphere x Condition	3.06	45.85	1.59

Notes. Poweranalysis for non-rapid eye movement (NREM)-sleep (N2 + N3) and rapid eye movement sleep (REM). As factors Hemisphere (left vs. right), topography (frontal, central, parietal), and condition ("bad", "good", "neutral") were used. M and SEM are used to represent mean and standard error of means. * indicates $p < 0.05$. ** indicates $p < 0.01$. ** indicates $p < 0.001$.

3.2 Pre-Sleep Social Media Use does not strongly disturb Sleep: A Sleep Laboratory Study in healthy young participants [4]

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Highlights

- Pre-sleep social media consumption did not disturb sleep.
- No increased (pre-sleep) arousal due to social media use.
- Social media consumption leads to less time in sleep stage N2.
- Progressive muscle relaxation decreased arousal, improved sleep efficiency, reduced sleep onset latency, and shortened the time to reach slow-wave sleep.

[4] A similar version of this manuscript has been published in "Sleep Medicine"

Abstract

Objective: Sleep is critical for our mental health and optimal cognitive functioning. Social media use is increasingly common and suspected to disturb sleep due to increasing bedtime arousal. However, most studies rely on self-reported sleep.

Methods: We tested the effects of 30 min social media use on arousal and subsequent sleep in the sleep laboratory in 32 healthy young volunteers. Effects of blue-light were excluded in this study. We compared it to 30 min progressive muscle relaxation (PMR) and neutral sleep in a within-subject design.

Results: Thirty minutes of social media use immediately before sleep did not significantly increase arousal and did neither disturb objective nor subjective sleep. After social media use, participants only spent less time in sleep stage N2. In contrast, PMR had the expected positive effects on pre-sleep arousal level indicated by reduced heart rate. In addition, PMR improved sleep efficiency, reduced sleep onset latency, and shortened the time to reach slow-wave sleep compared to a neutral night. Oscillatory power in the slow-wave activity and spindle bands remained unaffected.

Conclusion: Social media use before sleep (controlling for effects of blue-light) had little effect on bedtime arousal and sleep quality than what was previously expected. The most notable effect appears to be the additional time spent engaging in social media use at bedtime, potentially keeping people from going to sleep. As wake up-time is mostly determined externally, due to school or working hours, limiting personal media use at bedtime — and especially in bed — is recommended to get sufficient hours of sleep.

Keywords

Social Media, Pre-sleep Arousal, PMR, Sleep, Sleep Quality

Introduction

The last decades have brought a rapidly increasing spread and use of new (mobile) communication technologies (Gradisar et al., 2013; Rideout et al., 2010; Statista, 2021b; Twenge et al., 2019). Increasing portability and affordability can be seen as a gateway for devices, such as TVs, laptops, or mobile phones, to enter peoples' bedrooms. This increasing prevalence has led to an ongoing and recurring discussion about the adverse effects of using such technologies in the bedroom and especially immediately before sleeping. Studies have shown that the availability of electronic devices (such as a television, a gaming console, or a mobile phone) in the bedroom impairs subjective reports of sleep (Cain & Gradisar, 2010; Exelmans & Van den Bulck, 2016; Exelmans & Van Den Bulck, 2017). In children and adolescents the availability and use of electronic devices is associated with shorter total sleep time (TST) due to delayed bedtime (Cain & Gradisar, 2010). In addition, adolescents with an increased use of laptops (for gaming, communicating, and surfing the internet) and mobile phones have been found to have poorer sleeping habits, thus leading to increased day-time tiredness compared to those with less use (Punamäki et al., 2007). Similar effects are reported for adults (Exelmans & Van den Bulck, 2016). Moreover, social media use – in particular on mobile phones – at bedtime impairs sleep, as indexed by later bedtime and sleep latencies, consequently decreasing daytime functioning (Pieters et al., 2014). Finally, social media use has been linked to a decrease in mental health and subjective sleep quality (Alonzo et al., 2021; Garett et al., 2018; Levenson et al., 2016). Despite the increased interest and efforts in research, studies to date almost exclusively rely on self-reports to assess the effects of social media use on sleep. Therefore, existing findings are based on subjective perceptions. There is a clear lack of data substantiating these effects from a sleep physiological perspective. This study aims to fill this gap by investigating the potentially harmful effects of media use immediately before sleep in a laboratory setting as indexed by physiological parameters (i.e., objectively measured sleep architecture and sleep-related brain oscillations like slow-waves, which are associated with a high sleep quality (Borbély et al., 1981; Borbély & Achermann, 1999; Maren J. Cordi et al., 2014)).

The physiology of social media use before bedtime

On a physiological level, studies demonstrate that the impairing effect of social media use at bedtime on subsequent sleep could be attributed to an increased arousal (Della Monica et al., 2018; Woods & Scott, 2016). This assumption is supported by several correlational studies which find subjective arousal at bedtime to be a significant explanatory factor for the positive association between media use at bedtime and sleep impairments, as indexed by, e.g., subjective reports of reduced sleep quality, interrupted sleep (Mauri et al., 2011; Van den Bulck, 2003; Woods & Scott, 2016). Insight from patients with insomnia also attribute a central role to increased arousal to explain sleep disturbances (Kalmbach et al., 2018; Riemann et al., 2010). This line of research identifies cognitive factors such as rumination or worrying as contributing to increase psychophysiological arousal (e.g., the activity of the sympathetic nervous system (SNS)), which consequently inhibits sleep-inducing processes and results

in sleep delays and disturbances. Decreasing physiological and cognitive arousal using cognitive-behavioral interventions are effective treatments of insomnia disorders and are, thus, key elements in cognitive-behavioral therapy of insomnia (Blake et al., 2017; Morin, 2004). As social media use – especially before bedtime – promotes cognitive and biological aspects of arousal (Anderson et al., 2004; Bodas et al., 2015; Carnagey, Anderson, & Bushman, 2007; Grabe & Kamhawi, 2006), it seems logical to assume that degradations in sleep can be attributed and explained by this process.

Despite the face validity of this explanation and outlined above, there is a lack of experimental studies that aim to rigorously investigate the effects of social media use at bedtime on objective sleep parameters (i.e. arousal and sleep itself). This study aims to contribute to fill this existing gap. In light of the existing theorizations and empirical findings outlined above, we predict that social media use at bedtime will impair sleep quality as indexed by both subjective and objective parameters. The interference should be attributable to an increase in pre-sleep arousal induced by social media use. In contrast, preforming PMR before sleep should decrease pre-sleep arousal and improve sleep quality.

In addition to sleep parameters, we included a memory measurement (Rasch et al., 2006) and a vigilance test (Dinges & Powell, 1985) to capture behavioural consequences of impaired sleep: Consolidation of memory benefits from sleep (Maren Jasmin Cordi & Rasch, 2021; Rasch & Born, 2013) and Vigilance is a sensitive measure of low-quality sleep (Lim & Dinges, 2008; Oken et al., 2006). We therefore hypothesized that lower sleep quality induces by social media use leads to impaired memory consolidation and vigilance, while higher sleep quality should improve both behavioral measures.

Materials and methods

Experimental design and conditions

To investigate these assumptions, we conducted a within-subject experimental study in a sleep laboratory. In these well-controlled conditions the effects of pre-sleep social media use can be investigated using both subjective and objective measures of sleep quality and sleep architecture. The within-subject design serves to insure high statistical power and enables the detection of medium effect sizes. In addition to an adaptation night, it included three experimental conditions which will be described below.

Neutral night (control)

In the “neutral” night participants were told to sleep as regularly as possible and went straight to bed without any intervention after completing questionnaires and preparatory procedures for sleep recording (see below). Thus, the “neutral” night serves as a baseline in the analyses.

Progressive muscle relaxation (PMR, relax)

In the "relax" condition participants performed an established intervention that aimed to reduce arousal (progressive muscle relaxation, PMR, (Mackereth & Tomlinson, 2010; Walters et al., 2003)), instead of using social media. A 30-minute instruction tape was used to ensure a comparison with the pre-sleep social media condition and to standardize the PMR. Participants followed a male voice guiding them through the PMR exercises by providing instructions (Furcht, 2003). We used the long form of 20 exercises. The instructions started with clenching the right hand into a fist and subsequent relaxation. Following this scheme and to reach deep relaxation, the major muscle groups of the body were tensed and relaxed throughout the PMR session. The experimenter checked the EMG several times to verify that participants performed the PMR. The tape was started 30 minutes before the lights were turned off. Participants were already lying in bed while performing this task.

Pre-sleep social media use

For the social media use condition, we chose to focus on social media use since it has been previously reported to have very severe effects on subsequent sleep (Levenson et al., 2017; Tandon et al., 2020; van der Schuur et al., 2019). To keep internal validity high, we limited the services to two social messaging providers, i.e., WhatsApp (WhatsApp LLC., Mountain View) and Snapchat (Snap Inc., Santa Monica), based on their capacity to allow individuals to engage in direct communication instead of passive consumption (Aichner & Jacob, 2015; Pagani et al., 2010). Passive social media use (e.g. viewing posts, scrolling through news feeds) means to consume information on social media platforms without connecting to others or commenting (Chen et al., 2016). WhatsApp was chosen as it is (or was at that time) the most popular social messaging provider. Snapchat was chosen because of its ability to allow active communication over passive consumption. In doing so, we limited the variety of other forms of usage, such as posting on other people's profiles, news consumption, etc. Introducing more ways of media use would have potentially introduced confounding factors that, given the complexity of studies in a sleep laboratory, we wouldn't have been able to account for in the subsequent analysis. Participants were instructed to use social media for 30 minutes. The use was restricted to the previously mentioned social media providers, which are both popular (Statista, 2021a) and allow users to actively engage in conversations instead of passive consumption of ([audio-]visual) content (Levenson et al., 2016). WhatsApp is a social messaging service allowing users to send and receive text and voice messages (WhatsApp LLC, Mountain View). Snapchat is a social multimedia service allowing person-to-person photo and video sharing within a restricted timeframe (Snap Inc., Santa Monica). To ensure only those two social media apps were used, only the servers of WhatsApp (WhatsApp LLC., Mountain View) and Snapchat (Snap Inc., Santa Monica) could be reached through the wireless located area

network (WLAN) available to participants[5]. Therefore, a so-called "whitelist" (the opposite of a blacklist, which contains all prohibited servers) was created, where access to all other connections was denied. This setting was active for 30 minutes before the experimenter collected the smartphone and locked it away.

Procedure

The within-subject design included one adaptation and three experimental nights in the sleep laboratory at the University of Fribourg. The adaptation night serves to get to know the complex setting in a sleep laboratory, to get used to sleep with electrodes, and to adapt to the situation of sleeping in a new environment. The procedure for all experimental conditions was identical until the instructions for the specific night were provided. Consequently, participants they were blind towards the condition until the very moment before sleeping (see Figure 1). The order of the conditions was randomly assigned and balanced between the participants.

Upon arrival at the sleep laboratory, participants gave written informed consent before handing in their smartphone to the experimenter and answering the pre-sleep questionnaires. Afterwards electrodes for polysomnography (PSG including electroencephalogram (EEG), electromyogram (EMG), electrooculogram (EOG), electrocardiogram (ECG)) were attached by the experimenter. As a first task, participants were instructed to complete a memory task where they learned 80 semantically associated word pairs (paired associated learning (PAL) task (Rasch et al., 2006)) before laying down in bed. Thereafter, they received the instructions for the night. In both the media condition and the relax condition, the last 30 minutes before sleep included the experimental manipulations described above. In the "media" condition, participants wore glasses which blocked blue light of 400-520 nm (uvex Winter Holding GmbH & Co. KG, Fürth) to control for any effects of blue light (Cajochen et al., 2006). They used their own smartphone to connect to a specially modified WLAN, modified to only allow them to use the services of WhatsApp (WhatsApp Inc., Menlo Park) and Snapchat (Snap Inc., Santa Monica), see above. No other server or service was reachable. Usage time was restricted to the last 30 min before sleep. The ambience light during this time was 62.3 – 68.9 lux (measured at the pillow). Before switching off the lights, the experimenter went back into the room and collected glasses and smartphone. In the "relax" condition, participants were instructed to not fall asleep while listening to the tape – approximately 30 minutes. Participants were lying in bed with the light on (light level at the source of light: 2000 – 3500 lux). Afterwards the experimenter went back into the room, turned off the speakers and the light. The participants were woken up 8 hours after lights off. Immediately after getting up, participants filled in the post-sleep questionnaires (assessing subjective sleep quality and mood), and

[5] The WLAN was created using a modem (TP-LINK Archer C9 AC 1900 Wireless Dual Band Gigabit Router) with a separate router (D-Link 4G LTE Router N300) that connected to the internet via the mobile network on 4G basis.

completed a psychomotor vigilance test (PVT), as well as the morning-recall of the PAL. The session ended with the removal of the PSG (see Figure 1).

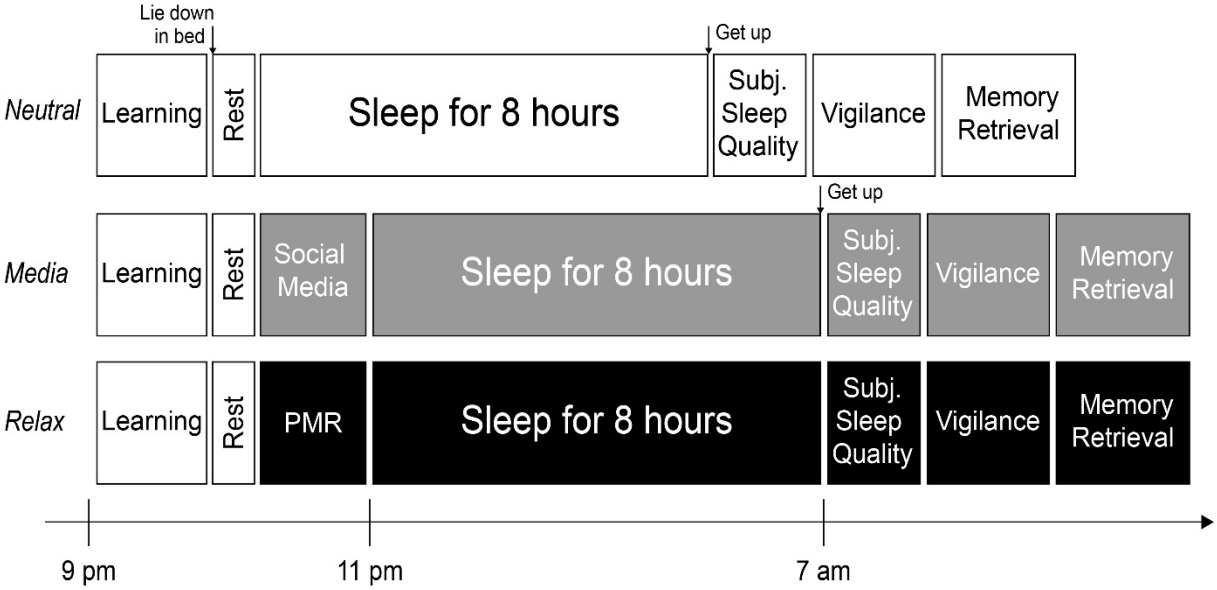

Figure 1. Procedure and experimental design. Participants spent one adaptation night and three experimental nights in the sleep lab, all four nights separated by one week. Directly before going to sleep and when lying in bed participants received their condition. In "media" participants were instructed to use WhatsApp (WhatsApp Inc. Menlo Park) and Snapchat (Snap Inc., Santa Monica) for 30 minutes. In "relax" participants listened to a tape with instructions to a PMR (progressive muscle relaxation) training for 30 minutes. In "neutral", participants went to bed directly after a 10-15 min rest and were allowed to sleep straight away. Sleep was recorded using PSG. Before the instructions for the conditions, participants filled out questionnaires, performed a word-pair learning (PAL) task (Rasch et al., 2006) and while already lying in the bed they rested for 10-15 min to calibrate the PSG. In the morning after sleep participants filled out questionnaires, performed a psycho-vigilance task (PVT) and retrieved word-pairs learned before sleep.

Participants

Thirty young and healthy students (21 women) with a mean age of 22.5 ± 3.0 [SD] completed the study. They were recruited through an advertisement and newsletter for students. Participants were regular users of social media (the recruitment process required them to be users of WhatsApp (WhatsApp Inc. Menlo Park), Snapchat (Snap Inc., Santa Monica), or both services). Participants were required to have a regular day-night rhythm, i.e., not working night shifts and a regular sleep rhythm. Furthermore, they were screened for extreme chronotypes of morningness or eveningness. Based on the German translations of the Morningness-Eveningness Questionnaire (D-MEQ (Griefahn et al., 2001)) six participants were classified as moderate morningness-types and two were moderate eveningness-types, which were included in the dataset. None of the participants reported to suffer from any known sleep disorders. The overall reported subjective sleep quality (Pittsburgh Sleep Quality Index (PSQI, Buysse et al., 1989) was M = 5.8 ± 1.4 [SD]; scale range: 3 (min) to 9 (max)). They were also instructed to abstain from alcoholic and caffeinated beverages within 48 hours prior to participation. After

completing the study, participants either received course credits or financial compensation of at least 200 CHF. In case of an early drop out compensation was provided proportionately. All procedures throughout the study followed the ethical recommendations of the Declaration of Helsinki. In addition, the study was approved by the local ethics committee of the University of Fribourg.

Measurements

Polysomnographic recordings

EEG, EMG, EOG and ECG were used to measure sleep as it is suggested by the American Association for Sleep Medicine (AASM; see Iber et al., 2007 for more information). EEG was recorded using 10 single gold-cap-electrodes following the 10-20-EEG-system with a sampling rate of 500 Hz. No participant complained about the electrodes neither for the nights nor for the pre-sleep examination. Physiological parameters were recorded using Brain Vision Recorder (Brain Products GmbH, Gilching). Impedances were kept below 5 kΩ. Following the AASM Manual for the Scoring of Sleep and Associated Events (short AASM guidelines, (Iber et al., 2007)), electrodes were referenced against Cz during recording and then re-referenced to the mastoids when conducting offline analyses. Data were preprocessed with Brain Vision Analyzer 2.0 (Brain Products GmbH, Gilching), filtering the data by 0.3 – 35 Hz following the guidelines suggested by the American Association of Sleep (Iber et al., 2007). Two trained sleep scorers visually scored sleep independently (interrater variability of Cohen's $\kappa = 0.83$) in 30s periods based on derivations F4, C4, O4, EOG (left and right), and EMG. Stages N1–N3, REM sleep, and WASO were scored following the AASM-guidelines (Iber et al., 2007). The scorers were blind to conditions. Sleep efficiency (SE) figured as objective sleep quality. SE is based on polysomnographic recordings and is the calculated ratio of TST to TIB (time in bed from lights off to lights on).

Questionnaires

General information (T1, evening): A self-created questionnaire was used to qualitatively assess further information regarding participants general health, sleep and sleep habits, or experience in sleep laboratories.

Handedness (T1, evening): The German version of the Edinburgh Inventory for Handedness (Oldfield, 1971; 10 items with 3 response options (left, right, mixed)) was used as a measure for participants' dominant hand to provide further information about the distribution within the sample (93.1 % right, 3.4% left, and 3.4% mixed handed).

General sleep quality (T1, evening): Pittsburgh Sleep Questionnaire Index (PSQI; Buysse et al., 1989; 19 items (each weighted on a 0–3 interval scale; core ranging from 0 to 21) was used to asses participants subjective sleep quality for the last month ($M = 5.8$, $SD = 1.4$; scale range: 3 (min) to 9 (max)), with lower scores denoting a healthier sleep quality.

Chronotype (T1, evening): The German version of the Morning-Evening-Questionnaire (D-MEQ; Griefahn et al., 2001; 19 items, scale ranging 14 to 86) was used to exclude extreme chronotypes (< 13 = clear night owls and > 70 = clear early birds) in the sample. In our sample were 2 moderate evening-types (M = 39, SD = 0), 22 neutral-types (M = 49, SD = 3.81), and 6 moderate morning-types (M = 61, SD = 1.67).

Mood (T1-4, evening and morning): To assess state mood before sleep as well as in the morning, and to look for any effects of social media / sleep quality on mood, participants filled in the multi-dimensional mood questionnaire (MDBF; Steyer et al., 1997; 12 items, each weighted on a 1–5 interval scale). The MDBF score is divided in three subscales (Good-Bad, Awake-Tired, and Calm-Nervous (= Subjective arousal) with higher scores denote better mood/ wakefulness/ calmness.

General health and drug use (T2-4, evening): In every experimental session, participants filled in a (self-created, qualitative) questionnaire about actual health problems (e.g. participants having a cold or headache), control for alcohol, and caffeine, drug, or nicotine abuse during the study. Regarding the answers, participants with drug or alcohol abuse to exclude participants.

Subjective sleep quality (T1-4, morning): In the morning, participants were asked to rate their subjective sleep quality [SF-A-R; 50; 25 items; scale range is 0 to 21; cut-off < 6] . Questions 2, 9, and 23 were used to analyze subjective SOL, WASO, and sleep depth.

Social media consumption and smartphone use (T2/3/4, morning): In the "social media" condition, participants filled in a created questionnaire (some items adapted to Ko et al., 2005) to qualitatively asses their smartphone use and social media consumption. This provided further information about participants' smartphone habits, the apps they use the most, and the social media providers they follow.

Experience with relaxation techniques (T2/3/4, morning): In the "relax" condition, participants filled in a created questionnaire to qualitatively assess previous experience with relaxation techniques (e.g. PMR, meditation, hypnosis), which would be used as a control if participants regularly practice any relaxation technique (which was not the case in this sample).

About the experiment (T4, morning): A created post-experiment questionnaire was used to qualitatively assess further information regarding participants hypothesis of the experiment, satisfaction with the setting and the experimental situation in the sleep laboratory, open questions, or any remarks for the experiment.

Memory measurement

Episodic memory was assessed with a paired-associated learning (PAL) task (Rasch et al., 2006). To avoid a ceiling effect, we expanded the list from Rasch and colleagues to 80 semantically related word-pairs (Feld et al., 2016). Each trial started with the presentation of all word-pairs, where each pair was presented for 3000 ms, followed by a 500 ms blank interval to separate two word-pairs. The words were presented in black font on a white screen via E-Prime (Psychology Software Tools,

Pittsburgh). Each pair was presented only once while the order was kept constant across subjects. Immediately after learning, participants were confronted with a cued recall test. Here, they had to complete the word pairs by typing in the corresponding second word. During the recall test, the cue word was presented for an infinite amount of time, or until the participant pressed enter. The correct answer was then presented for 1000 ms followed by a 500 ms blank interval which, again, separated the single trials. A second cued recall test followed in which the participants did not receive feedback; right after filling in the answer, a 500 ms blank interval followed before the next cue word was displayed. During recall the order of the word pairs differed from the learning phase but was kept constant across subjects. A third recall test took place in the morning. Performance was measured as the percentage of words recalled in the morning relative to the number of words remembered in the second recall phase.

Psychomotor Vigilance Test (PVT)

In the morning, participants performed a psychomotor vigilance test (PVT) designed to measure the effect of sleepiness on vigilance (Dinges & Powell, 1985). Subjects were asked to press the space key with their non-dominant hand as soon as they recognized the millisecond counter on the screen, which appeared at random intervals. After the keypress, the reaction time in milliseconds was shown for 1 s. Performance was measured as reaction time (RT) in milliseconds (ms), numbers of reactions, and numbers of errors.

Analytical procedures

Analysis of the EEG data and power analysis

For the EEG, the mean power was calculated for different frequency bands. This power analysis provides additional information to the sleep stages and allows a more nuanced analysis of sleep. Therefore, we used the SleepTrip toolbox (Weber, 2019; github.com/Frederik-D-Weber/sleeptrip) for Matlab (Mathworks, Natick). SleepTrip is a branch of FieldTrip with added functions for sleep analysis to provide a Matlab-based analysis of different frequency bands relevant for sleep (for further information and validation see Weber, 2019). Data were preprocessed semi-automatically with Brain Vision Analyzer 2.0 (Brain Products GmbH, Gilching) before spectral analysis. We calculated the average power of oscillatory activity in different frequency bands: slow-wave activity (SWA) (0.5 – 4.5 Hz), slow spindles (11-13 Hz) and fast spindles (13-15 Hz). Data from lights off in the evening to lights on in the morning were analyzed, segmented for NREM sleep (N2 and N3 sleep) and REM sleep.

Analysis of sleep cycles

Sleep follows a cyclic pattern (Steiger, 2010), which can be analyzed in a cycle analysis. This cycle analysis was performed following the criteria of Feinberg and Floyd (1979). These criteria define that a NREM period (NREMP) starts with N1 and has a minimum duration of 15 min. NREMP with a duration longer than 120 min excluding waketime can be split in two parts. These periods can include

waketime but no epochs of REM sleep. An epoch of REM sleep (REMP) is defined to start after a NREMP of 5 min. One exception is the first REMP, as its duration can be less then 5 min. The first cycle includes the first NREMP and the first REMP. A new cycle starts with the first N3 episode following a phase of more than 12 min with any other stage other than N3 (Jenni & Carskadon, 2004; Kurth et al., 2010; Rudzik et al., 2020). For each cycle, we calculated the amount of N1, N2, N3, REM, and WASO as well as the duration for each cycle. In addition, oscillatory power in the slow-wave and spindle bands across sleep cycles was used as a fine-grained measure.

Analysis of ECG

ECG (electrocardiogram) analysis was carried out using Kubios HRV Premium 3.2.0 (Kubios Oy, Kuopio). The ECG signal was segmented in resting-states (RS) of approximately two minutes (see e.g. Abdullah & Cvetkovic, 2014). A first RS was taken while participants were in bed during the calibration of the polysomnographic recording, a second one was taken after the calibration. In the experimental conditions, a third resting state was taken 5 minutes before the condition ended. Additionally, we segmented an RS while participants fell asleep (the first 2 minutes after turning off the light) and for pre-sleep (lights off to the first stage of N1). We exported all segments in EDF+-format using BrainVisionAnalyzer Version 2.0 (Brain Products GmbH, Gilching) and analyzed them with Kubios HRV Premium (Kubios Oy, Kuopio). This software offers an automatic artifact correction based on RR series (the interval between two R-signals) to eliminate ectopic beats and artifacts in unfiltered data (Tarvainen et al., 2019). Afterwards, data were analyzed in a time and frequency domain and subsequently used for calculating the mean heart rate (mean HR measured in beats per minutes) which served as an index for physiological arousal (Kogler et al., 2015). As a second index of physiological arousal, Kubios provides an index to analyze the heart rate variability (HRV Triangular Index), which is based on the RR interval (Tarvainen et al., 2019). One participant was excluded from analysis due to insufficient ECG data quality throughout one of the nights.

Statistical analyses

According to the pre-sleep experimental manipulation (see section 2.1), the main outcome variables for the analyses were subjective and objective sleep quality, SOL, WASO, and sleep depth. For each of these main outcome variables, a repeated measures analysis of variance (ANOVA) with the within-subject factor condition (“neutral”, “media”, and “relax”) and a contrast of no-pre-sleep manipulation (“neutral”) vs. pre-sleep manipulation (“media” and “relax”) was calculated.

In a separate analysis, we used a 2x3 ANOVA with the within-subject factors “type of parameter” (subjective vs. objective) and condition (“neutral”, “media”, and “relax”) for the four main outcome variables. In addition, we reported Pearson’s correlation coefficient (r) for the strength of association between these objective and subjective sleep parameters.

In exploratory analyses we examined further sleep parameters, vigilance, sleep-associated memory consolidation, and heart rate variability (HRV) by means of repeated measures ANOVAs with condition ("neutral", "media", "relax") as within-subject factor. For oscillatory power during NREM and REM sleep, a 3x2x3 repeated measurements ANOVA with factors "condition" ("neutral", "media", "relax), "hemisphere" (left, right) and "topography" (frontal, central, parietal) was conducted. A Pearson's correlation coefficient (r) was calculated to indicate the strength of association between sleep parameters and vigilance.

In case of a significant main or interaction effect, post-hoc paired t-tests with a Bonferroni correction were computed; level of significance $p \leq 0.05$, effect sizes (□2) were only reported for significant data. Data analysis was carried out using IBM SPSS Statistics 25 (IBM Corp., Armonk) and R Studio (RStudio Team, Boston). Results are presented as means □ standard errors of the mean (SEM).

Data availability

Datasets analyzed during the current study are available online on https://osf.io/z9af2/.

Results

Subjective sleep parameters

Contrarily to our expectations, consuming social media before sleep did not affect subjective sleep quality (measured by the SFAR questionnaire (Görtelmeyer, 2011)). Participants reported similar subjective sleep quality (on a scale from 7 up to 35, with higher value indicating better sleep quality) in the neutral (25.17 ± 0.54) compared to the "media" condition (24.88 ± 0.58, $p > 0.90$, for a post hoc pairwise comparison, see Figure 2a). However, participants indicated better subjective sleep quality in the "relax" compared with the other two conditions (26.80 ± 0.44, both $p < 0.001$ for post hoc tests, main effect of condition: $F(2, 58) = 7.186$, $p = 0.002$, $\eta^2 = 0.199$). Reports of subjective sleep onset latency (SOL) did not differ in the neutral (16.50 ± 2.89 min) and "media" conditions (15.33 ± 2.35 min, $p > 0.90$, see Figure 2b). Again, performing PMR improved SOL to only 5.0 ± 1.34 min compared to the other two conditions (both $p < 0.01$). The main effect of condition was significant; $F(2, 58) = 9.157$, $p < 0.001$, $\eta^2 = 0.240$). Results for subjective sleep depth showed the same pattern, with comparable ratings for neutral and "media" conditions, the deepest sleep ratings occurring in the "relax" sessions (Figure 2c). Other subjective sleep parameters were not affected by the experimental manipulation (see Table 1).

Interestingly, participants also reported to feel more awake after sleep in the "relax" condition (3.28 ± 0.14, scale from 1 (not at all awake) to 5 (very much awake), as measured by the multi-dimensional mood questionnaire (MDBF) (Steyer et al., 1997). Ratings in the "neutral" (3.02 ± 0.15) and "media" conditions (3.03 ± 0.14) did not differ ($F(2, 58) = 3.28$, $p = 0.045$, $\eta^2 = 0.10$), see Figure

2d. The remaining two subscales of the measure (good-bad and calm-nervous) did not differ, neither between conditions nor for time of measurement (all $p > 0.14$, see Table 1).

Table 1. Sleep parameters for the three conditions (subjective and objective)

	Neutral	**Media**	**Relax**		
	M ± SEM	M ± SEM	M ± SEM	F-Test	p-Value
Subjective Sleep Parameters					
Sleep Quality	25.17 ± 0.54	24.88 ± 0.58	26.80 ± 0.44	7.186	**0.002****
SOL [min]	16.50 ± 2.89	15.33 ± 2.35	5.0 ± 1.34	9.157	**< 0.001****
WASO [min]	8.48 ± 1.62	10.63 ± 4.64	11.53 ± 2.52	0.284	0.75
Sleep Depth	3.97 ± 0.14	3.77 ± 0.16	4.20 ± 0.13	3.167	**0.05***
Objective Sleep Parameters					
Sleep Efficiency [%]	96.70 ± 0.52	95.71 ± 0.83	98.54 ± 0.45	6.738	**0.002****
SOL [min]	14.78 ± 2.11	19.23 ± 3.59	6.50 ± 2.16	7.095	**0.002****
WASO [min]	14.62 ± 3.44	20.82 ± 5.49	18.10 ± 4.54	0.61	0.48
Sleep Depth [SWA in Hz]	30.76 ± 4.62	28.01 ± 4.54	32.11 ± 5.55	1.79	0.14
N1 [min]	30.18 ± 1.59	32.80 ± 2.11	28.92 ± 1.52	3.099	0.05
N2 [min]	218.97 ± 5.30	199.02 ± 6.71	220.18 ± 5.06	8.297	**0.001****
N3 [min]	101.60 ± 5.08	102.32 ± 5.17	100.75 ± 4.92	0.088	0.92
REM [min]	90.23 ± 2.81	94.72 ± 3.63	98.40 ± 3.80	2.258	0.11
Move [min]	8.27 ± 0.65	8.05 ± 0.67	8.27 ± 0.55	0.116	0.89
N1 [%]	6.51 ± 0.34	7.19 ± 0.47	6.11 ± 0.33	4.507	**0.015****
N2 [%]	47.19 ± 1.09	43.37 ± 1.33	46.40 ± 1.04	5.935	**0.005****
N3 [%]	21.92 ± 1.11	22.46 ± 1.20	21.21 ± 1.02	1.173	0.32
REM [%]	19.46 ± .59	20.65 ± 0.75	20.70 ± 0.77	1.419	0.25
Move [%]	1.79 ± 0.14	1.76 ± 0.14	1.75 ± 0.12	0.068	0.94
WASO [%]	3.14 ± 0.74	4.56 ± 1.20	3.83 ± 0.97	1.711	0.50
TST [min]	463.87 ± 2.53	457.72 ± 4.04	474.62 ± 2.68	8.911	**< 0.001****
SWS Latency [min]	12.12 ± 0.74	13.78 ± 0.86	10.33 ± 1.06	5.57	**0.006****
REM Latency [min]	93.00 ± 5.82	85.58 ± 6.00	78.67 ± 6.55	1.549	0.22
Sleep Cycles					
Number of Cycle	5.14 ± 0.11	4.89 ± 0.11	5.15 ± 0.14	1.66	0.20
Mean Cycle Duration [min]	91.78 ± 6.30	95.09 ± 5.60	93.96 ± 5.77	0.63	0.54
Cycle 1 (n = 30)	91.33 ± 4.20	87.20 ± 4.29	82.25 ± 4.39	1.41	0.25
Mean Duration: Cycle 2 (n = 30)	88.25 ± 4.31	94.50 ± 4.84	105.17 ± 6.65	3.57	**0.034***
Mean Duration: Cycle 3 (n = 30)	100.43 ± 6.16	107.33 ± 4.09	108.82 ± 4.34	0.91	0.41
Mean Duration: Cycle 4 (n = 29)	113.05 ± 7.40	113.286.27	101.294.63	1.26	0.29
Mean Duration: Cycle 5 (n = 15)	110.9 ± 10.62	110.57 ± 6.82	104.70 ± 4.97	0.25	0.71

Heart Rate Variability (HRV)					
Mean HR [bpm]	67.84 ± 10.90	68.37 ± 12.32	63.62 ± 11.55	5.99	**0.004****
HRV Triangular index	13.20 ± 1.94	13.61 ± 2.16	13.22 ± 18.83	0.17	0.85
Memory					
Encoding (evening)	53.83 ± 2.38	53.66 ± 2.32	53.72 ± 2.78	0.005	0.99
Recall (morning)	53.21 ± 2.45	52.51 ± 2.60	51.35 ± 2.78	0.511	0.60
Consolidation [%]	98.93 ± 1.76	97.02 ± 1.63	96.49 ± 2.30	0.468	0.63
Vigilance (Morning)					
Recaction Time (RT)	338.24 ± 6.77	340.64 ± 8.04	340.24 ± 7.80	0.061	0.94
Reactions	77.00 ± 0.42	76.47 ± 0.46	76.60 ± 0.32	0.64	0.38
Errors	3.53 ± 2.08	4.63 ± 1.92	5.97 ± 3.76	0.793	0.43
Mood					
Good-Bad, Evening	4.33 ± 0.11	4.33 ± 0.11	4.27 ± 0.10	0.14	0.87
Good-Bad, Morning	4.21 ± 0.12	4.05 ± 0.11	4.16 ± 0.13	0.99	0.38
Awake-Tired, Evening	3.15 ± 0.16	3.11 ± 0.15	3.18 ± 0.16	0.09	0.88
Awake-Tired, Morning	3.02 ± 0.15	3.03 ± 0.14	3.28 ± 0.14	3.28	**0.045***
Arousal (Calm-Nervous), Evening	3.83 ± 0.14	4.06 ± 0.11	3.87 ± 0.13	1.47	0.24
Arousal (Calm-Nervous), Morning	3.92 ± 0.12	3.78 ± 0.13	3.90 ± 0.14	0.56	0.58
	M ± SEM	M ± SEM	M ± SEM	F-Test	p-Value
	Neutral	**Media**	**Relax**		

Notes: Subjective parameters are based on subjective ratings in the SF-A-R (Görtelmeyer, 2011). Objective values are based on polysomnographic recordings. Non-rapid eye movement (NREM)-sleep, stage 1, 2, and 3 sleep (N1, N2, N3), rapid eye movement sleep (REM), waketime after sleep onset (WASO), total sleep time (TST), sleep onset latency (SOL), slow wave sleep latency (SWS latency), REM sleep latency (REM latency) are all measured in minutes [min] and the percentages indicate parietal percentage of TST [%]. Sleep cycles were calculated using the criteria of Feinberg and Floyd (1979). The numbers of participants (n) differed between the cycles as not all participants reached the 4^{th} and 5^{th} cycle. Heart rate (HR, in beats per minute (bpm)) and heart rate variability (HRV) indexes (provided by Kubios (Kubios Oy, Kuopio) were used as an objective measurement of arousal. For memory, numbers indicate absolute or relative values of correctly recalled words that were presented in the evening (learning phase with first recall) and in the morning (retrieval phase with second recall). Consolidation refers to the difference in performance between learning and retrieval phases. For vigilance, the reaction time (RT), the number of reactions, and number of errors during the 10 minutes of the psychomotor vigilance task (PVT) were measured. Mood parameters are based on subjective ratings in the mood questionnaire with the three subscales (Good-Bad, Awake-Tired, and Calm-Nervous) (Steyer et al., 1997). Calm-Nervous indicates the subjective arousal. Values are means (M) ± standard error of mean (SEM). * indicates $p \leq 0.05$. ** indicates $p \leq 0.01$. *** indicates $p \leq 0.001$. Significant results are highlighted in bold.

Objective sleep parameters

The results concerning objective sleep parameters confirm findings the findings reported above. Sleep efficiency was generally high and did not differ between "media" (95.71 ± 0.83 %) and "neutral" (96.70 ± 0.52 %, $p > 0.60$) conditions. However, sleep efficiency significantly improved in the "relax" condition (98.54 ± 0.45 %, both $p > 0.016$, main effect condition $F(2,58) = 6.738$, $p = 0.002$, $\eta^2 = 0.189$, see Figure 2e). Similarly, objective time to fall asleep (SOL) did not differ between "media" (19.23 ± 3.59 min) and neutral conditions (14.78 ± 2.11 min; $p > 0.40$), but was significantly shortened in the "relax" condition to only 6.50 ± 2.16 min (both $p < 0.03$, main effect condition: $F(2,58) = 7.095$, $p = 0.002$, $\eta^2 = 0.197$, see Figure 2f). Sleep depth did not differ between conditions ($F(2, 58) = 0.61$, $p = 0.484$, see Figure 2g).

The sole indications for an influence of media use before sleep can be found in individuals' sleep architecture. As total sleep time (TST) in minutes differed significantly between the conditions ($F(2,58) = 8.911$, $p < 0.001$, $\eta^2 = 0.235$), percentages of sleep stages were analyzed instead. Participants slept less after media consumption (457.72 ± 4.04 min, $p = 0.005$) compared to "relax" (474.62 ± 2.68 min, $p = 0.005$) and "neutral" (463.87 ± 2.53 min, $p = 0.199$) conditions, which both differed significantly as well ($p = 0.01$). The relative amount of N1 sleep in the "media" condition (7.19 ± 0.47 %) did not differ compared to the neutral condition (6.51 ± 0.34 %, $p = 0.18$) but decreased after "relax" (6.11 ± 0.33 %, $p = 0.044$, main effect of condition: $F(2,58) = 4.507$, $p = 0.015$, $\eta^2 = 0.135$, see Figure 2i). The relative amount of N2 sleep (43.37 ± 1.33%) after media use was significantly reduced compared to the neutral condition (47.19 ± 1.09 %, $p = 0.008$). However, we did not find differences compared to the "relax" condition (46.40 ± 1.04 %, $p = 0.12$, main effect of condition: $F(2,58) = 5.935$, $p = 0.005$, $\eta^2 = 0.170$, see Figure 2j). The time spent in N3 (SWS) was not affected by social media consumption before sleep and did not differ between the three conditions (main effect condition: $F(2,58) = 1.173$, $p = 0.317$, see Figure 2k). In addition, participants reached SWS earlier when they had performed PMR before sleep (10.33 ± 1.06 min) compared to the "media" condition (13.78 ± 0.86 min, $p = 0.009$), but not compared to the neutral condition (12.12 ± 0.74 min, $p > 0.19$, main effect of condition $F(2,58) = 5.570$, $p = 0.006$, $\eta^2 = 0.161$, see Table 1 and Figure 2h). Time spent awake during the night (WASO) or in the other sleep stages was comparable across all conditions (all $p > 0.11$, see Table 1).

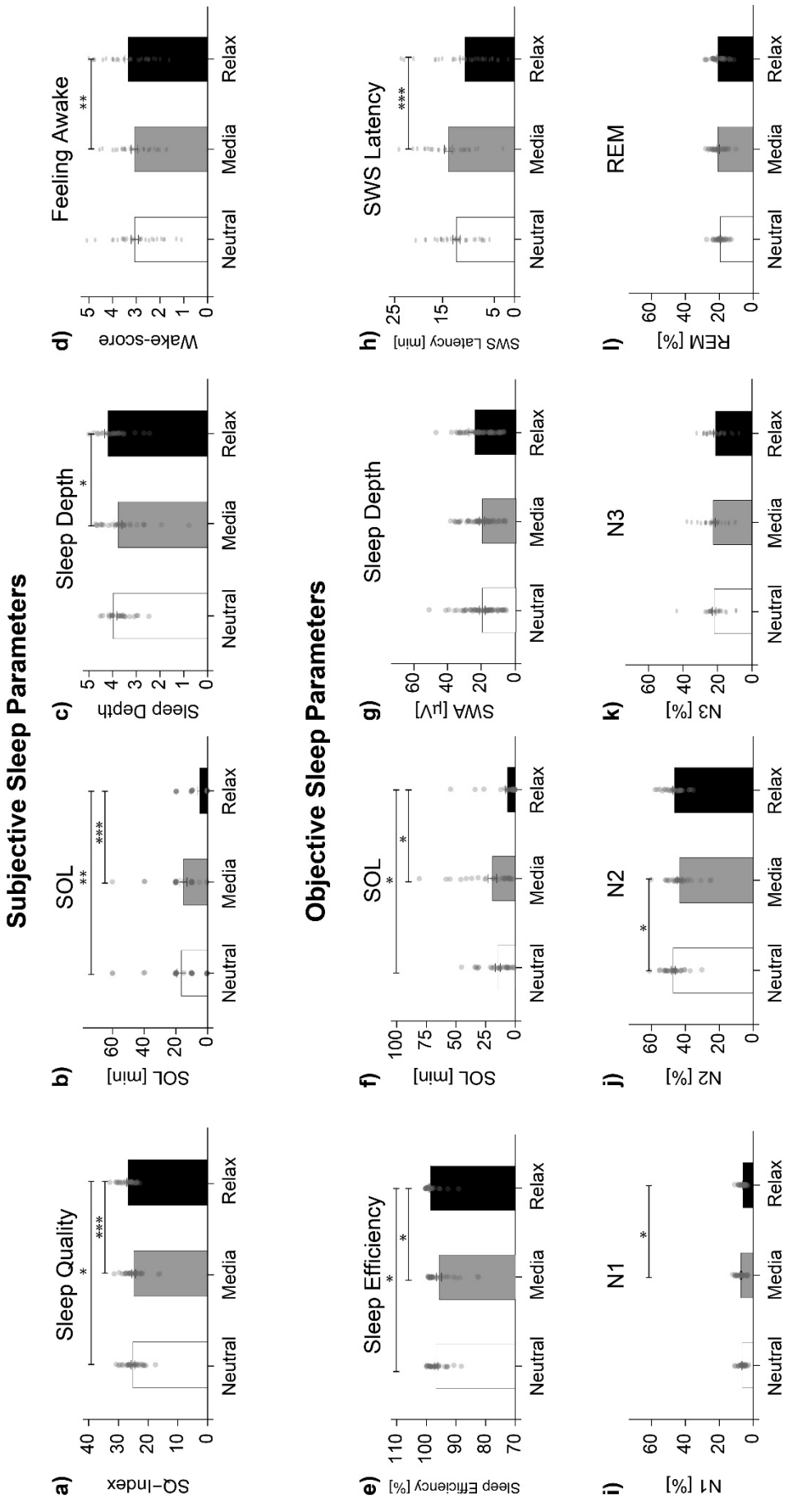

Figure 2. Subjective and objective sleep parameters. On a subjective level, participants reported to sleep worse (sleep quality (a)) and less deep (c) after using social media (grey bar) compared to "relax" (black bar). For sleep onset latency (SOL (b)), participants showed significant differences between "media" and "relax" and between "relax" and "neutral". Participants felt significantly more rested in the morning after the "relax" condition compared to "media" (d). On an objective level, we showed an effect of sleep quality (sleep efficiency (e)) as after social media consumption the sleep efficiency was the lowest and differed significantly to "relax". Furthermore, sleep efficiency was the highest in "relax" and differed significantly to "neutral". Both results for SOL shown in the subjective parameters we confirmed in our data objectively (f). Sleep depth (slow wave activity (SWA) (g)) confirmed the subject results as well as there was no difference between the three conditions. Additionally the different sleep stages were analyzed (i-l). N1 (i) showed significant difference between the "media" and "relax" condition with less N1 (as percentage of total sleep time, TST) in "relax". N2 (j) showed a significant reduction of N2 after social media consumption compared to the "neutral" condition. N3 (k) and REM (l) showed any significant changes. Means ± standard errors of the mean are indicated. Significant pair-wise comparisons from post-hoc tests are indicated by *: $p \leq 0.05$. **: $p \leq 0.01$. ***: $p \leq 0.001$.

In addition to sleep architecture, we investigated the dynamics of sleep using a power analysis for frontal slow-wave activity (SWA; 0.5 – 4.5 Hz), frontal slow sleep spindles (11 – 13 Hz) and parietal fast sleep spindles (13 – 15 Hz). The analysis was conducted with the additional factor of sleep cycle using Feinberg and Floyd's (1979) criteria. For SWA power, we observed the typical decrease in power over the cycles ($F(3, 84) = 41.75$, $p < 0.001$, $\eta^2 = 0.60$, see Figure 3a). However, using neither social media nor PMR had an effect on SWA power over all cycles ($p > 0.58$). We also did not observe any effects of the experimental manipulation on power in the slow or fast spindle bands (all $p > 0.30$). Both, slow spindle power ($F(3, 84) = 4.73$, $p = 0.024$, $\eta^2 = 0.024$) and fast spindles ($F(3, 84) = 7.85$, $p = 0.006$, $\eta^2 = 0.22$) generally differed between sleep cycles (see Figure 3b-c).

Generally, the three conditions did not differ in average number of sleep cycles ("neutral": 5.14 ± 0.11; "media": 4.89 ± 0.11, and "relax" 5.15 ± 0.14 cycles, respectively, $p = 0.198$). Mean cycle length was also comparable ("neutral": 91.78 ± 6.30; "media": 95.09 ± 5.60; "relax": 93.96 ± 5.77, $p = 0.538$).

Figure 3. Sleep dynamics for slow-wave-activity, fast and slow spindles.

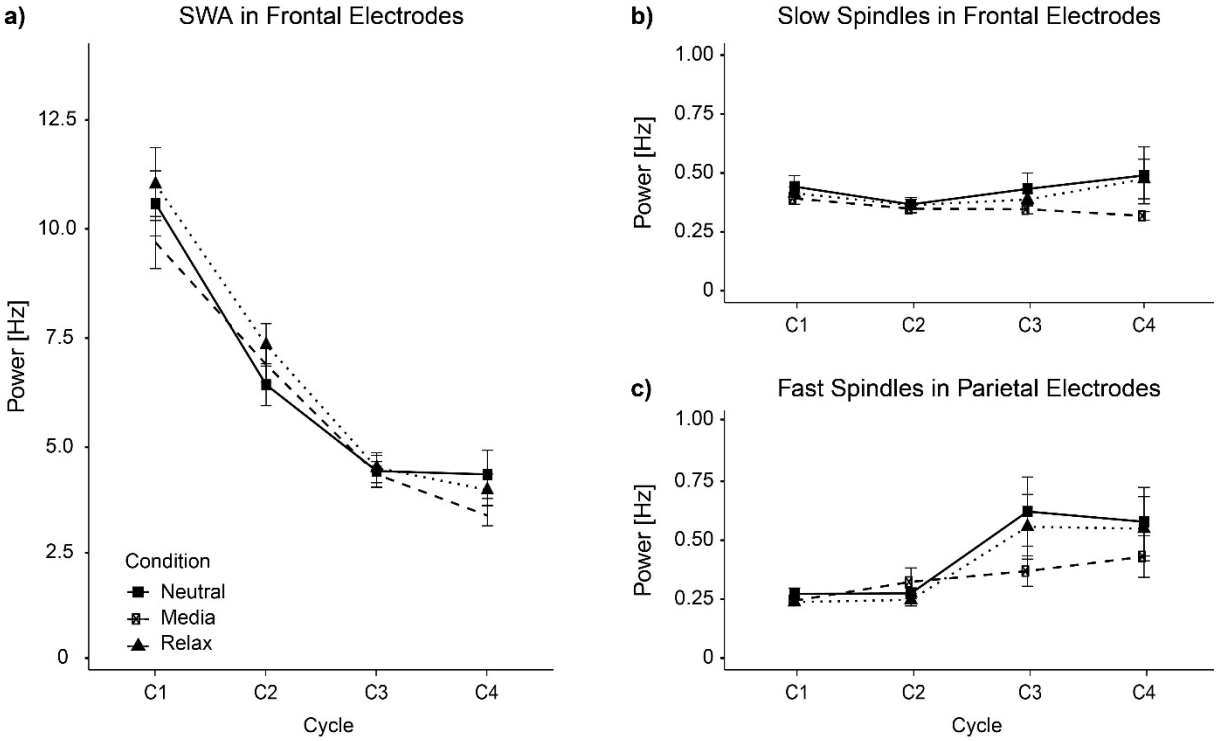

Figure 3. Sleep dynamics for slow-wave-activity, fast and slow spindles. Power analysis in the first four cycles (C1 – C4) for slow-wave activity (SWA; 0.5 – 4.5 Hz) measured in frontal electrodes (F3 and F4) (a), slow spindles (11-13 Hz) measured in F3 and F4 (b), and fast spindles (13-15 Hz) measured in parietal electrodes (P3 and P4) (c). No significant effect on condition was found in any frequency band.

Physiological arousal

To identify the role of pre-sleep physiological arousal on subsequent changes in sleep, we analyzed the effect of social media use on heart rate and heart rate variability during the period of falling asleep (2 minutes after lights off) compared to the "neutral" and "relax" conditions. We expected social media consumption to increase pre-sleep arousal. Contrary to our expectations, using social media before sleep did not significantly impact heart rate (68.37 ± 12.32 bpm) as compared to "neutral" (67.84 ± 10.90 bpm; $p > 0.72$). In contrast, PMR decreased heart rate significantly (63.62 ± 11.55 bpm) compared to the two other conditions (both $p < 0.008$, main effect of condition: $F(2, 58) = 5.99$, $p = 0.004$, $\eta^2 = 0.17$, see Figure 4a). Heart rate variability (HRV triangular index) did not significantly differ between the experimental conditions; $F(2, 58) = 0.17$, $p = 0.846$, for the HRV triangular index provided by Kubios (Kubios Oy, Kuopio). For all results, see Table 1.

To test whether the differences between the experimental conditions in HR and HRV predicted the differences in sleep parameters, an explorative correlational analysis was computed. To control for general individual differences, values were corrected for the neutral condition, which resulted in ΔMedia ("media" – "neutral") and ΔRelax ("relax" – "neutral"), both for pre-sleep arousal and sleep parameters. For the "media" condition, changes in pre-sleep arousal after social media use (ΔMedia) was not related to any differences in objective sleep parameters. Particularly, the reduction in N2 sleep was not correlated with pre-sleep changes in HR or HRV (Mean HR: ΔMedia: $r = -0.0804$, ΔRelax: $r = 0.207$ and HRV Triangular Index: ΔMedia: $r = -0.082$, ΔRelax: $r = -0.002$). Results for the remaining objective sleep parameters show the same pattern (see Table 2 and Figure 4b for REM sleep). For subjective sleep evaluations, changes in heart rate tended to predict subjective sleep quality (Mean HR: ΔMedia: $r = -0.36$, $p = 0.060$), subjective SOL (Mean HR: ΔMedia: $r = 0.34$, $p = 0.074$) and subjective sleep depth (Mean HR: ΔMedia: $r = -0.36$, $p = 0.060$, see Table 2).

In the "relax" condition, a higher reduction in heart rate predicted an increase in REM sleep (relative to the neutral condition, $r = -0.563$, $p = 0.002$, see Figure 4c). The reduction in SOL, the early SWS latency, the increase in SWS or the increase in N1 sleep was not associated with the reduced pre-sleep arousal after performing PMR (all $r < 0.262$, all $p > 0.177$). HRV triangular index was not correlated with any sleep parameter (all $r < 0.356$, all $p > 0.06$). We did not find any significant correlations or subjective sleep parameters in the "relax" condition; neither for mean HR nor the HRV triangular index (see Table 2).

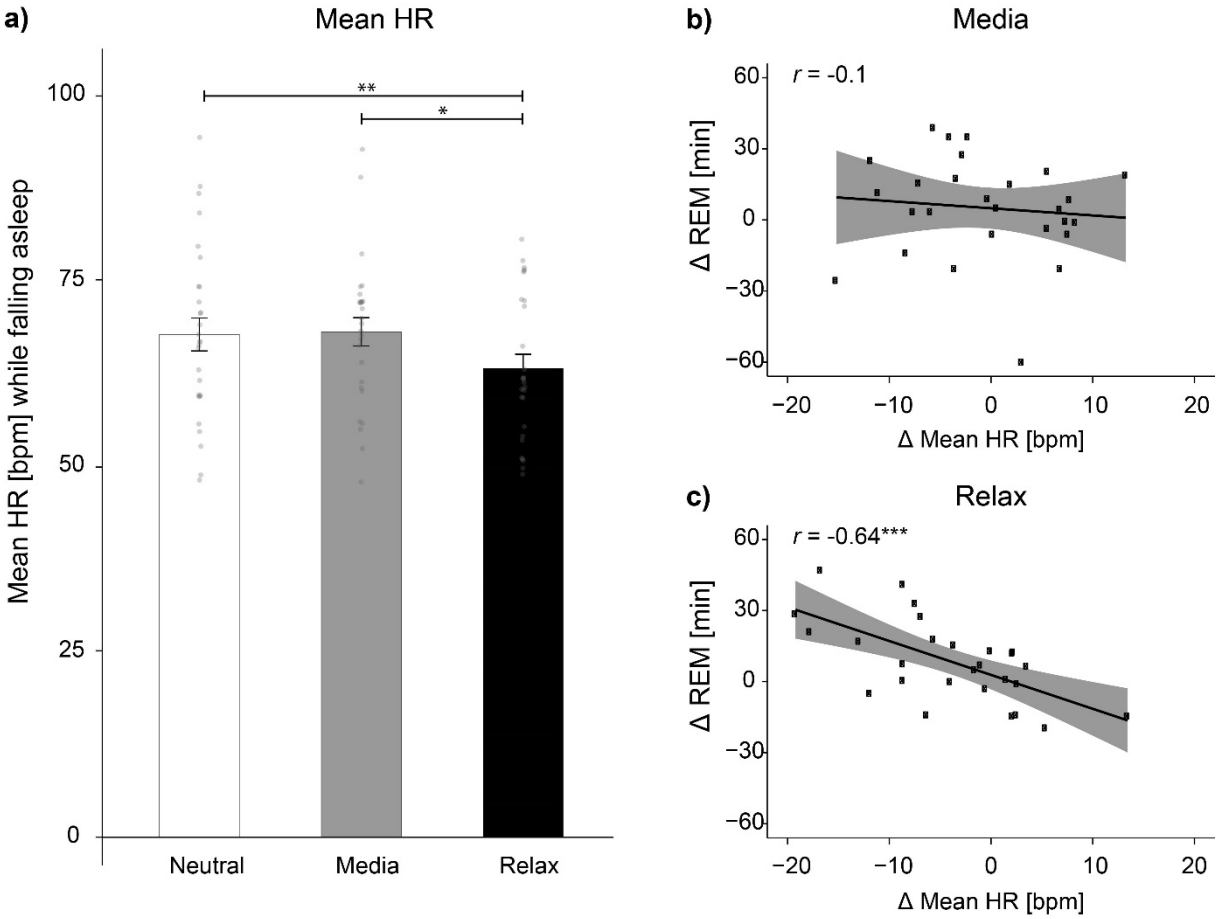

Figure 4. Effects of social media and PMR on pre-sleep arousal. Pre-sleep arousal was measured using mean heart rate (HR) in beats per minute (bpm) while participants fell asleep. (a) Social media consumption had no arousing effect of mean HR ($p > 0.72$), whereas progressive muscle relaxation (PMR) decreased mean HR compared to "media" ($p = 0.004$) and "neutral" ($p = 0.008$). Means ± standard errors of the mean are indicated. (b) In the "media" condition, the overall increase in arousal (mean HR) did not correlate with the amount of REM sleep (in min). Both values were corrected for "neutral" (ΔMean HR = Mean HR "media" – Mean HR "neutral" and ΔREM = REM "media" – REM "neutral"). (c) In the "relax" condition, the change in ΔMean HR ("relax" corrected for "neutral") correlated positively with the change in Δ REM sleep (in minutes and corrected for "neutral"). * indicates $p \leq 0.05$. ** indicates $p \leq 0.01$. *** indicates $p \leq 0.001$.

Table 2. Heart rate and heart rate variability correlated with sleep parameters

		Mean HR [bpm]		**HRV Triangular Index**	
		Δ Media	Δ Relax	Δ Media	Δ Relax
Subjective	Δ Sleep Quality	-0.360	0.148	0.056	-0.146
	Δ SOL	0.340	-0.037	-0.022	0.164
	Δ WASO	0.073	0.015	0.194	-0.152
	Δ Depth	-0.363	0.224	0.032	0.021
Objective	Δ Sleep Efficiency	-0.110	-0.184	-0.002	-0.102
	Δ SOL	0.096	0.197	-0.007	0.104
	Δ WASO	0.180	0.016	0.170	0.208
	Δ Depth / SWA	0.140	0.262	0.356	0.289
	Δ N1	0.058	0.161	-0.082	-0.026
	Δ N2	-0.080	0.207	-0.082	-0.002
	Δ N3 / SWS	-0.033	0.166	0.108	0.078
	Δ REM	-0.170	**-0.563****	-0.163	-0.316
	Δ TST	-0.015	-0.025	0.046	-0.120
	Δ SWS Latency	0.114	0.293	-0.121	-0.110
	Δ REM Latency	0.166	0.272	0.065	0.021

Notes: Correlations were calculated separately for the "media" and "relax" conditions. All parameters are corrected for the values obtained in the neutral condition (ΔMedia: "media" – "neutral" and ΔRelax: "relax" – "neutral"). Subjective parameters were measured by SF-A-R (Görtelmeyer, 2011): Sleep quality, sleep onset latency (SOL), wake time after sleep onset (WASO), numbers of awakenings (NWAK) and sleep depth. Objective sleep parameters are based on polysomnographic recordings (Sleep quality = sleep efficiency, SOL, WASO, NWAK, depth = sleep stage N3 in minutes). Significant correlations are highlighted in bold. ** indicates $p \leq 0.01$.

Effects on memory consolidation and vigilance

We did not observe any differences on neither sleep dependent memory consolidation in our experimental conditions ($p > 0.80$), nor on psychomotor vigilance tested after sleep ($p > 0.90$, see Table 1).

Discussion

This study investigated the effect of pre-sleep media use on subjective and objective sleep parameters in a controlled experimental setting, i.e., sleep laboratory. We assumed that pre-sleep social media use would impair sleep as indexed by subjective (self-report) and objective (arousal, sleep) indicators. To investigate these effects, a condition with social media use was compared to a neutral (no specific pre-sleep activity) and a pre-sleep relaxation (progressive muscle relaxation, PMR) condition. Contrarily to our assumptions, social media use did not impair subjective sleep parameters. This result contradicts findings from previous studies providing proof for the existence of such an adverse effect as indicated by self-reports about a decrease in sleep quality (Alonzo et al., 2021; Levenson et al., 2017; Li et al., 2015; Woods & Scott, 2016), shorter sleep duration (Chahal et al., 2013; Espinoza & Juvonen, 2011), prolonged sleep onset (Arora et al., 2014; Bartel et al., 2015), and more wake reactions (Arora et

al., 2014). Regarding objective indicators, we only found a reduction in the percentage of N2 sleep after social media use compared to the neutral night. This was, however, not at the expense of other sleep stages. Rather, this change seemed to be equally distributed across all sleep stages as it was accompanied by non-significant increases in WASO, N1, N3 and REM sleep. None of the other objective sleep measures were affected by social media use.

In going beyond existing studies relying on self-reports, this study took place in a sleep laboratory. This setting not only standardises measurements, but it also increases controllability of confounding factors, such as reachability via smartphones during the night. As smartphones were removed from the sleep cabin during the night, participants as well as their social contacts (peers, family, partners, etc.) with whom they regularly interact, were aware that they would not be reachable during the night. This is important because having a smartphone in the bedroom together and being potentially reachable throughout the night reduces subjective sleep quality (Klein Murdock et al., 2017). This finding is supported by another study conducted in our laboratory, in which we demonstrated that performing on-call duty during the night leads to differences in sleep quality. We showed that the instruction to be on-call, even only for one night, reduced sleep quality. Importantly, this effect is apparent even if no physical interruption occures during the night (Combertaldi, Wick, & Rasch, in preparation). Consequently and irrespective of its usage, the presence of a smartphones reduces sleep quality, TST and leads to daytime sleepiness (Carter et al., 2016).

By taking away our participants' smartphone for the night we aimed to minimize nocturnal disturbances. However, by taking into account findings on the so called "Fear of Missing Out" (FoMO; Vorderer et al., 2016), which is defined as the need to stay up to date and connected with others (Przybylski et al., 2013), we cannot be sure if this led to an increased arousal or discomfort for some of our participants. FoMO has been associated with a decrease total sleep time (TST) and delayed sleep onset (Conlin et al., 2016; Rogers & Barber, 2019; Scott & Woods, 2018; Tandon et al., 2020), probably after promoting cognitive pre-sleep arousal (Blake et al., 2017; Carnagey, Anderson, & Bartholow, 2007; Mauri et al., 2011; Scott & Woods, 2018). Following our goal to reduce potential and effective nocturnal disturbances, we might have introduced an unsystematic error.

We expected that disturbed sleep after social media use would be mediated by pre-sleep arousal. We found no disturbing effects on sleep, nor did media use increase subjective or objective pre-sleep arousal. Subjective ratings of calmness indicated that participants did not feel more aroused after social media use compared to directly going to sleep. Physiologically, neither heart rate (HR) nor heart rate variability (HRV) were affected by social media consumption. Even though the latter has been found to be a particularly sensitive marker for (psychosocial) stress (Brugnera et al., 2018; Camm et al., 1996; Taelman et al., 2009). Neither HR nor HRV correlated with the reported N2 reduction after social media use, rendering a contribution of pre-sleep arousal to the N2 differences unlikely. One explanation for the difference between this and previous studies might be the lack of blue-light. Human alertness is blue-light sensitive and heart rate increases with short wavelength light (Cajochen et al., 2005). Normally,

smartphone use is associated with exposure to blue-light which can delay sleep onset (Blume et al., 2019). In our study we used blue-light blocking glasses, which differs from everyday life and existing studies.

While we did not find detrimental effects of social media use – compared to the neutral night – progressive muscle relaxation (PMR) clearly had positive effects on pre-sleep arousal and sleep. After practicing PMR, heart rate (while falling asleep) and sleep onset latency were reduced. Moreover, PMR also improved subjective and objective sleep quality. Consequently, participants in the PMR condition felt more awake the next morning. Practicing PMR instead of using social media might be recommendable to improve sleep. This not only applies when compared to social media use but even when compared to a neutral condition. When compared to the other two conditions, PMR before sleep improved sleep quality, sleep efficiency, and SOL. Other indicators (e.g. reduction of SWS latency, reduction in N1 sleep etc.) were only found to differ between the PMR and the social media condition. Hence, these effects can only to a limited extent be interpreted as improvements resulting from PMR. As we tested a healthy sample of young adults, we cannot exclude potential ceiling effects. Regardless of these effects, our results are still in line with previous research and demonstrate why relaxation (e.g. PMR) is a frequently chosen treatment for insomnia (Mackereth & Tomlinson, 2010; Means et al., 2000; Morin et al., 1999; Walters et al., 2003). In our study, the positive effects of PMR on sleep not only demonstrate that pre-sleep arousal can influence sleep, but also provide evidence that the applied methods of measurements are well-suited and sufficiently sensitive to map these changes. Conversely, this also underpins the findings that the use of SM at bedtime had a neglectable influence on sleep parameters. One might still argue that changes in pre-sleep arousal or sleep might have been too fine grained for the applied measurement. However, any impairments would have also be likely to result in poorer daytime performance (Johansson et al., 2016). The non-impact of social media use on sleep is, thus, further supported by the fact, that neither morning indicators of vigilance nor memory differed between conditions; even though we cannot be absolutely sure that such effects could appear later in the day.

On a related note, this study's findings are limited to two specific ways of social media use, i.e., communication via WhatsApp and Snapchat. Even though these apps are widely used and we did require our participants to be active users of at least one of the two, other ways of communicating with peers before sleep as well as other forms of media use might produce different effects. One could assume that being able to communicate with others before sleep serves as a way of clearing one's mind; thereby working as a calming buffer. Contrarily, the use of news, entertaining, and stimulating audiovisual content might provide additional cognitive and physiological stimulation, leading to an increased arousal. Therefore, future studies should investigate and compare the effects of different ways of media use its interaction with the resulting arousal on sleep as well as on daytime alertness, preferably not only in the morning.

Limitations

In this study, the exposure to blue light was reduced, thereby attempting to isolate the effects of electronic social media use independent of nocturnal light exposure. While the use of blue-blocking glasses to exclude the effect of light is interesting and helps to distill more specifically the effects of social media use per se, another experimental condition with normal light (including short wavelengths) exposure would have been useful. Although the aim of this study was to observe effects of social media on sleep, the question of whether or not light from personal devices impairs sleep remains open for further research.

Due to the standardization of the type of use, real-time chatting was used as an active way of engaging in social media (WhatsApp chatting and Snapchat) rather than more passive ways of engaging with social media (e.g. Facebook, Instagram). It could be possible that real-time chatting is less arousing than other types of activities on social media (e.g. reading dubious or provocative news stories on Facebook etc.). Thus, we cannot exclude that other types of social media use might result in stronger effects on sleep. Further it was not possible to check for the content of their social media use due to data protection of the participants. In a next study it would be helpful to get this information to get an impression about the arousing effect some content might cause.

Another limitation given by the controlled setting of a sleep laboratory is not only the reduced external validity, but also that social media use was restricted to 30 minutes. This restriction might be not long enough to result in potential sleep disturbing effects. Although we choose the long format of a PMR (30 minutes) for comparability purposes, there was no possibility to expand the social media exposure. To remove this limitation in a next study, a non-laboratory setting at the participants' home would be helpful to investigate the potential disturbing effects of social media on sleep.

Conclusion

In sum, this study does not provide empirical evidence for a general adverse effect of social media consumption on pre-sleep arousal, sleep, or performance the following day. Thereby, it contradicts findings from previous studies. However, negative effects of pre-sleep social media use on subjective and objective pre-sleep arousal and sleep emerged when comparing social media use nights to those nights in which participants actively applied PMR. Thus, performing progressive muscle relaxation is clearly a better alternative to social media use before sleep. This is substantiated, as social media use before sleep in real-life oftentimes exceeds 30 minutes (the limit in our study procedure). Delaying bedtime due to prolonged media use might have additional impairing effects on sleep and on the duration of sleep going beyond the findings of this study, especially if we consider when wake-up times are externally determined by school hours or working schedules. Thus, while the sole activity of using social media itself does not appear to have strong detrimental effects on sleep architecture, it is

still recommendable to limit the use of any media activity at bedtime in order to get a sufficient amount of restorative sleep.

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3.3 Pre-sleep intentions to react to stimuli during sleep impair sleep and alter stimulus processing during sleep[6]

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Abstract

Sleep is a state of strongly reduced consciousness. Still, pre-sleep intentions to react to specific stimuli during sleep can strongly affect the sleep process. For example, being on call as a firefighter or medical doctor will facilitate reactions to waking sounds during sleep. However, the exact contributions of the pre-sleep intentions itself on the sleep process is still not fully understood. Here we replicated a study by Wuyts and colleagues (2012) and tested the impact of pre-sleep intentions to react to stimuli independently from the actual sound presentation during sleep. In addition to the manipulation of the original study, we introduced a "sound" group, where sounds were actually presented. Twenty-six healthy young participants spent two nights in the sleep laboratory. Sleep was recorded using polysomnography. In one night, they were instructed to react to sounds during sleep ("on call" condition), while they did not need to react to sounds in the other night ("neutral" condition), in a balanced order. Unknown to the subjects, sounds were only presented in one group during both nights ("sound" group), while no sounds were presented in any of the two nights in the other group ("no sound" group). In accordance with our hypotheses, the instruction of being "on call" decreased objective sleep efficiency similarly in both groups, independently of sounds being present during sleep or not. In addition in the "sound" group, event-related potentials in response to sounds as well as slow-wave activity were reduced in the "on call" condition compared with the "neutral" condition. Our results show that pre-sleep intentions to react to stimuli impairs sleep independently of sounds actually being present. Furthermore, pre-sleep intentions alters processing of sounds during sleep. Our results enhance our understanding of the impact of pre-sleep cognitive activity on sleep processes. In addition, they highlight the importance of subjective relevance for reducing negative impact of external noise sources like traffic or church bells and thereby improving sleep.

Keywords

On Call, Sleep, Alarms, Instructions, ERP, Replication

Introduction

Sleep is beneficial for our mental and physical health with many factors affecting sleep quality (Cordi et al., 2019; Kaneita et al., 2009; Lange et al., 2010). While sleep is a state of strongly reduced consciousness, acoustic stimuli are processed during sleep and can lead to arousal and awakenings when presented above a certain threshold (Nir et al., 2013). During conscious wakefulness, we can voluntarily increase our ability to detect and perceive acoustic stimuli by shifting our attention to the relevant stimuli (Hugdahl, 2011; Posner, 1994; Westerhausen & Kompus, 2018). It is not fully clear whether attentional shifts are possible when consciousness is reduced. On one hand, automatic attentional shifts appear almost absent during sleep, as infrequent sounds in a strain of repetitive sounds ("Oddball" paradigm) do not evoke a clear mismatch negativity (MMN) during non-rapid eye movement (NREM) sleep (Ceklic & Bastien, 2015; Loewy et al., 2000; Sabri & Campbell, 2002). On the other hand, voluntary focusing of attention appears to be preserved during sleep, as pre-sleep intentions to react to specific stimuli (e.g. the cry of one's own child) typically lead to facilitated awakenings from sleep (Cottrell & Khan, 2005; McCann et al., 2015). Similarly, knowing to be on call facilitates awakenings by specific sounds, but can also lead to general sleep disturbances in many professions like nurses or physicians (Gaba & Howard, 2002; Smithers, 1995), firefighters (Finnerty, 1977; Paterson et al., 2016), or engineers (Pilcher & Coplen, 2000; Torsvall & Åkerstedt, 1988). Importantly, Wuyts and colleagues (2012) showed that even when no wakening sounds occurred during sleep, the pre-sleep instructions to be "on call" impaired objective sleep efficiency, as well as increasing the time spent awake during the sleep period and increasing beta-activity during sleep. As no group that actually listened to the sounds during sleep was included in this study, the relative impact of the instructions on sleep quality, compared to the physical sounds, remains unknown.

Here we aimed at replicating the results of Wuyts and colleagues (2012) as well as further investigate the influences of sounds by adding an additional group that actually received sounds during sleep. We propose that pre-sleep intentions to react to specific stimuli during sleep per se will affect the sleeping process independently of sounds actually being presented during sleep. In contrast, presentation of the same sounds without the instruction to react will cause no, or only minor, sleep impairments.

To test our hypothesis, 26 young and healthy participants spent two nights in the sleep laboratory with different instructions: in one night, they were instructed to press a button whenever they heard a tone during sleep ("on call" condition). And in the other night, no instruction to press a button was given ("neutral" condition). The nights occurred in a balanced order. Unknown to the participants, only a portion of the participants (n = 12) received tones during sleep in both nights ("sound" group). The other group (n = 14) did not receive any sounds in any night of sleep ("no sound" group). We hypothesized the pre-sleep instruction to react to tones will decrease sleep efficiency to a similar extent in both groups, independent of whether sounds are presented during sleep or not. In the "sound" group only, we predicted that the instructions to be "on call" will alter event-related potentials elicited by the sound

compared to the neutral condition, while no clear sleep impairments will be detected in the neutral condition in this group.

Materials and methods

Participants

Twenty-six young healthy students (17 women) with a mean age of 22.9 ± 3.0 [SD]) participated in the study. Twelve participants were randomly assigned to the "sound" group, whereas 14 participants were included in the "no sound" group. Participants were recruited using an advertisement newsletter for students and among friends of experimenter. They had no known sleep disorders (screened by the Pittsburgh Sleep Quality Index (Buysse et al., 1989)). Participants were asked to keep a regular sleep rhythm and avoid night shifts. They were instructed to abstain from alcoholic and caffeinated beverages on the day before experimental day and on the testing day. The local ethics committee of the University of Fribourg approved the study, and all subjects gave written informed consent prior to participating. The study was conducted in accordance with the declaration of Helsinki. Subjects received either university course credits or monetary compensation of 150 CHF for their participation. Additionally, they were rewarded with 20 CHF for their efforts to react to sounds during the night. In case of an early drop out, payment was provided proportionately.

Experimental Design and Procedure

The within-subject design included one adaptation and two experimental nights in the sleep laboratory at the University of Fribourg. In one of the experimental nights, we instructed participants to stay on call and react to auditory stimuli presented during the night ("on call" condition) by pressing a buzzer installed next to the pillow. To create a more realistic situation, the written instructions informed the participants to imagine that someone's life is depending on their reaction time and that they get rewarded the faster their reaction to the stimuli is. The other night figured as baseline ("neutral" condition) and participants slept regularly. The experimenter and the subjects were blind towards the type of instruction until the very moment before sleeping. The order of the instructions was randomly assigned and balanced between subjects.

Secondly, we included a between-subject factor of "sound" group. Each participant was randomly assigned to a "sound" or "no sound" group. In the "sound" group, during both experimental nights ("on call" and "neutral" condition), 80 sounds in different volumes were presented. A speaker automatically presented four blocks of 10 alarm-sounds in ascending volumes during the night. The setting ensured alarm sounds during the whole night. Sounds were presented using E-Prime (Psychology Software Tools Inc., Pittsburgh). The between-subject factor was double-blinded: neither participant nor experimenter knew about it until the end of data collection.

Each night, participants arrived at the sleep lab and filled in some standardized questionnaires before the installation of the polysomnography (PSG including electroencephalogram (EEG), electromyogram (EMG), electrooculogram (EOG), electrocardiogram (ECG)). Afterwards they completed a memory task, where they learned 80 semantically associated word pairs (PAL (Rasch et al., 2006)). When lying in bed, they received the instruction for the night ("on call" or "neutral" condition). After 8 hours of sleep the experimenter woke the participants up by turning on the light. Directly after getting up, participants filled in standardized questionnaires (subjective sleep rating, mood), did a psychomotor vigilance test (PVT), and the morning-recall of the PAL. The session ended with removal the PSG (see Figure 1).

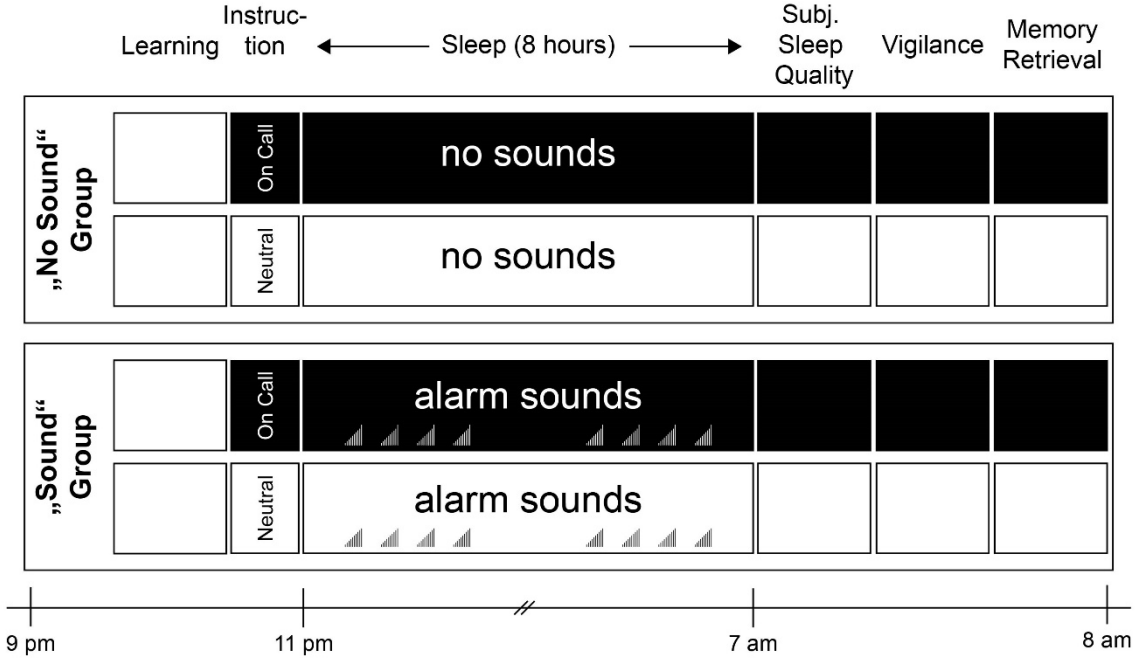

Figure 1. Procedure and experimental design. Participants spent one adaptation night and two experimental nights in the sleep lab. Directly before going to sleep and when lying in bed participants received the instructions related to the condition. Receiving the instruction "on call", participants were instructed react to auditory stimuli presented during the night by pressing a buzzer installed next to the pillow. The written instructions informed participants to imagine that someone's life is depending on their reaction time, and that they would be rewarded proportionately to the speed of their reaction to the stimuli. The "neutral" instruction gave participants the permission to sleep. In addition to the within-subject condition (on call vs neutral), a between-subject condition was implemented: One group actually heard sounds during the night ("sound" group) whereas the other half of the participants had no sounds presented ("no sound" group). Sleep was recorded using PSG. Before the instructions for the night, participants filled out questionnaires, performed the learning part of a word-pair learning (PAL, Learning) task (Rasch et al., 2006). In the morning after sleep participants filled out questionnaires to rate their subjective sleep quality, performed a psycho-vigilance task (PVT) to measure their vigilance and retrieved word-pairs learned before sleep (Memory Retrieval). The time in bed was restricted to 8 hours and constant over all participants

Material

On call instruction

Instructions were given in a written form when participants were lying in bed and were ready to sleep. Participants were instructed to be on call duty and to act as if they were responsible for another person, who would ring an alarm. The faster they reacted, the better, which would be compensated by receiving additional money for each correct reaction, and losing money for each incorrect stimulus reaction. They were told that 10 to 100 alarm sounds would be presented at different volumes, and that it might be difficult to hear all of them. An example of this alarm sound was given.

Sound presentation during the night

The experimenter started an E-Prime run (Psychology Software Tools Inc., Pittsburgh) after turning off the light. The file was started each night ("on call" and "neutral" condition) to prevent a double-blind setting, where neither participant nor experimenter knew that there was a between-subject-factor sound vs. no sound and a within-subject-factor type of instruction ("neutral" vs. "on call"). During the "on call" condition, sounds were presented block-wise using E-Prime (Psychology Software Tools Inc., Pittsburgh) for stimulus presentation. Directly after starting E-Prime no sounds were presented for 30 min. Afterwards, sounds were presented in eight blocks. Each block included 10 sounds at different volumes. The volume of the sound raised up to 40dB. A break of 1 min allowed participants to fall back asleep between two alarm sounds. After 10 sounds (= one stimulus-block) a 15-minute-break followed, where no sounds were presented. After the fourth block, a break of 2 hours was added to enable sounds in both night-halves. After finishing the presentation, E-prime showed a black screen informing experimenter that he/she could end the experiment by pressing any key.

During the neutral night no sounds were presented. E-prime presented a black page for 1 hour before telling the experimenter to end the experiment by pressing any key.

Polysomnographic recordings and sleep analysis

Sleep was measured using single gold-cap electrodes following the 10-20-EEG-system (F3, F4, C3, C4, P3, P4, O1, O2) with a sampling rate of 500 Hz. Impedances were kept below 5kΩ. During recording electrodes were referenced against Cz and re-referenced to the mastoids. Additionally, we measured EMG, EOG and ECG to complete the polysomnographic recording. Data was preprocessed with Brain Vision Analyzer 2.0 (Brain Products GmbH, 2017), filtering the data following the guidelines suggested by the American Association of Sleep Manual (Iber et al., 2007). Two independent scorers, blind to the conditions, scored sleep in 30s periods following the AASM scoring setting (Iber et al., 2007). One scoring was performed by a human scorer and the second one was performed by The Siesta Group (The Siesta Group Schlafanalyse GmbH, Vienna). The two independent scorings were controlled by a third, independent scorer (human).

Analysis of EEG data

EEG data were preprocessed using BrainVision Analyzer 2.1 (Brain Products GmbH, Gilching). First all data were filtered using a low-pass filter of 50 Hz and a high-pass filter of 0.1 Hz. A semi-automatic artefact correction was done (preselecting artefacts of ± 600 µV and an interval length of 200ms) and remaining artefacts were removed manually. We calculated the average power of oscillatory activity in different frequency bands: slow wave activity (SWA, 0.5 – 4.5 Hz), slow oscillations (SO, 0.5-1 Hz), slow spindles (11-13 Hz), and fast spindles (13-15 Hz), as well as delta (1 – 4 Hz), theta activity (4.5 – 8 Hz), alpha activity (8 – 11 Hz), beta activity (15-30 Hz). Data from lights off in the evening to lights on in the morning were analyzed, segmented for NREM sleep (N2 and N3 sleep) and REM sleep. Prior to data analyzes, we conducted a conservative outlier correction (individual group mean ± 4 SEM).

Fast Fourier Transformation analysis

For the frequency analysis we used BrainVisionAnalyzer 2.1 (Brain-Products Inc, GmbH, Gilching). To conduct the Fast Fourier Transformation (FFT) we followed the procedure described by Ackermann and colleagues (2015). We set a high-pass (0.1 Hz) and a low-pass filter (40 Hz), and data were re-referenced to the average of both mastoid electrodes. Finally, an FFT with a 10% Hanning window and a resolution of 0.25 Hz was performed for every EEG channel to calculate the power in each frequency band. We analyzed segments from sleep onset to sleep offset (sleep without SOL), as well as sound blocks and arbitrary created blocks, at the same time, when no sounds were presented (sound presentation took place block-wise with a fixed starting and end point based on the start of E-prime). Within these segments, sections of 2048 data points (4000ms) with a 100-point overlap were created. An automatic artifact rejection excluded segments (exclusion criteria: EMG > 150 µV difference; EEG > 500 µV) before running the FFT. In the end, the power was averaged. For a more thorough analysis in RStudio (R Core Team, Vienna; RStudio Team, Boston), the mean power of all frequency bands were exported as mentioned above.

Eevent-related potentials (ERP) analysis during sleep

Event-related potentials (ERP) reflect neurophysiologic activity without participant's awareness for stimuli during sleep (Atienza, Cantero, & Escera, 2001; Yang & Lo, 2007). ERPs were analyzed using BrainVision Analyzer 2.1 (Brain Products GmbH, Gilching). Only sounds presented during artefact-free NREM sleep stages N2 and N3 were included in the analysis. Segments included the signal from 3000ms before the tone until 8000ms after the tone. The signal was baseline corrected using 1000ms before the onset of the tone. For the analyses, the mean potential between 400ms to 800ms was extracted and further analyses using 'R'. ERPs were plotted in Matlab (Mathworks, Natick).

Analysis of reactions to alarm sounds

Participants were instructed to react to the alarm sounds by pressing a buzzer, which was fixed on the mattress next to the pillow. For this analysis we used BrainVision Analyzer 2.1 (Brain Products GmbH, Gilching). Data were analyzed in two steps: first an automated threshold analysis was performed (± 3000 μV) with a time tolerance of 60000ms to avoid multiple counting of one buzzer reaction. In a second step a visually correction was performed by a human scorer. Reactions only counted when there was a visible reaction in the electromyogram (EMG) as well. In a second part of this analysis, we created segments for arousal analysis (3000ms before and 10000ms after each buzzer reaction). These segments were pre-processed by squaring and smoothing the EEG-signal. In these segments an automated threshold detection (threshold ± 3000 μV with a time-tolerance of 60000ms) was performed by Brain Vision Analyzer 2.1 (Brain Products, Gilching) and a human scorer performed an analysis of arousals.

Analysis of ECG and physiological arousal

For the analysis of ECG we used Kubios HRV Premium 3.2.0 (Kubios Oy, Kuopio). Therefore, the ECG signal for the whole night (lights off to lights on) was exported in EDF+-format using BrainVisionAnalyzer Version 2.0 (Brain Products GmbH, Gilching). Kubios HRV Premium offers an automatic artifact correction based on RR series to eliminate ectopic beats and artifacts on unfiltered data (Tarvainen et al., 2019). Afterwards data were analyzed in time and frequency domain and used for calculation the mean heart rate (mean HR, in beats per minutes (bpm)) and mean RR-interval (mean RR, in milliseconds) as an index for physiological arousal (Kogler et al., 2015). Additionally, we used the HRV triangular index, PNS-, SNS-, and stress index provided by Kubios to evaluate an index for physiological stress. PNS-index is a parasympathetic nervous system (PNS) tone index based on mean RR whereas SNS-index the sympathetic nervous system (SNS) reflects based on mean HR (Tarvainen et al., 2019).

Questionnaires

In the first night, participants filled out a general questionnaire for personal information, the Edinburgh Inventory for Handedness (Oldfield, 1971), the Pittsburgh Sleep Questionnaire Inventory (Buysse et al., 1989) and a questionnaire for chronotype (Griefahn et al., 2001). Additionally, in each session, participants filled out a general questionnaire and a questionnaire on mood (Steyer et al., 1997). In the morning participants were asked to rate their subjective sleep quality (Görtelmeyer, 2011) and again their mood (Steyer et al., 1997). All questionnaires were presented online using SoSci Survey (Leiner, 2018).

Memory measurement

Episodic memory was tested with a paired-associated learning task (PAL; Rasch et al., 2006). Each night, the participants learned a list of 80 semantically related word pairs (Feld et al., 2016; Rasch et al., 2006). Each trial started with the first word of a pair, which was presented for 3000ms and followed by a 500ms blank interval, which separated the single trials. The words were presented in black font on a white screen via E-Prime (Psychology Software Tools Inc., Pittsburgh). Every pair was presented only once while the order was kept constant. Immediately after learning participants were confronted with a cued recall test. Here, they had to come up with the corresponding word when the first word was displayed. During the recall test, the word pairs were presented for an infinite amount of time, or until the participant pressed enter. Right after, the correct answer was presented for 1000ms, followed by a 500ms blank interval which separated the single trials. After each word pair was tested once and provided the participant gave the correct answers, a second recall test followed. There participants did not get any correction, and right after filling in the answer a 500ms blank interval followed. During recall, the order of the word pairs differed from learning phase but was kept constant across subjects. A second recall phase took place in the morning. Performance was measured as percentage of words recalled in the morning relative to the amount remembered immediately after learning. Response time was not restricted.

Psychomotor Vigilance Test

In the morning participants performed a test to measure their vigilance (psychomotor vigilance test (PVT), Dinges & Powell, 1985). Subjects were asked to press the space key with their non-dominant hand as soon as they recognized the millisecond counter on the screen, which appeared at random intervals. After the keypress the reaction time in milliseconds was shown for 1000ms, then the counter was zeroed, and this was repeated.

Statistical analysis

Data were analyzed using a repeated measurement analysis of variance (ANOVA) with the within-subject factor instruction (“on call” vs. “neutral”) and the between-subject factor sound-group (“sound” vs. “no sound” group). According to sleep quality, our main outcome variables were subjective and objective sleep quality, sleep onset latency (SOL), wake after sleep onset (WASO), number of awakenings during sleep (NWAK) and sleep depth. Total sleep length was excluded from this primary analysis as participants were awakened 8 hours after lights off. In the exploratory analysis, we additionally analyzed further sleep parameters, oscillatory power during NREM sleep as well as vigilance, sleep-associated memory consolidation, and heart rate variability (HRV).

Analysis of variance (ANOVA) for repeated measurements with the between-subject-factor sound-group (“sound” vs. “no sound” group) and the within-subject factor instruction (“on call” vs. “neutral”) were used for the analyses. In addition, for the primary outcome variables, a factor type of

parameter ("subjective" vs. "objective" parameters) was used. Post hoc paired t-test were conducted in case of a significant main effects or interaction effects. We set the level of significance to $p = 0.05$ and report effect sizes (η^2) only for significant data. Data were analyzed using R Studio (R Core Team, Vienna; RStudio Team, Boston). Results are presented as means ± standard errors. As soon as the sphericity was violated, we applied the Greenhouse-Geisser correction.

Data availability

Datasets analyzed during the current study are available online on https://osf.io.

Results

Buzzer Analysis

In a first step, we analyzed the buzzer reactions of participants. In the "sound" group, in which sounds were played during both nights, participants pressed the buzzer 27.67 ± 2.31 times when they were instructed to be "on call" compared to a "neutral" condition without instructions to react (4.92 ± 0.69; $p < 0.001$). Remarkably, the instruction had almost identical effects in the "no sound" group in which no sounds were played neither in the "on call" nor in the "neutral" condition: participants pressed the buzzer 26.87 ± 1.60 times in the "on call" condition and only 1.93 ± 0.43 times in the "neutral" condition ($p < 0.001$). During "on call", the amount of buzzer responses was comparable between the "sound" and "no sound" group ($p > 0.84$). In the "neutral" condition, button presses were significantly more frequent when sounds were actually played during sleep as compared to the "no sound" group ($p = 0.021$). Overall, only the instruction to be "on call" or not had a significant impact on buzzer presses during sleep ($F(1, 24) = 145.18$, $p < 0.001$, $\eta^2 = 0.74$). In contrast, the main effect of sound presentation ("sound" vs. "no sound" group) did not reach significance ($F(1,24) = 0.81$, $p = 0.377$) , neither did the interaction ($F(1, 24) = 0.30$, $p = 0.587$). All results are presented in Table 1.

Objective and subjective sleep parameters

In accordance with our hypothesis, the instructions to be "on call" or not generally influenced objective sleep, independently of whether sounds were actually played during the night or not. First of all, objective sleep efficiency was significantly lower in the "on call" condition (89.84 ± 1.68) as compared to the "neutral" condition (92.50 ± 1.11 (main effect of instruction: $F(1,24) = 5.42$, $p = 0.029$, $\eta^2 = 0.18$)). In contrast, presentation of physical sounds during sleep did not generally impair sleep efficiency ("sound" group: 92.56 ± 1.10 vs. "no sound" group: 89.98 ± 1.65; $F(1,24) = 0.98$, $p = 0.333$), Exploratory pair-wise comparisons revealed that the instructions significantly impaired sleep efficiency in the "no sound" group only ($p = 0.042$), but not in the "sound" group ($p > 0.90$). However, the interaction between group ("sound" vs. "no sound" group) and instruction ("on call" vs. "neutral"

condition) was not significant ((F(1,24) = 1.24, p = 0.276, see Figure 2a and Table 1). Sleep onset latency (SOL) did not differ, neither between sound-groups nor instruction (see Table 1). However, participants spent significantly more time awake during the night (WASO) when they received the instruction to be "on call" (31.23 ± 4.51 min) compared to "neutral" (20.17 ± 2.43 min), both in the "sound" group and in the "no sound" group (F(1,24) = 5.55, p = 0.027, η^2 = 0.19), see Figure 2b. Furthermore, they woke up more often (number of awakenings, NWAK) when they were "on call" (14.50 ± 1.23) compared to the "neutral" condition 11.58 ± 1.24), again independent of the presence of real sounds during sleep ("sound" group 12.92 ± 1.11 vs. "no sound" group 13.14 ± 1.38; F(1,24) = 5.42, p = 0.029, η^2 = 0.18, see Figure 2c). Neither for time spent awake nor for the number of awakenings we observed a main effect of sound-group ("sound" vs. "no sound") or any interaction (see Table 1). For subjective sleep parameters, we did not observe any significant influences of pre-sleep instructions or sound presentations during sleep (see Table1).

The impact of pre-sleep instructions was also visible on the level of sleep architecture, although the effects did not reach significance. Participants spent generally less time during slow-wave sleep in the "on call" (94.67 ± 5.43 min) compared to the "neutral" condition (103.92 ± 5.91 min). However, the effect only reached a statistical trend F(1,24) = 3.39, p = 0.078, η^2 = 0.15). Similar, participants spent more time in the light sleep stage N1 in the "on call" (27.00 ± 1.84 min) vs. "neutral" condition (23.44 ± 1.83 min) (F(1,24) = 4.16, p = 0.052). Other sleep stages (N2, REM sleep) were not affected by the instructions. Importantly, we did not observe a change in sleep stages with respect to the presentation of sounds (all main effects of sound-group ("sound" vs. "no sound") p > 0.30) and no interactions (p > 0.11). However, exploratory pairwise comparisons revealed that the effects of instruction on sleep architecture were significant in the sound, but not the "no sound", group both for SWS and N1 sleep (see Figure 2d-e). Interestingly, the only significant interaction was observed for the time to reach SWS (SWS latency, F(1,24) = 8.64, p = 0.007, η^2 = 0.26): only when sounds were actually presented during sleep ("sound" group), participants reached SWS significantly later when being "on call" (17.42 ± 1.91 min) compared to the "neutral" condition (13.17 ± 1.05 min, p = 0.014). In contrast in the "no sound" group, participants reach SWS earlier when being "on call" vs. the "neutral" condition (13.93 ± 0.78 min vs. 15.43 ± 0.88 min, p > 0.78, see Figure 2f). For REM latency neither a main effect nor the interaction reached significance (all p > 0.17).

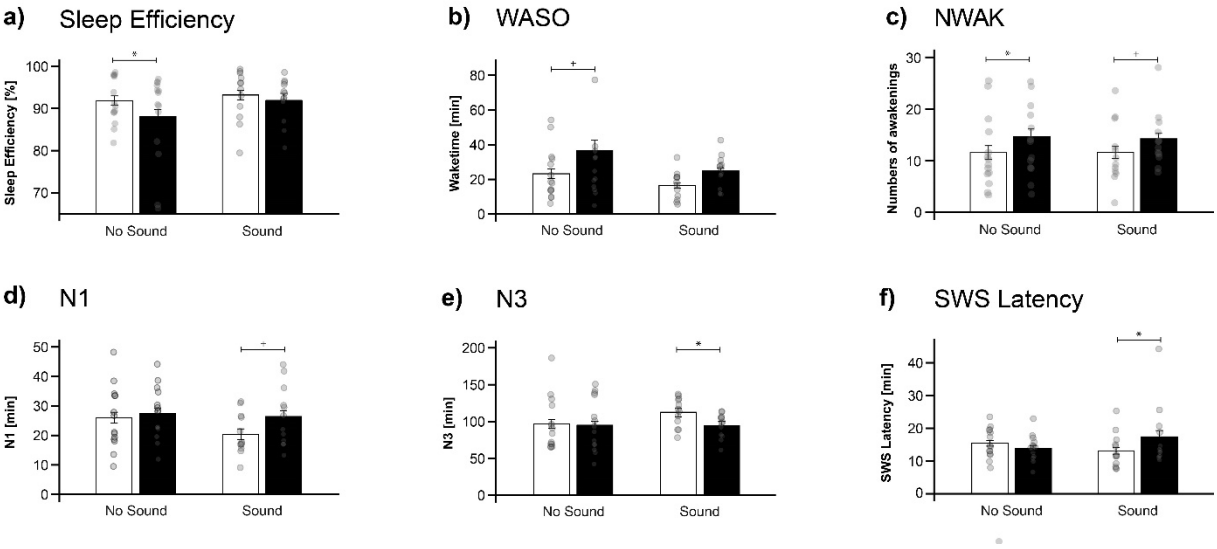

Figure 2. Objective sleep parameters. On an objective level Participants we looked at sleep efficiency (a) as an objective measurement of sleep quality, which showed a significant effect of instruction with decreased sleep efficiency in the night with on call duty. Participants spent significantly more time awake (WASO (b)) when they were instructed to be on call compared to the neutral instruction The same pattern was shown as well for the numbers of awakenings (NWAK (c)). Additionally we looked at different sleep stages. N1 (d) showed a tendency for an instruction effect with more N1 sleep when instructed to be on call for this night. Sleep stage N3 (deep sleep (e)) showed the same pattern but with more deep sleep in the neutral night. All effects remained independent of the actual presence of sounds (Sound) or no sounds (No Sound). One exception was the time until reaching the first slow wave sleep (SWS Latency (f)), which showed an interaction between the sound/no sound group and the instruction (on call vs. neutral). Means ± standard errors of the mean are indicated. Significant pair-wise comparisons from post-hoc tests are indicated by +: $p \leq 0.1$ *: $p \leq 0.05$.

Table 1. Sleep parameters for the three conditions (subjective and objective)

	Sound				No Sound									
	Neutral		On Call		Neutral		On Call		Instruction		Sound Group		Interaction	
	mean ± SEM		**mean ± SEM**		**mean ± SEM**		**mean ± SEM**		**F-Value**	**p-Value**	**F-Value**	**p-Value**	**F-Value**	**p-Value**
Buzzer Reactions	4.92 ± 0.69		27.67 ± 2.31		1.93 ± 0.43		26.87 ± 1.60		145.18	< 0.001	0.81	0.377	0.30	0.587
Obj. Sleep Parameters in min														
Sleep Efficiency [%]	93.20 ± 1.11		91.91 ± 1.68		91.90 ± 1.11		88.06 ± 1.68		5.42	**0.029**	0.98	0.333	1.24	0.276
SOL [min]	21.96 ± 4.80		21.96 ± 4.54		22.82 ± 4.36		22.11 ± 3.50		0.01	0.905	0.00	0.950	0.01	0.912
WASO [min]	16.58 ± 1.59		24.83 ± 1.83		23.25 ± 2.89		36.71 ± 5.80		5.55	**0.027**	2.96	0.098	0.31	0.585
NWAK	11.58 ± 1.17		14.25 ± 1.04		11.57 ± 1.34		14.71 ± 1.41		14.32	**0.001**	0.01	0.926	0.09	0.761
N1 [min]	20.42 ± 1.33		26.42 ± 2.00		26.04 ± 2.07		27.50 ± 1.75		4.16	0.052	1.09	0.307	1.68	0.207
N2 [min]	213.08 ± 4.91		219.42 ± 6.25		212.71 ± 6.51		203.71 ± 7.37		0.14	0.716	0.47	0.948	2.15	0.156
N3 [min]	112.58 ± 3.91		94.46 ± 3.20		96.50 ± 7.02		94.86 ± 6.94		3.39	0.078	0.59	0.450	2.68	0.115
REM [min]	95.17 ± 2.84		92.88 ± 4.21		100.50 ± 3.91		93.36 ± 3.80		2.10	0.160	0.19	0.668	0.51	0.481
SWS Latency [min]	13.17 ± 1.05		17.42 ± 1.91		15.43 ± 0.88		13.93 ± 0.78		1.40	0.248	0.08	0.784	8.64	**0.007**
REM Latency [min]	99.88 ± 8.43		78.88 ± 5.10		86.46 ± 4.81		84.21 ± 4.95		2.00	0.170	0.20	0.659	1.47	0.237
TST [min]	457.83 ± 4.75		458.04 ± 4.46		459.00 ± 5.99		456.18 ± 3.48		0.12	0.730	0.00	0.960	0.14	0.710
N1 [%]	4.48 ± 0.30		5.77 ± 0.42		5.69 ± 0.46		6.02 ± 0.37		4.25	**0.050**	1.11	0.303	1.63	0.214
N2 [%]	46.50 ± 0.86		47.84 ± 1.15		46.49 ± 1.51		44.55 ± 1.50		0.13	0.723	0.50	0.485	1.92	0.179
N3 [%]	24.59 ± 0.82		20.70 ± 0.75		20.96 ± 1.46		20.78 ± 1.51		2.93	0.100	0.67	0.421	2.81	0.107
REM [%]	20.76 ± 0.54		20.28 ± 0.90		21.86 ± 0.76		20.47 ± 0.81		1.56	0.223	0.24	0.630	0.35	0.561
WASO [%]	3.68 ± 0.38		5.43 ± 0.42		5.02 ± 0.60		8.18 ± 1.34		5.85	**0.024**	0.67	0.115	0.46	0.505

Sleep Quality	3.74 ± 0.16	3.19 ± 0.12	3.30 ± 0.16	3.34 ± 0.12	1.71	0.204	0.41	0.528	2.65	0.117
SOL [min]	17.58 ± 2.64	17.92 ± 2.85	22.14 ± 2.54	20.00 ± 2.21	0.17	0.685	0.54	0.468	0.26	0.617
WASO [min]	10.83 ± 2.97	13.58 ± 3.29	11.00 ± 2.28	15.93 ± 3.44	1.61	0.216	0.06	0.811	0.12	0.728
NWAK	2.25 ± 0.31	3.17 ± 0.20	2.36 ± 0.27	2.21 ± 0.27	1.03	0.320	1.04	0.318	2.40	0.135
Depth	3.75 ± 0.22	3.33 ± 0.21	3.14 ± 0.22	3.14 ± 0.23	0.54	0.470	1.27	0.271	0.63	0.436
Memory										
Learning	58.75 ± 2.60	59.67 ± 2.82	55.36 ± 3.35	54.64 ± 2.75	0.00	0.981	0.56	0.461	0.27	0.608
Recall	57.58 ± 2.49	58.58 ± 3.02	53.71 ± 3.36	52.36 ± 3.04	0.03	0.864	0.75	0.395	0.57	0.459
Consolidation [%]	98.21 ± 0.83	97.71 ± 0.95	97.05 ± 0.95	94.92 ± 1.14	1.47	0.230	1.52	0.229	0.51	0.483
Vigilance										
Mean RT [ms]	327.82 ± 5.92	326.83 ± 6.61	328.73 ± 6.96	332.84 ± 5.90	0.11	0.738	0.09	0.771	0.27	0.609
Errors	0.67 ± 0.16	1.33 ± 0.37	0.77 ± 0.15	1.08 ± 0.33	1.59	0.220	0.04	0.840	0.22	0.642
Reactions	76.75 ± 0.65	76.58 ± 0.67	76.69 ± 0.39	77.54 ± 0.66	0.18	0.675	0.28	0.604	0.36	0.556
Physiological Arousal										
PNS index	1.60 ± 0.30	1.34 ± 0.34	1.30 ± 0.40	1.25 ± 0.40	0.68	0.418	0.11	0.741	0.32	0.577
SNS index	-0.48 ± 0.33	-0.43 ± 0.27	-0.51 ± 0.24	-0.45 ± 0.21	0.06	0.809	0.00	0.958	0.00	0.986
HRV triangular index	13.21 ± 1.04	12.18 ± 1.00	11.45 ± 0.90	11.94 ± 0.98	0.29	0.593	0.45	0.507	2.34	0.139

Notes. Buzzer reactions were counted in both instruction conditions (on call vs. neutral) over the whole night for the total sleep time (TST) Objective values are based on polysomnographic recordings. Non-rapid eye movement (NREM)-sleep, stage 1, 2, and 3 sleep (N1, N2, N3), rapid eye movement sleep (REM), waketime after sleep onset (WASO), total sleep time (TST), sleep onset latency (SOL), slow wave sleep latency (SWS latency), REM sleep latency (REM latency) are all measured in minutes [min] and the percentages indicate parietal percentage of TST [%]. Subjective parameters are based on subjective ratings in the SF-A-R (Görtelmeyer, 2011). For memory, numbers indicate absolute or relative values of correctly recalled words that were presented in the evening (learning phase with first recall) and in the morning (retrieval phase with second recall). Consolidation refers to the difference in performance between learning and retrieval phases. For vigilance, the reaction time (RT), the number of reactions, and amount of errors during the 10 minutes of the psychomotor vigilance task (PVT) were measured. As an objective measurement of arousal, Kubios (Kubios Oy, Kuopio) provided a in index for the parasympathetic nervous system (PNS index; based on mean RR interval in ms), sympathetic nervous system (SNS index; based on mean HR in beats per minute (bpm)), and heart rate variability (HRV triangular index). Values are means (M) ± standard error of mean (SEM).Values are means (M) ± standard error of mean (SEM)

The effect if instruction on Event related potentials (ERP) during sleep

In the "sound" group only, we analyzed the effects on instructions on event-related potentials (ERPs) elicited by the sounds during sleep. Sound presentation during N2 and N3 sleep elicited the well-known K-complex-like response, with a large negative peak between 400 to 800ms after the tone. The instruction to be "on call" significantly decreased the average amplitude in this time range: in N2 sleep, the amplitude in frontal electrodes (F3 and F4) was significantly lower in the "on call" condition (-40.90 ± 8.04 μV) compared with the ERPs to sounds in the "neutral" conditions (-76.04 ± 9.48 μV; p = 0.04). The same pattern occurred during N3 sleep (-51.62 ± 10.84 μV vs. -82.48 ± 9.88 μV, for "on call" and "neutral" conditions, respectively, p = 0.04, See Figure 3 a-d, and Table 2.). The effect of instructions was mainly seen in frontal electrodes, as the overall ANOVA including all electrodes using the factors instruction ("on call" vs. neutral), hemisphere (left vs. right), sleep stage (N2 vs. N3), and topography (frontal, central, parietal, occipital) revealed a significant interaction between the instruction ("on call" vs. "neutral") and topography, ($F(1.55, 13.92) = 5.23$, $p=.026$, $\eta^2 = 0.06$).

Figure 3. Grand-average ERP in frontal electrodes

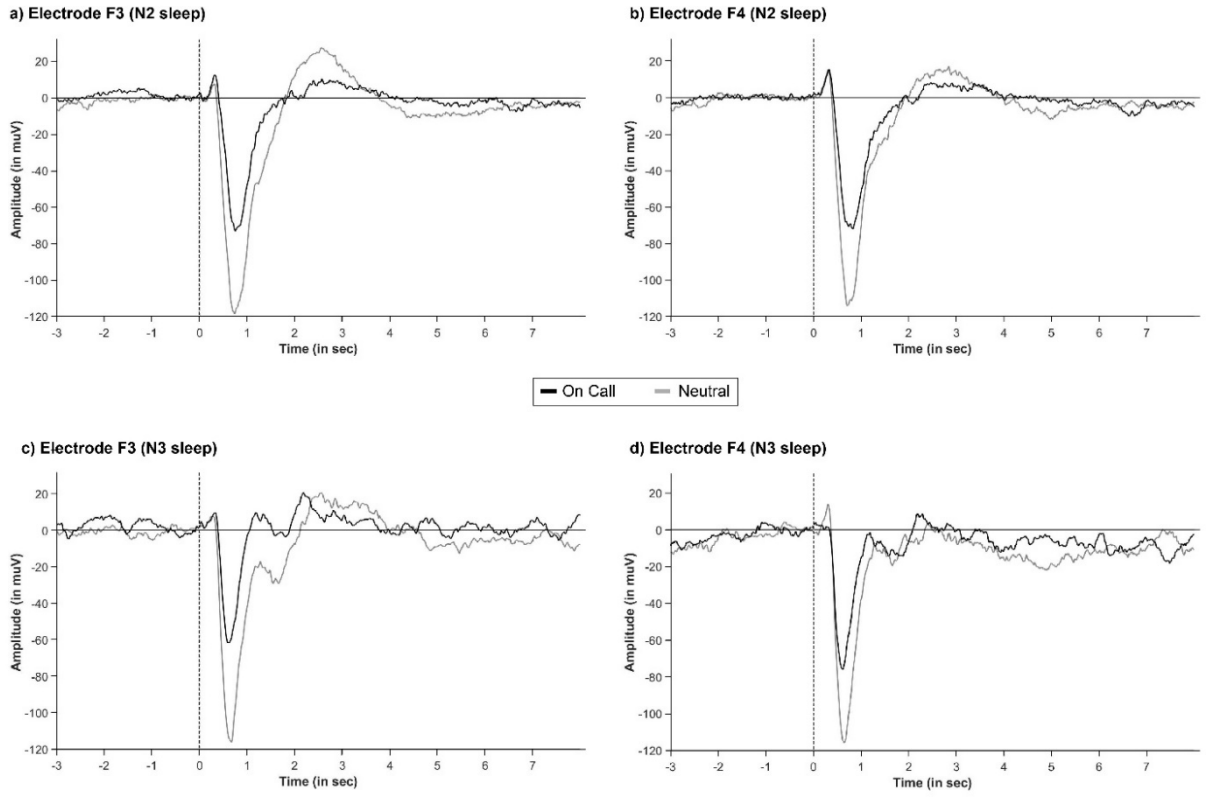

Figure 3. Grand-average event-related potential (ERP) and reaction times to alarm sounds in the "sound" group. (a) shows the mean ERP in the frontal electrode F3 during sleep stage N2. (b) shows mean ERP in frontal electrode F4 during sleep stage N2. (c) shows mean ERP in frontal electrode F3 during deep sleep (sleep state N3). (d) shows mean ERP in frontal electrode F4 as well during N3 sleep.

Power Analysis

Additionally, we analyzed the effect of the on call setting on oscillatory power in the SWA band (0.5 – 4.5 Hz). There was no significant effect of instruction ("on call" vs. "neutral") or significant interaction ("instruction" x "sound-group") in neither N2 nor N3 (all $p > 0.78$) Analysis showed significant differences between the two groups ("sound" vs. "no sound" group) in sleep stage N2 ($F(1, 24) = 9.63$, $p = 0.015$, $\eta^2 = 0.19$) with more SWA in the "sound" group (10.74 ± 0.59 Hz) compared the "no sound" group (8.15 ± 0.49 Hz). Similarly, in N3 ($F(1, 24) = 9.63$, $p = 0.005$, $\eta^2 = 0.26$) there was more SWA in the "sound" group (33.29 ± 5.15 Hz) compared to the "no sound" group (26.57 ± 6.42 Hz).

As a second aspect, we looked at the blocks where sounds were actually presented (stimulation-blocks) and their arbitrary counterparts. The instruction to be "on call" revealed less SWA power (14.87 ± 0.853 μV) compared to the "neutral" condition (17.09 ± 1.00 μV; $F(1, 24) = 6.56$, $p = 0.017$, $\eta^2 = 0.06$). Further the SWA power was higher in the "sound" group (17.90 ± 0.53 μV) compared to the "no sound" group (14.32 ± 1.07 μV; $F(1, 24) = 5.54$, $p = 0.027$, $\eta^2 = 0.15$). No interaction occurred ($p = 0.13$).

During the time between the blocks, the "sound" group showed higher SWA power (16.7932 ± 0.75 μV) compared to the "no sound" group (12.79 ± .80 μV; ($F(1, 24) = 5.91$, $p = 0.023$, $\eta^2 = 0.17$). There was no interaction ($p = 0.47$).

Heart rate and heart rate variability

In addition to brain activity, we analyzed whether participants differed in physiological arousal during stimulation blocks depending on the instruction of being "on call" or not. We used three different indices provided by the analysis software Kubios HRV Premium 3.2.0 (Kubios Oy, Kuopio): an index of the tone of the parasympathetic (PNS) and sympathetic nervous system (SNS) (Tarvainen et al., 2019) as well as an index for heart rate variability (HRV triangular index). We focussed our analysis on the stimulation blocks in the "sound" group. In the "no sound" group, arbitrary stimulation blocks were created (see methods section). However, neither instruction nor sound presentations during sleep influenced the indices of heart rate and heart rate variability (all $p > 0.41$; see Table 1).

Memory consolidation and Vigilance

We also tested whether the on-call instruction as well as the presence of sounds during the night ("sound" vs. "no sound" group) affected memory consolidation or vigilance. Neither instruction ("on call" vs. "neutral" ($F(1,24) = 1.45$, $p = 0.240$)), sound-group ($F(1,24) = 1.41$, $p = 0.246$), nor their interaction ($F(1,24) = 0.25$, $p = 0.625$) was significant. Encoding in the evening and retrieval in the morning did not differ (all $p > 0.34$). All results are presented in Table 1.

Vigilance was tested using a psychomotor vigilance test (PVT; Dinges & Powell, 1985). Vigilance in the morning showed no significant effect ($p > 0.73$) or interaction, neither for reaction time ($p > 0.70$), the numbers of reactions ($p > 0.36$), or errors ($p > 0.22$). For results see Table 1.

Discussion

This study investigated the effect of pre-sleep intentions to react to auditory stimuli in the controlled setting of a sleep laboratory. We predicted that the instruction to be "on call" would impair sleep quality compared to a control condition, independently of sounds actually being present during sleep. Our results confirmed this prediction and showed that the pre-sleep instruction to be "on call" impairs objective sleep efficiency and increased the time awake and number of awakenings during sleep. Furthermore, we observed statistical trends for more light sleep (N1) and less deep sleep (N3) in the "on call" condition.

Our results from the "no sound" group replicated the results reported previously by Wuyts and colleagues (2012): they also reported lower sleep efficiency and prolonged WASO after pre-sleep instruction to be "on call" without any sounds played during sleep. In contrast, we were not able to replicate an increased sleep onset latency and decrease subjective sleep quality ratings in the "on call" condition as reported by Wuyts et al (2012). Possible reasons are that participants in our study received the instruction directly before sleep when already lying in bed, whereas in the original study the instructions were presented 25 min before sleep. Furthermore, Wuyts' instructions precised that no sound would be presented during the first 30 minutes after lights out. This instruction was left out in our "on call" instruction as a previous study in our lab showed that pre-sleep instruction affects SOL (Combertaldi & Rasch, 2020).

In addition to the successful replication, we added a second group to the experimental design, which received sounds during sleep ("sound" group). Importantly, the effects of pre-sleep instructions to be "on call" were similar in this group compared to the "no sound" group. Thus, listening to alarm sounds during sleep did not further increase the impairment of sleep. The presence of the pre-sleep instruction actually almost fully explained the sleep impairments. As slow-wave activity was even increased in the sound group (probably due to sound-induced slow-waves and K-complexes (Bellesi et al., 2014; Dang-Vu et al., 2011; Laurino et al., 2014)), one could even argue that sounds deepened sleep in our study, thereby counteracting the disturbing effects of the instruction. However, this finding might be due to our experimental setting in which we play sounds at different volumes, including several sounds not leading to awaking. Still, our results clearly show that the effect of the pre-sleep instruction to be "on call" is the main factor responsible for inducing sleep disturbances, whether the presence of sounds during sleep plays no or only a minor role.

The outstanding question is how do the presented alarm sounds affect our brain during sleep? Our results showed that alarm sounds induced K-complexes, therefore we assume that there could be a

system coinciding with the presence of K-complexes which evaluates the importance of the presented sounds in relation to the suppression of cortical arousal (Cash et al., 2009). On a neurobiological level, ERP-results showed that the instruction stayed activated during N2 and N3 sleep. Furthermore, we found differences between the left and the right hemisphere with higher ERP in the right hemisphere. This interhemispheric asymmetry might work as a night-watch system not only in the first night in a novel environment (Tamaki et al., 2016) but also in a stressful on call session. Previous studies confirmed an association with stress for on call work (for a review, see Hall et al., 2017). One possibility to measure physiological stress is physiological arousal (Berntson et al., 1997; Tarvainen et al., 2014). Therefore, we looked at the heart rate variability (HRV) as well as the activity of PNS and SNS. Neither the instruction nor the presence of sounds affected any arousal parameter. Our data anticipate that on call work is not associated with physiological arousal. This is in line with studies looking at salivary cortisol, as a marker of physiological stress, during on call duty (Bamberg et al., 2012; Hall et al., 2019). Together with the lack of an enhanced arousal due to on call work, there were no effects on vigilance in the morning or memory consolidation due to the "on call" instruction.

Following the two-process model of sleep regulation (Borbély, 1982), SWA figures as an important measurement of homeostatic component of sleep. Our result confirmed that the instruction "on call" revealed less SWA during the blocks, where the sounds were presented (stimulation-blocks). This difference confirmed that the pre-sleep instruction affects sleep depth, and it is possible to manipulate sleep depth by "on call" instructions. This result led to the conclusion that it is possible to maintain our sleep with cognition. For example, pre-sleep intentions (e.g. instructions) remain active during sleep, and presumably directly modulate sleep regulatory processes (Combertaldi & Rasch, 2020). Beck and colleagues (2020) give an example showing that cognition can affect our sleep depth. They increased sleep depth by reactivation of mental concepts with relaxing words. It seems that the activation of sleep-related cognition, before or during sleep, affects sleep positively whereas the activation of cognitively arousing concepts might decrease sleep quality. This is in line with the assumption, that semantic concepts might be stored in multimodal representations. Due to its embodiment, the cognitive activation thereby modifies the activity in neural networks, which are associated with the processing of bodily functions (Beck et al., 2020). Based on this assumption, in this study, the "on call" instruction may increase monitoring of the environment during sleep and thereby prepare the organism for faster reactions to expected and behaviorally relevant stimuli. While it was not possible to measure the reaction time between stimulus presentation and buzzer reaction, it is important to measure this in future studies. Possible mechanisms could be similar to prospective memory processes during wakefulness, where the intention to react to relevant stimuli facilitates the detection of these stimuli (Einstein & McDaniel, 2005; Rusted et al., 2009). This is in line with the assumption that disturbed sleep is dependent on the cognitive state in the evening. If someone expects to get woken up not only because of on call duty, but also e.g. because of the church bell ringing or the train passing during the night, the chances might be very high to do so. On the other hand, sleepers might benefit from

the pre-sleep instruction “do not wake up because of the church bell ringing”. This seems an important aspect for adaptive sleeping behavior and may increase chances of survival in dangerous surroundings, and therefore remains interesting for future research.

To sum it up, our results replicate Wuyts et al’s showing that pre-sleep instructions to be “on call” impair objective sleep quality. In addition, we extended their result by showing that the impairing effects of pre-sleep intentions to react to specific stimuli are comparable with a group, which actually listened to sounds during sleep. Thereby, we identified the pre-sleep intention as the major factor to explain sleep disturbances in “on call” situations, while the actual disturbance from sounds might play only a minor role. Our results have important consequences for actual on call situations in real life, which are known to impair sleep quality (Bamberg et al., 2012; Hall et al., 2017; Torsvall et al., 1987). Mechanistically, our results highlight the importance of our pre-sleep cognitions for sleep regulation and sleep quality, as we are able to intentionally influence the external monitoring of acoustic stimuli during sleep. Future studies need to examine whether also sleep disturbances caused by external noises such as traffic or church bells depend on our pre-sleep cognitions (e.g. subjective relevance we assign to these stimuli), and need to test whether reducing this relevance is a key factor for improving sleep quality.

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4 Discussion

The purpose of this section is to discuss the combined results of the three studies presented above with respect to the initial hypotheses. Thereby this thesis would like to find an initial explanation to how cognitive factors affect sleep. First, the most important findings will be summarized, interpreted, and embedded within existing literature. Then, results will be interpreted in relation to the three models introduced (cognitive model of insomnia (Harvey, 2002), hyperarousal model of insomnia (Riemann et al., 2010), and MemoSleep hypothesis (Rasch, 2016)) and implications will be drawn. Before providing an overview of the most important upcoming research questions, future directions in sleep research, suggestions for future studies, and some limitations will be outlined.

To start, the focus lays on the first study and aims to answer the question regarding what the role of intention in the interplay between sleep and cognition is. Results of the first study showed that it was possible to affect sleep cognitively by wanting to do so. The intention to sleep worse affected sleep quality negatively. However, sleep quality was not affected by the intention to sleep better. Objective measurements of sleep quality, namely the changes in WASO and sleep depth did not confirm the subjective ones, showing a misperception between subjective ratings and objective parameters of sleep, which has been shown in insomnia patients as well (Maes et al., 2014). Participants were able to reduce sleep quality subjectively while enhancing SOL, WASO, and NWAK. They even reported a decreased perception of sleep depth, which was in line with the instruction to reduce deep sleep intentionally. Results for objective sleep parameters went in a same direction with reduced sleep efficiency as well as enhanced SOL and NWAK. This is in line with the first hypothesis, that the intention to sleep worse decreases sleep quality and that participants would be able to sleep worse, but not better than normal. Although we focused on sleep in healthy participants with no prior known sleep disorder (e.g. insomnia), participants reported that they tried to worsen their sleep quality by rumination and worrying. These are cognitions mentioned frequently by patients with insomnia-related rumination (Zoccola et al., 2009). Following the hyperarousal model of insomnia (Riemann et al., 2010), this kind of pre-sleep cognitive activity prompts a cognitive hyperarousal leading to reduced sleep quality (e.g. reduced SOL and TST). In this first study, participants were able to induce an acute insomnia by wanting to do so.

Before coming to a general explanation and implementing the results with all three models, the following will first sum up the results of the second study showing that pre-sleep social media use does not impair subjective or objective sleep parameters compared to a neutral

night without instructions. This result goes against the predicted hypothesis, i.e., expecting that pre-sleep social media consumption induces a physiological arousal and thereby reduces subjective and objective sleep quality. This effect on pre-sleep physiological arousal was not visible, as social media affected neither HR nor HRV. The study recruited heavy users of social media. It might be possible, that they developed skills to counteract the physiological arousal and thereby eliminate it. In a next study, it would be interesting to see how light users of social media would deal with this situation. Furthermore, the known arousing effect of social media consumption (Cain & Gradisar, 2010; Kater & Schlarb, 2020) were not observed and thereby the hypothesized negative effect of social media on sleep quality was not visible. Although objectively expected changes in SOL, NWAK, WASO, sleep depth, or other sleep parameters were missing, the study confirmed that social media consumption decreased sleep stage N2. So far there is a lack of studies looking at the effects of social media on sleep architecture measured by polysomnography, which would provide an explanation for our results.

Moreover, we introduced a relaxation condition as a control in this second study. Interestingly, apart from our hypothesis, these results showed that pre-sleep relaxation leads to a decreased arousal as measured by HR and affects different sleep parameters positively (for example reduced SOL, enhanced sleep efficiency, or less light sleep (N1)). These results confirmed that pre-sleep arousal can modulate sleep. Even the next morning, the hypothesized effect was visible with the feeling of being more awake.

This paragraph would like to look at the instructions in an on call setting. The third study is a replication of a previous study conducted by Wuyts and colleagues (2012) showing that the instruction to be on call was important to show that an on call-setting in a sleep laboratory negatively affects sleep, although no sounds actually were present. The study looked at the pre-sleep instruction to be on call compared to a neutral condition. Our results confirmed that it is the instruction of being on call that affected sleep, and not the actual presence of alarm sounds. In addition to the original study, we introduced a between-subject factor with two different groups: one group actually received alarm sounds in both nights (“sound” group) whereas the other group heard none (“no sound” group, similar to Wuyts et al’s study). Participants were instructed to react to the sounds presented during the night and showed more reactions to the alarm sounds when they were instructed to be on call. Concerning our hypothesis, i.e., that on call-work affects sleep quality negatively, we found that sleep efficiency was reduced in the on call-night independently of the actual presence of sounds during the night. On a neurophysiological level, participants in the “sound” group showed more SWA, independently of the instruction on call. One could argue that the presentation of sounds deepened sleep

although it seems reasonable that these changes were due to sound-induced slow-waves and K-complexes (Bellesi et al., 2014; Dang-Vu et al., 2011). On a neurophysiological level, we analyzed the event related potentials (ERP) provoked by the sounds during sleep. Sound presentation elicited the well-known K-complex-like response and participants showed higher amplitudes in ERP as a reaction to the sounds when they did not expect any sounds. This is in line with the hypothesis: the presentation of sounds affected sleep, and alarm sounds disturbed sleep when they were not expected. One might assume that this ERP represents a protective role for sleep when no sounds are expected, but actually were presented. Indeed, it has been shown that K-complexes show a sleep-protecting function (Cash et al., 2009; Halász, 2016).

In the next section, this thesis would like to integrate all findings in the three cognitive models presented in the introduction to see how cognitive factors affect sleep. It therefore would like to describe the general aim of this thesis and discuss which of the models introduced above can best explain how and which cognitive factors affect sleep the most.

4.1 What can or cannot be explained by the three models?

In the first study, participants reported that worrying and rumination were strategies they used to worsen sleep. This implied using methods to induce cognitive arousal that insomnia patients suffer from and cannot control (Galbiati et al., 2018). While there was no information about negatively toned cognitive activity in the second study, in the third study the instruction to be on call for one night might have led to negative cognitive activity: a participant fears to miss a sound or is apprehensive of the expected awakenings during the night. Following the cognitive model of insomnia (Harvey, 2002) negatively toned cognitive activity leads to a physiological arousal. In the three studies, we looked at the HR and/or HRV to have a measurement of physiological arousal. Against this first model, data of the first study showed no arousal, although participants reported rumination and worry. The third study showed the same lack of arousal. In the second study, we intended to create an arousal by using social media before sleep. Against our assumptions, participants showed no changes in objective stress responses after social media consumption. The cognitive model of insomnia assumes that negative beliefs about sleep exacerbate the negative cognitive activity in patients. The instruction to sleep worse might create such a belief known by insomnia patients (e.g. “I know I will not be able to sleep (well) tonight”). Although our participants were healthy sleepers, they were able to impair negative cognitive activity, even if this manipulation was not reflected by

a physiological arousal. Similarly, participants in the third study activated negative beliefs such as “I won’t sleep through the night” or “Tonight I will have disturbed sleep due to alarm sounds”. Although the data confirmed that negative cognitive activity leads to (emotional) distress and that negative beliefs worsens this, the model itself explains sleep deficits only for insomnia, which must meet the criteria that “the predominant complaint must be [...] for at least one month” (Harvey, 2002, p. 870). Therefore, the model explains the relationship between cognition and sleep in the long run, but not for an acute change.

This gap was filled by the hyperarousal model of insomnia (Riemann et al., 2010), which differs between acute (< 90 days), subchronic (3-6 months), and chronic insomnia (> 6 months). As predicted, results from the first and the last studies confirmed that rumination or worry led to a decreased subjective and/or objective sleep quality. Results of the first study corroborated the model: participants reported that they used strategies like rumination about the past day or worry about the future to prolong their SOL. Although not all sleep parameters were affected, neither the first nor the last study, participants showed sleep related problems predicted by this model due to participants worrying. Again, the lack of a physiological hyperarousal contradicts this model, which was shown in all of the three studies. Following this model, on a neurobiological level the acute cortical arousal does not show its consequences until a patient reaches the state of a subchronic insomnia. The state of an acute insomnia, which should explain the situation in our participants, only shows adaptive changes in the neurobiological component of this model. As our data did not include measurement of stress hormones (e.g. cortisol) or other neurotransmitters, we could neither confirm nor decline the neurobiological level (Spiegelhalder et al., 2011). Although this model differs between different subtypes of insomnia, the validity of this model is based on insomnia patients (Riemann et al., 2015). Moreover, independently from cortical hyperarousal, in the second study this model might explain the missing results of social media consumption on sleep: participants were able to stay in contact intensively with their social environment for 30 minutes and consequently did not worry about their peers and loved ones. The intention to worry about them is therefore countered, whereas this was not the case when participants handed in their smartphone right after arriving in the laboratory. On the other hand, someone might wonder how it was possible to show the beneficial results on sleep quality after PMR. Indeed, PMR is a method used in cognitive-behavioral therapy for insomnia (CBT-I), which is based on this model (Riemann et al., 2017). In insomnia patients, PMR had a reducing effect on both chronic and acute cortical hyperarousal (Morin, 2004). The second study confirmed these beneficial effects of PMR in healthy sleepers. Even if the hyperarousal model of insomnia (Riemann et al., 2010) differs

between different subtypes of insomnia, our results could not properly explain the relationship between cognitive factors and sleep using this model. More, other than cognitive and physiological (hyper-) arousal, is needed to explain this interrelationship.

Therefore, the MemoSleep hypothesis was introduced as a last model in this thesis. Contrarily to the first two models, this hypothesis explains the connection between cognition and sleep in healthy sleepers. In addition to the other two models, the MemoSleep hypothesis assumes that cognitive factors are able to affect sleep through (spontaneous) reactivation of embodied sleep/wake memories. Therefore, the intention to sleep worse in the first study, might activate wake memories (e.g. “stay awake”, “wake up during the night”), which then are spontaneously reactivated during the night. Although there were no increases in physiological arousal, sleep quality was still reduced, and participants felt less recovered in the morning showing decreased vigilance. The question to whether these results are based on an increased cognitive arousal remains open, as it was not measured in this study. As already mentioned, in the second study, the pre-sleep social media consumption decreased physiological arousal. Compared to the other two models, the MemoSleep hypothesis might explain this phenomenon: most probably, participants’ social media consumption was related to wishing their peers a good night of sleep. In so doing the content of their last messages before sleep was related to good sleep (e.g. “good night”, “sleep tight”). Following the MemoSleep hypothesis, this activates (embodied) sleep memories, reducing not only the physiological but also the cognitive arousal. In the relax condition, this effect was enhanced by PMR, therefore more sleep memories were activated during 30 minutes of PMR, showing the beneficial effects of PMR on sleep. It has been shown, that the body position affects the processing of sleep-related words (Hülsemann & Rasch, 2020). Due to the use of words like “relax” or “calm” during this 30-minutes-PMR-session, while lying in the bed, these words were embodied better, and the spontaneous reactivation of these sleep memories was enhanced. This provides an explanation for the results showing beneficial effects on different sleep parameters (e.g. reduced SOL, enhanced sleep efficiency). This positive result of PMR was still visible in the morning, when participants felt more awake and better recovered. The results of the third study were also in line with the MemoSleep hypothesis. The instruction to be on call, might help to activate wake-related cognition and embody wake memories (e.g. “wake up” or “on call”). During the night, even though alarm sounds were not presented in both groups, the instruction to wake up might have stayed active during the night, and was shown by the effect of instruction in different sleep parameters (e.g. WASO, NWAK). When sounds were present, this could be considered as targeted reactivation, as before sleep the alarm sound was presented together with the

instruction to be on call. However, even though there was a combined presentation of sound and instruction in the evening, changes in ERP depended on the instruction on call and not the actual presence of sounds. This is in line with the assumption that the "wake up"-instruction is an embodied wake-memory and is reactivated during the night, even though participants do not wake up after a sound. One step ahead, a study reactivating sleep memories during the night showed beneficial effects on subjective and objective sleep quality (Beck et al., 2020). In their study, they showed that reactivated embodied concepts related to sleep and relax are capable of influencing sleep. One explanation for these results are multimodal networks, where embodied cognition and the semantic meaning are stored (Shapiro, 2014).

Taken together, the three studies of this thesis appear to be in line with the assumptions of the MemoSleep hypothesis, i.e., that the activation of pre-sleep cognition are embodied in sleep/wake memories, which are reactivated during the night. It seems that the cognitive arousal before sleep in healthy sleepers is important for ongoing processes during sleep (e.g. memory reactivation). In the next section, this thesis takes a closer look at the practical implications of these findings before passing on to a description of the limitations. As the first two models are models for insomnia, some might wonder what the implications of these studies are for patients suffering from insomnia. Are there practical implications for patients or their treatment?

4.2 What are practical implications?

The three studies showed that healthy sleepers were able to affect sleep. For the treatment of insomnia, the results from the first study seem interesting, as participants were able to induce a state of acute insomnia. Therefore, the first study helped to provide a better understanding of the interrelationship between cognition and sleep. Here we have seen that healthy sleepers were not able to affect sleep quality in a positive direction. Good intention alone is not enough to enhance sleep quality – even in healthy sleepers. Considering this information in the case of patients, it is clear that they need more than just willpower. Indeed, for insomnia patients it seems hard to find a way out of insomnia without an accurate treatment (e.g. cognitive therapy for insomnia ((CBT-I), see Crönlein et al., 2020; Morin et al., 1999; Perlis et al., 2011; Riemann & Perlis, 2009). One explanation is given by the second study, confirming not only the benefits of PMR (Low et al., 2020; McCallie et al., 2006; Walters et al., 2003) but also underlining the missing negative effects of social media consumption right before sleep. Insomnia patients often have false beliefs about sleep. Results of the second study

helped in the treatment of these beliefs as it explains that pre-sleep social media consumption does not have the expected negative effects on sleep. Some patients might benefit of this information to reduce their FoMO, which actually could decrease the arousal (Kater & Schlarb, 2020; Scott & Woods, 2018). More concretely this means that a person who likes to know about their peers and who likes to be connected before sleep, could implement a last check of social media before going to bed without the expectation of negative effects. However, most beneficial for insomnia patients are results of the last study. These showed that the activation of an on call instruction in the evening helps to stabilize or destabilize sleep. Adapting from these results, we could help insomnia patients by giving them an instruction before sleep. If a patient complains about waking up during the night due to slight noises and not being able to fall back asleep, the pre-sleep instruction that during this night there will not be any disturbances or sounds, might reduce the actual wake periods.

Further results of the first and the last study confirmed a misperception between subjective and objective sleep parameters in healthy participants. Especially in the first study, results showed a massive difference between subjective and objective sleep quality, which is seen in insomnia patients as well (Maes et al., 2014; Manconi et al., 2010). Some might argue this misperception is because participants like to fulfill the experimenter's expectations. This was not confirmed by the results of the first study, as it seems unrealistic to follow the experimenter's expectations in one but not the other condition: the misperception only occurred in the instruction to worsen sleep but not in the instruction to increase sleep quality. One parameter that allows a direct comparison was SOL, as subjective and objective values both were rated in minutes. Subjectively, participants rated their time to fall asleep two times higher as measured objectively when they were instructed to sleep badly. For the other two conditions, the two parameters were almost the same. One explanation is that participants were actually able to reach the state of an (acute) insomnia due to the instruction and the pre-sleep cognitive activity. One aspect of a therapy for insomnia is therefore to reduce the SOL, which was seen after PMR in our second study. In this study, the misperception between subjective and objective sleep parameters was negligible. Results of this study did not show a negative effect of pre-sleep cognitive activity. These results help to provide a better understanding to the fact that a misperception of sleep is an important sign of reduced sleep quality and insomnia. In the last study, the misperception was seen especially in the NWAK, where participants underestimated their values especially in the on call-nights. To conclude, the misperception presents itself as a marker of reduced sleep quality and therefore should be considered in the therapy of insomnia.

One last practical implication that deserves further attention is the importance of pre-sleep instruction. Especially in the first and in the last study, the instructions produced significant effects. Although in the first study, the instruction to enhance sleep quality did not affect sleep, the instruction to sleep badly showed the expected effects. In the last study, the instruction to be on call was more important than the actual presence of alarm sounds. Therefore, it would be interesting to see if this instruction would be enough to change sleep quality e.g. in a person affected by noise pollution. For example, as one practical implication of the third study, pre-sleep instructions might help people who live near church bells. All over Switzerland church bells ring every (quarter) hour, day and night. Therefore, the association of wake memories with the sound of a church bell might disturb sleep in many people. From results of the third study, we already know that a sound is perceived differently if a person is or is not instructed to hear it. During the neutral night, in this study, participants showed sleep-providing reactions to sounds. After an alarm sound was presented, participants showed lower amplitudes in ERP in frequency bands similar to K-complexes, than when they were instructed to be on call. Indeed, it has been shown that K-complexes have a sleep-protecting function (Cash et al., 2009; Halász, 2016). Does this mean that these ERPs were protective for sleep? So far, this question remains unanswered. What the results of this third study confirmed is that the instruction to be on call seemed to remain active during the night. These results are highly relevant as on call-work gains growing interest in our society (Roberts et al., 2019; Vincent et al., 2021). As the results of this last study not only showed effects of on call-work but of pre-sleep instructions as well, the implications are relevant for even more people and more research is needed in this field. What does this result mean for the church bells? Conversely, instructing people not to hear the church bell during the night, or emphasizing its non-disruptive character might help. However, not only individuals who live near church bells suffer of disrupted nocturnal sleep. A person living or sleeping next to a railway or a road is also affected by noise during the night. Noise pollution is a severe problem, and during the night 1 in 8 people in Switzerland complain about noise exposure (Bundesamt für Umwelt BAFU, 2020; European Environment Agency, 2021) thus finding a solution is very important. Is it enough to tell the person that no train will be perceived, or no hooting of a horn will be heard? This question remains open and further research is needed, for example by looking at the exact effects of instructions.

4.3 Are there limitations?

Although the results of this thesis confirmed the importance of cognition on sleep, there are still some limitations and research gaps left open. We have seen that pre-sleep cognitive activation seems highly relevant for sleep quality. Even if this thesis showed how to worsen sleep by pre-sleep cognitive activation, the opposite is not possible. This would be of high relevance as the interest in enhancing sleep quality – instead of worsening it – seems even more important. Is it possible to enhance sleep quality positively with cognitive factors? This thesis cannot answer this question. One mechanism is suggested by the MemoSleep hypothesis (Rasch, 2016), as it expects beneficial effects of targeted memory reactivation of sleep memories during the night, which seems to be a promising approach (Beck et al., 2020).

Another limitation of this thesis is that its results were based on healthy sleepers. Although it is important to modify sleep in a healthy population with no known sleep disorders, the application of these results to a (sub-)clinical population might be helpful. In upcoming research, it would be interesting to see how cognition affects sleep in patients with sleep disorders such as insomnia. Especially for the first study, the transposition to a (sub-)clinical population is interesting to see if patients are able to worsen sleep as well, or if the same instruction might even lead to better sleep quality. Are the instructions enough to see beneficial effects? What strategies would patients implement as they are used to rumination and worry in the "sleep badly" condition? Could the instruction to sleep badly have beneficial effects in this population? So far, we do not know the answer to these questions and future research should focus on applying results of this thesis to a (sub-)clinical population suffering from insomnia or other sleep disorders.

Apart from insomnia patients, the subpopulation of elderly people might be interested in these beneficial results on sleep quality as well. In the three studies of this thesis, the results were based on young participants (< 35 years). As sleep changes with age (Foley et al., 1995; Ohayon et al., 2004), there is a lack of understanding of cognitive effects on sleep in the elderly. Although it has been shown that elderly participants benefit from cognitive influence via hypnosis (Cordi et al., 2015), the aim of this thesis was to look at the interrelationship between cognitive factors and sleep directly. It would be interesting to see if the results of the three studies would differ in elderly compared to the young participants. So far there is a lack of studies looking at the interplay of pre-sleep cognition and sleep quality in elderly sleepers.

One important question that remains unclear regarding the MemoSleep hypothesis (Rasch, 2016) is which sleep/wake memories are reactivated during sleep, and which are not? For example, in the first study on the one hand it seems as if the wake memory "wake up" was

reactivated thereby inducing participants to wake up more often. On the other hand, the sleep memories “light sleep” or “deep sleep” were not reactivated during the night. Participants were not able to increase or decrease neither light nor deep sleep when instructed to do so. It would seem as if not all sleep/wake memories are reactivated during the night. So far, studies have posited that reactivation is not selective to content, but on the contrary, spontaneous (O’Neill et al., 2010; Oudiette & Paller, 2013; Rasch & Born, 2013), which only partly seems to be the case in this study as well. One assumption might be that reactivation is not as independent of the context of the sleep/wake memory. It might be possible that the wake memory “wake up” in the first study or the embodied instruction “on call” in the last study were more important than other sleep/wake memories. One explanation for this bias in reactivation might be that some sleep/wake memories (e.g. “wake up”) are associated with life protecting factors, thereby giving them a higher relevance (e.g. Bruck, 2001). In the third study, this increased relevance was given by the presentation of alarm sounds in addition to the instruction to be on call. It has been shown that targeted memory reactivation is beneficial for memory consolidation (Cellini & Capuozzo, 2018; Hu et al., 2020; Lewis & Bendor, 2019). This concept is included the MemoSleep hypothesis (Rasch, 2016) to enhance e.g. beneficial effects of embodied sleep memories by targeting their reactivation. Here, some might wonder if the presentation of alarm sounds was sufficient. Consequently, in the third study, the presentation of alarm sounds might have figured as a targeted memory reactivation of the wake memories “wake up” and “on call”. However, results showed that the instruction to be on call was more important than the actual presence of alarm sounds during the night. Therefore, it seems reasonable that the spontaneous reactivation was not targeted and thereby enhanced by alarm sounds. Thus, the exact mechanisms by which sleep/wake memory are selected to be reactivated still remains unclear and further research in this field is needed.

4.4 General conclusion

To conclude, it is possible that pre-sleep cognition affects sleep, not only in patients with insomnia, but also in healthy sleepers. In particular, pre-sleep cognitions seem very important, which is what is proposed by the MemoSleep hypothesis (Rasch, 2016). Although it was not possible to enhance sleep quality in any of the studies, the opposite was possible. Therefore, it did not matter if it was the instruction to sleep worse or to follow on call-duty for one night, results showed that the instruction stayed active during the night. Furthermore, participants were able to create and/or use strategies to deal with pre-sleep social media consumption and it did not affect sleep negatively.

This thesis provides a first glimpse relative to the importance of the MemoSleep hypothesis. Although previous models of insomnia (the cognitive model of insomnia (Harvey, 2002) and the hyperarousal model for insomnia (Riemann et al., 2010)) explain the interrelationship between cognitive functions, physiological (hyper-)arousal, and sleep in insomnia patients, the here introduced MemoSleep hypothesis (Rasch, 2016) helps to understand this connection in healthy sleepers as well as clinical (sub-)populations as well. The latter not only considers the content of pre-sleep cognition, but additionally differs between sleep and wake memory. The three studies not only showed that it is possible to reduce sleep quality, but that it is also possible to enhance it – using different techniques. Moreover, this thesis helps provide a better understanding on how processing during the night takes place, and that pre-sleep instructions are maintained during sleep thereby affecting its quality.

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6 Appendix

6.1 Declaration of Authorship

I declare that this thesis has been composed solely by myself and that it has not been submitted, in whole or in part, in any previous application for a degree. Except where states otherwise by reference or acknowledgment, the work presented is entirely my own.

Selina Ladina Combertaldi
Zofingen, October 5, 2021

6.2 Curriculum Vitae

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Education

2016 – present	Ph.D. Student in Psychology • *Institution*: Department of Psychology, Division of Cognitive Biopsychology and Methods, University of Fribourg, CH • *Title*: Influences of Cognition on SleepI: How (pre-sleep) manipulation affects nocturnal me sleep • *Supervisor*: Prof. Dr. rer nat. Björn Rasch
2014 – 2016	M.Sc. in Psychology – *cum laude* • *Field*: Neuropsychology; Clinical Psychology & Therapy • *Institution*: University of Bern, CH • *Title*: Web-based Memory Test for Accelerated Long-Term Forgetting (ALF) • *Supervisors*: Prof. Dr. phil. Clemens Gutbrod, Prof. Dr. phil. Beat Meier
2014 – 2016	Technical Research Assistant • *Institution*: Schlaf, Wach und Epilepsie Zentrum (SWEZ), Neurology, Inselspital University Hospital, Bern, CH • *Supervisors*: Dr. rer. nat. Corinne Roth, Prof. Dr. med. Claudio L. Bassetti
2014-2015	Teaching Assistant for Introduction in Psychology • *Institution*: Cognitive Neuroscience of Memory and Consciousness, University of Bern, CH • *Supervisor*: Prof. Dr. phil. Katharina Henke
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Funding & Grants

2019 SNF Scientific Exchanges for organizing the Workshop for Young Sleep Researchers. **10’700.- CHF**

Publications

Combertaldi, S.L. & Rasch, B. (2020). Healthy sleepers can worsen their sleep by wanting to do so: The effects of willpower on objective and subjective sleep parameters. Nature and Science of Sleep.

Combertaldi, S.L., Cordi, M. J., Ort, A., Fahr, A., & Rasch, B. (2021). Pre-Sleep Social Media Use does not strongly disturb Sleep: A Sleep Laboratory Study in healthy young participants. Sleep Medicine.

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Baselgia, S., **Combertaldi, S.**L., Ort, A., Wirz, D., Fahr, A., & Rasch, B. (in preparation). Netflis, Cliffhangers and Sleep: How Binge-Watching affects sleep.

Combertaldi, S.L. & Rasch, B. (in preparation). Evaluation and Validation of the Sleep Beliefs Questionnaire (SBQ).

Conference Papers

Renevey P., Delgado, R., Lemkaddem, A., Verjus, C., **Combertaldi, S.L.**, Rasch, B., Leeners, B., Dammeier, F., Kuebler, F. (2018). Respiratory and cardiac monitoring at night using a wrist wearable optical system. *Annual International Conference of the IEEE Engineering in Medicine and Biology Society, IEEE Engineering in Medicine and Biology Society, Conference 2018:2861-2864, DOI: 10.1109/EMBC.2018.8512881.*

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Stress, binge-viewing and artificial light: How is our modern life-style related to sleep? (June 2020). Organized by **Combertaldi, S.L.** with Blume, C. (Psychiatric Hospital of the University of Basel, Switzerland), Rasch, B. (University of Fribourg, Switzerland), **Combertaldi, S. L.** (University of Fribourg, Switzerland), and Schwarz, J. (Stockholm University, Sweden). *Symposium accepted for the Psychologie und Gehirn, Freiburg i.Br.*

Emotion, binge-viewing and artificial light: How is our modern life-style related to sleep? (October 30, 2021). Organized by **Combertaldi, S.L.** with Eti Ben Simon (UC Berkeley, USA), **Combertaldi, S. L.** (University of Fribourg, Switzerland), Baselgia, S. (University of Fribourg, Switzerland), and Blume, C. (Psychiatric Hospital of the University of Basel, Switzerland). *Symposium at the 29th DGSM annual meeting 2021, Freiburg i.Br.*

Conference Talks

Combertaldi, S.L., Ort, A., Fahr, A., & Rasch, B. (2020, October). The effect of Pre-Sleep Social Media Consumption on Sleep Quality. *Talk delivered for* the *Binge-Watching Meeting, Fribourg.*

Combertaldi, S.L., Wick, A., & Rasch, B. (2019, June). Sleep and On Call situations – How On Call Situations affect our night sleep. *Talk delivered within the Symposium "Just in your head? Effects of suggestions and mental imagery on perception and appraisal" at the Psychologie und Gehirn, Dresden.*

Combertaldi, S.L., Ort, A., Fahr, A., & Rasch, B. (2019, May). The effect of Pre-Sleep Social Media Consumption and Relaxation on Sleep Quality and Memory Consolidation. *Talk delivered for* the *69th Annual International Communication Association (ICA) Conference, Washington, D.C.*

Combertaldi, S.L. (2017, June). I want to Sleep Better. *Talk delivered for JuWi-Posterblitz at the Psychologie und Gehirn, Trier.*

Conference Posters

Combertaldi, S.L., Ort, A., Fahr, A., & Rasch, B. (2019, October). Influence of Pre-Sleep Social Media Consumption on Sleep. *Poster presented at the Annual Meeting of the SSSC, Fribourg.*

Combertaldi, S.L., Ort, A., Fahr, A., & Rasch, B. (2018, October). Influence of Pre-Sleep Social Media Consumption on Sleep. *Poster presented at the Annual Meeting of the Society for Psychophysiological Research, Quebec City.*

Combertaldi, S.L. & Rasch, B. (2018, June): Influence of Pre-Sleep Social Media Consumption on Sleep (Preliminary Data). *Poster presented at the Psychologie und Gehirn, Giessen.*

Combertaldi, S.L. & Rasch, B (2017, May): I Want to Sleep Better: Effects of Voluntarily Control on Objective Sleep Parameters. *Poster presented at the Psychologie und Gehirn, Trier.*