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Sleep On It:

The Effect of Daytime Napping on Emotional Reactivity

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> 1 In the beginning, God created the heavens and the earth. 2 The earth was without form and void, and darkness was over the face of the deep. And the Spirit of God was hovering over the face of the waters. 3 And God said, “Let there be light,” and there was light. 4 And God saw that the light was good. And God separated the light from the darkness. 5 God called the light Day, and the darkness he called Night (English Standard Version Bible, 2001, Genesis 1:5).

Recognized and observed by human civilization for thousands of years, the distinction between day and night is one of the most fundamental divisions of time. Essential for survival to constantly adjust to alterations in one’s external environment, the dichotomy of day and night profoundly shaped the evolution of every living being. The adaptation is reflected in a natural cycle ubiquitous in every living organism, referred to as the circadian rhythm. It comprises an internal biological clock orchestrating cycles of wakefulness and sleep in living organisms in a 24-hour rhythm.

While the adaptation to changing daylight likely dates back to ancient times, the scientific study of sleep is yet in its relatively early stages of development. Formerly regarded as a passive state of mind, in which the organism shuts down to conserve energy, the conceptualization and investigation of sleep experienced a transformation with the groundbreaking invention of the electroencephalogram (EEG) by Hans Berger. With his invention, in 1929 Hans Berger was the first to observe that brain activities during sleep differed from those during wakefulness rendering the theory of sleep as a passive state obsolete. Berger’s EEG marked a turning point in sleep research, as it enabled researchers to objectively measure and record brain activity. Endowed with a means to objectively measure and record brain activity ensuing studies yielded significant advancements in the research on sleep, i.e., identifying a sleep architecture and different sleep stages, and pathed the way for a

comprehensive manual colloquially referred to as the “bible of sleep”: The AASM Manual for the Scoring of Sleep and Associated Events (AASM). The AASM serves as one of the most essential references in sleep research, as it provides standardized guidelines for the scoring and interpretation of different cyclic sleep stages.

Far from a simple absence of wakefulness, sleep is nowadays recognized as a brain state marked by a large-scale organization of neuronal activity comprising virtually all brain areas. For a closing definition of the phenomenon of sleep, one has to turn towards its behavioural features. Following Anafi et al. (2018), there are three behavioural criteria constituting the main pillars of sleep. The first two are a diminished responsiveness to weak stimuli and a quick reversibility in response to strong stimuli. Another principal element is sleep homeostasis, a natural regulatory mechanism that governs the need for sleep depending on the amount of wakefulness the organism has experienced. (Anafi et al., 2018).

1.1. Sleep Physiology

Sleep and wake states are regulated by two main factors: the circadian rhythm and sleep pressure (Baranwal et al., 2023). The circadian rhythm repeats itself roughly every 24 hours and controls large portions of behavioural and physiological processes, e.g., eating, drinking body temperature, neurohormone secretion and sleep-wake-cycles (Baranwal et al., 2023, Vasey et al., 2021). The circadian rhythm is driven by both external and internal signals, which are referred to as “zeitgebers” (Baranwal et al., 2023). In addition to social cues, nutrient availability, and temperature, the primary external influence on the circadian rhythm is light and dark exposure (Vasey et al., 2021).

Located within the hypothalamus of the brain in mammals sits the master pacemaker of the circadian rhythm, i.e., the suprachiasmatic nucleus (SCN). Owed to its position atop the optic chiasm – the crossing of the optic nerves in the middle of the brain – the SCN

directly receives photic input from the retina, allowing it to synchronize the organism with the light and dark cycle (Welsh et al., 2010). This synchronization is achieved by propagating information on changes in daylight to the brain and body via increased stimulation of the pineal gland in response to darkness. The pineal gland in turn produces and releases the neurohormone melatonin into the bloodstream, which triggers physiological events that promote the onset of sleep (Cajochen et al., 2003). However, it should be noted that even in the absence of light/dark cues the SCN produces a consistent output signal through internal coupling, resulting in an autonomously functioning circadian rhythm that coordinates physiology and behaviour (Welsh et al., 2010).

Besides the circadian rhythm, sleep pressure influences the initiation of sleep. Sleep pressure is tracked via the neurotransmitter adenosine, which accumulates during wakefulness. Adenosine decreases the activity of ascending arousal systems that promote wakefulness by inhibiting neurons of the ventrolateral preoptic area, thereby serving as a sleep switch (Carley & Farabi, 2016).

1.2. Sleep architecture

Compared to other mammalian species humans occupy a special position in terms of sleep patterns. Whilst the majority of mammalian species are polyphasic sleepers, sleeping for multiple short episodes within 24 hours, humans belong to the minority of monophasic sleepers or biphasic sleepers. Monophasic sleep is characterized by a single, prolonged period of natural sleep, whereas biphasic sleep refers to those including an afternoon nap in the routine.

Sleep in itself is not uniform. A broad division in mammalian sleep can be drawn between non-rapid eye movement (NREM) sleep and a stage of rapid eye movement (REM) sleep (Walker & van der Helm, 2009). Rechtschaffen & Kales (1986) subdivided NREM

sleep into four substages, i.e., N1, N2, N3, N4, corresponding, in that order, to increasing depth of sleep. This classification was updated by the AASM (2007) in which N3 represents both substage 3 and substage 4 combined. Each sleep stage possesses distinct functions in maintaining the cognitive processes of the brain and facilitating bodily repairs. Nonetheless, the optimal quality of sleep is considered to arise from the interaction among these sleep stages, in combination with the number of sleep cycles (Baranwal et al., 2023).

Fluctuations of distinct sleep stages can be observed throughout the night. The organization and cyclic pattern in which the different sleep stages occur is typically denoted as sleep architecture (Figure 1.1). In healthy adults, the first sleep cycle starts with sleep stage N1, followed by N2, N3 and REM sleep. Each sleep cycle lasts approximately 90 min and is repeated 4-5 times throughout an 8-hour night of sleep (Baranwal et al., 2023). A disruption in sleep architecture entails a reduction of the restorative benefits of sleep. The ratio of N3 and N4, N2 and REM change throughout the night. Deep sleep, stage N3 and N4 sleep predominates during the first half of the night, then decreases exponentially. Stage N2 and REM sleep in contrast prevail during the latter half of the night (Walker & van der Helm, 2009).

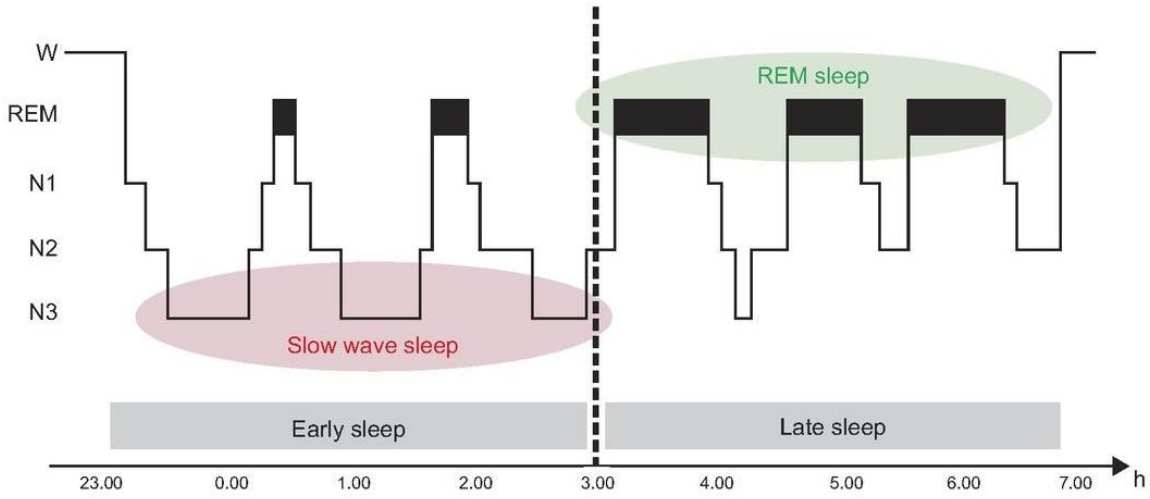

Figure 1.1. Hypnogram of a Healthy Subject. Throughout the course of a night's sleep, the composition of sleep stages changes. Initially, there is a prevalence of slow-wave sleep, whereas in the latter half of the night, REM sleep takes prevails. Image taken from Rasch & Born (2013).

1.3. Sleep electrophysiology.

Providing valuable insights into sleep patterns, sleep stages and disruptions, polysomnography (PSG) is the gold standard for the assessment of sleep. The term accounts for a multiparametric test which records an array of physiological parameters during sleep. It includes the EEG for brain activity, electromyogram (EMG) for muscle tone, electrooculogram (EOG) for eye movements and electrocardiogram (ECG) for the heart's rhythm (Adamantidis et al., 2019). Based on characteristic rhythms and events recorded with the PSG, the distinct stages of sleep can be identified and scored.

1.3.1. Wakefulness

The state of wakefulness spans the continuum from full alertness to the initial stages of drowsiness (AASM, 2012). Alert wakefulness is characterized by mixed-frequency low-amplitude EEG signal, i.e., beta waves, rhythms peaking in the 14-25 Hz range, and alpha waves, comprising the 8-13 Hz range. Alpha waves are most prevalent in drowsy wakefulness and with eye closure (Carley & Farabi, 2016). With drowsiness, slow rolling eye movements can be observed with EOG recordings (Carley & Farabi, 2016). The amplitude of chin EMG varies during wakefulness, typically being higher compared to sleep stages (AASM, 2012).

1.3.2. NREM N1

Stage N1 indicates the transition from wakefulness to sleep. When humans fall asleep, their EEG undergoes an initial shift from high-frequency, low-voltage waves during wakefulness to slower waves with higher voltage, indicating the onset of NREM sleep (Walker & Van der Helm, 2009). Marked by the attenuation of the alpha rhythm, the predominant neural oscillations during N1 sleep become theta waves, rhythms peaking in the

4-8 Hz range (Adamantidis et al., 2019). The onset of NREM sleep is often accompanied by hypnagogic hallucinations reminiscent of the dreams that occur during REM sleep (Adamantidis et al., 2019). During this stage, EOG recordings can pick up slow, sinusoidal eye movements with an initial deflection lasting > 500 msec (AASM, 2012). Compared to wakefulness, the chin EMG amplitude during stage N1 is lower, but still variable (AASM, 2012).

The duration of N1 sleep is typically between one and seven minutes. A person can still easily be woken up during this sleep stage. However, if not disturbed, one can move quickly into N2 sleep (Carskadon & Dement, 2011).

1.3.3. NREM N2

Stage N2 sleep is considered light sleep. During the first sleep cycle, N2 sleep stage normally lasts between 10 to 25 minutes, its duration increases with the progression in sleep cycles. Typically, the time spent in N2 sleep amount to 50 per cent of total sleep time (Carskadon & Dement, 2011). In this stage, the body enters a calmer state, including muscle relaxation, as well as slowed breathing and heart rate (Carskadon & Dement, 2011). Eye movements stop (AASM, 2012) and the brain activity slows down, except for some short bursts of activity (Carskadon & Dement, 2011). Brain activity during this stage is characterized by two widely investigated hallmarks of sleep on a background of theta activity, which are intermittent sleep spindles and K-complexes.

Sleep spindles are waxing and waning oscillations in the 11-16 Hz range with a total duration of ≥ 0.5 seconds, usually maximal using central derivations (Adamantidis et al., 2019). With regards to their topographic distribution and functional significance, sleep spindles can be further divided into two types: slow spindles at 11–13.5 Hz and fast spindles at 13.5–16 Hz (Antony & Paller, 2016). Slow spindles are located over frontal electrode sites,

whereas fast spindles are located over centroparietal regions and have been preferentially associated with memory consolidation during sleep (Page et al., 2018). Located in the nucleus reticularis of the thalamus, a network of GABAergic thalamic neurons is responsible for the generation of sleep spindles. Spindles are then propagated across the cortex, by thalamocortical fibres that project to central and posterior areas of the brain (Klinzing et al., 2019). Sleep spindles occurring during N2 sleep have been related to memory consolidation, but also be particularly responsible for potentiating procedural motor learning (Marshall et al., 2020).

The K-complex is a well-delineated, sharp wave lasting ≥ 0.5 seconds, with a spatial distribution predominately over frontal region (Adamantidis et al., 2019). The K in K-complex refers to knock, as they were first observed in response to auditory stimuli or modality-independent stimulation during NREM sleep in humans (Adamantidis et al., 2019). Despite their initial discovery, subsequent studies noticed that in addition to stimulus-evoked, K-complexes can also occur spontaneously in response to an internal stimulus, e.g., interruption in respiration (Jafari, 2017). As described by Laurino et al. (2014) the complex is characterized by 3 distinctive waves, i.e., a short positive voltage deflection, peaking at around 200 ms, with a subsequent large negative complex, usually at 550 ms, and lastly a long-lasting positive component, with the conventional peak at 900 ms. However, the whole complex can also be biphasic, when the first positive voltage peaking around 200 ms is undetectable or unnoticed (Laurino et al., 2014). This alternation between positive and negative voltage deflections represents states of neuronal depolarization (up states) and hyperpolarization (down states), respectively (Laurino et al., 2014). Similar to sleep spindles, K-complexes are considered to be produced following hyperpolarization of thalamo-cortical circuits by activation of GABAergic pathways (Amzica & Steriade, 1997).

Although the functional significance of K-complexes is unclear, two explanatory approaches are currently under debate. The first associates the phenomenon with arousal, given their co-occurrence with increased autonomic activation, such as heart rate, and the other with sleep promotion (Colrain, 2005). As proposed by Halász (2016) the functional implications of K-complexes might depend on the slope of the sleep cycle: The occurrence of K-complexes initiates an arousal-pattern the during ascending slope (from stage 3 towards stage 1 and REM sleep), whereas during the descending slope (from stage 1 towards stage 4) it promotes sleep by entailing a single slow wave or a series of them including sleep spindles.

Even though K-complexes play a role in evoking sleep spindles, this relationship remains to be clarified entirely, as studies yield results, on the one hand corroborating an inverse relationship (Curcio et al., 2003), on the other a positive correlation (Reynolds, 1988).

1.3.4. NREM N3

The N3 sleep stage comprises stages S3 and S4 and is often referred to as slow wave sleep (SWS). Marking the deepest NREM sleep stage, it is considered to be the most restorative period of sleep. This sleep stage is mainly characterized by the presence of high-amplitude, slow oscillations (<1 H; SOs) and delta waves (1-4 Hz), the latter indicating an underlying mass cortical synchrony (Adamantidis et al., 2019, Amzica & Steriade, 1995). Together with SOs, spindles (11-16 Hz) and ripples (80-140 Hz) are yet two more prominent microstates present during SWS. Additionally, large waves with a peak-to-peak amplitude of >75 µV can be detected, typically over the frontal regions (AASM, 2012).

Predominately generated by neocortical networks, prefrontal areas in particular, SOs represent neuronal network alternations between down- and up states, with concomitant synchronized membrane hyperpolarization and depolarization, respectively (Klinzing et al., 2019). In particular, SOs manifest a (shorter) down-state with synchronized membrane

hyperpolarization and extensive neuronal silence, accompanied by an up-state that varies in length with synchronized membrane depolarization and markedly increased firing of cortical neurons. These biphasic rhythms reflect enhanced activity of both excitatory and inhibitory neurons of the cortex. SOs are usually solely estimated on grounds of frequency and amplitude, given the EEG's incapability to measure these alterations in firing rate (Klinzing et al., 2019).

A body of findings corroborates the causal relation between SOs and memory consolidation (Atherton et al., 2015, Pavlides & Winson, 1989, Wilson & McNaughton, 1994). For instance, transcranial direct current stimulation using slow (0.75 Hz), but not fast (5 Hz), oscillatory stimulation seems to be able to improve hippocampus-dependent memory consolidation (Marshall et al., 2006). The explanatory mechanism behind the improvements in memory consolidation is assumed to be synaptic downscaling (Diekelmann & Born, 2010). Synaptic downscaling describes the adjustment of synaptic connections in a way that makes them sustainable with regard to energy and tissue volume. As a result, weak connections are eliminated, while the relative strength of the remaining connections is maintained. Thus, the memory is enhanced by improved signal-noise ratio after downscaling (Tononi & Cirelli, 2006). The amplitude of SOs is taken as an index for the synaptic strength between cortical neurons. Across the SWS cycle, the amplitude of SOs decreases, showing a maximum amplitude right at the beginning of sleep, which then decreases as a result of a gradual synaptic depotentiation (Diekelmann & Born, 2010). Likewise, studies showed an increase in amplitude and slope of SOs during SWS following a specific learning experience, whereas the prevention of information encoding yielded a decrease (Diekelmann & Born, 2010).

Besides the "classical" sleep spindles detectable during N2, spindles (11-16 Hz) are yet another prominent neural oscillation during SWS (Varela et al., 2013). Ever since initial studies correlated memory retention with concurrent spindle activity, they have been widely

investigated for their role in memory consolidation (Clemens et al., 2005, Gais et al., 2002). However, it is possible that spindles during SWS and stage N2 sleep contribute differently to the consolidation of memory types. In contrast to spindles in N2 sleep stage, which are associated with the potentiation of procedural memory, SWS spindles seem to be specifically related to declarative memories (Cox et al., 2012).

Albeit spindles and SOs can be observed independently, they often co-occur. Although predominantly referred to as one entity, slow and fast spindles differ with respect to their occurrence during SOs. Specifically, the up-state (depolarization phase) of SOs triggers the occurrence of fast spindles, whereas slow spindles most commonly emerge during the up-to-down slope up-state of SOs (Mölle et al., 2011). Coupling to SOs is especially strong for fast spindles (Cox et al., 2017). The synchronization of spindles and SOs points towards an underlying unified thalamo-cortical generating system (Contreras et al., 1996, Mölle et al., 2011).

The third oscillatory phenomenon of SWS are ripples (80-140 Hz). They are described as fast depolarizing events in conjunction with a so-called sharp-wave (Brodt et al., 2023). Specifically, the interaction between hippocampal structures gives rise to the observed oscillatory event. Divided into three layers (CA1 to CA3), spontaneous activation of pyramidal cells from layer CA3 is propagated to CA1, where a massive activation of CA1 pyramidal cells is provoked (Girardeau & Lopes-dos-Santos, 2021). The sharp wave emerges from CA3 pyramidal cells, the ripples from an interplay between pyramidal cells in CA1 and interneurons (Buzsáki, 2015). Interestingly, apart from the hippocampus during SWS, ripples can also be identified in the neocortex and entorhinal cortex during quiet wakefulness (Axmacher et al., 2008, Dickey et al., 2022, Khodagholy et al., 2017).

In addition to the well-established roles of SOs and spindles in memory formation, a crucial function in memory consolidation has been assigned to ripples. Ripples are closely

intertwined with the reactivation of neuron ensembles that showed activity during a preceding wake experience (Diba & Buzsáki, 2007). Consequently, it has been proposed that hippocampal ripples are critically involved in the coordinated reactivation of networks of widespread brain networks after learning (Marshall et al., 2006). Furthermore, since ripples also happen to temporally coincide with both SOs as well as spindles (Maingret et al., 2016) it has been suggested that the temporal convergence of SOs, spindles, and ripples facilitates memory consolidation. Just as (fast) spindles tend to nest within the up-state of neocortical SOs, ripples - concomitant with hippocampal network reactivations – nest in the troughs of spindles, resulting in the formation of spindle-ripple events (Staresina et al., 2015). The triple coupling is assumed to mediate the dialog between hippocampus and neocortical networks by facilitating the transfer of reactivated information into the corresponding neocortical network representing long-term storage (Brodt et al., 2023).

With regards to the first sleep cycle, 20 to 40 minutes are commonly spent in N3 stage sleep. With proceeding sleep cycles, the duration of N3 sleep shortens and is instead replaced by REM sleep (Carskadon & Dement, 2011).

1.3.5. REM

Originally called “paradoxical” sleep, REM sleep is a distinctive phase of the sleep cycle characterized by a unique interplay between brain activity and physical state. During REM brain activity closely resembles that during wakefulness, while the body undergoes skeletal muscle atonia, a temporary paralysis affecting most muscles, except for those responsible for breathing and eye movement. Despite the overall muscle atonia, intermittent muscle twitches can occur during this sleep stage (Peever & Fuller, 2017). In contrast to previous sleep stages, a distinct hallmark of REM sleep is the presence of periodic bursts of rapid eye movement, which gives it its infamous name. Although dreams can equally occur

during various sleep stages, REM sleep is the stage of sleep predominantly associated with dreaming (McNamara et al., 2010).

One peculiarity in terms of REM sleep is that it does not occur during the first 90 minutes of sleep. As the night progresses, REM stages lengthen, particularly during the second half of the night. While the initial REM stage might only last a couple of minutes, subsequent stages can last for about an hour (Carskadon & Dement, 2011).

Brain activity during REM sleep is hallmarked by a dominant theta rhythm (4-7 Hz), manifesting itself in the cortex and other subcortical structures, and indicating a prominent feature of hippocampal activity (Girardeau & Lopes-dos-Santos, 2021). Theta oscillations are accompanied by high-frequency activity in the 30 to 80 Hz gamma band range (Llinás & Ribrary, 1993; Steriade, Amzica & Contreras, 1996). During REM either sharp theta waves (sawtooth waves) - triangular-shaped rhythmic oscillations between 2-4 Hz with a most prominent deflection at frontocentral electrodes (Simor et al., 2020) - or wake-like EEG patterns can be observed (Carley & Farabi, 2016).

Another defining waveform of REM sleep, coinciding with bursts of rapid eye movement and twitching in distal muscles (and phasic theta rhythms) are ponto-geniculo-occipital waves, commonly referred to as PGO waves or P-waves (Hong et al., 2008). PGO waves arise from the brainstem (Mukai & Yamanaka, 2023) and as their name already implies, they can be recorded from the pons, the lateral geniculate, and the occipital cortex. Yet, more recently it has been suggested that information is propagated to a greater extent of downstream targets, including the amygdala and the hippocampal and thalamocortical systems (Gott et al., 2017). PGO waves have been associated with the maturation of the nervous system, memory formation and visual perception, including visual hallucinations during dreaming (Gott et al., 2017).

To facilitate the understanding of its mechanisms and functional significance, Simor et al. (2020) proposed to overcome the notion of REM sleep as a homogeneous state as they put forward a division of REM sleep into two contrasting microstates: tonic and phasic REM sleep. The authors drew the distinction due to differences in evoked and spontaneous cortical activity, mental experiences, and information processing. In general, phasic REM sleep describes a more activated state featuring bursts of rapid eye movement, PGO waves, muscle twitches, sawtooth waves, and irregular respiratory and cardiac activity. Interspersed segments of tonic REM sleep on the other hand represent longer and calmer states between periods of phasic activity. With regard to environmental alertness, the two states oppose each other. During phasic periods external information processing is attenuated and attention is directed internally (e.g., to promote dream experiences), whereas tonic periods resemble a wake-like state, in which alertness and environmental processing are partially preserved.

The two microstates show differences in spontaneous oscillatory activity within three frequency ranges, i.e., high-alpha and beta (12-30 Hz), gamma (30-50 Hz) as well as within delta and theta bands. Suggesting increased alertness and in line with the aforementioned distinction in environmental alertness, a relative enhancement in alpha and beta frequency band power - high-alpha and beta oscillations (12-30 Hz) in particular - was found during tonic periods compared to phasic periods (Simor et al., 2020). In turn, increased gamma power (30-50 Hz frequency range) and power within delta and theta bands (2-4 Hz in particular) were found during phasic REM sleep.

The distinction between phasic and tonic periods may yield a more refined comprehension of how REM sleep contributes to memory processes. Along with NREM sleep, REM sleep has been proposed as a potential candidate implicated in the consolidation of memories, specifically emotional memory consolidation (Nishida et al., 2008). Increases in theta frequencies and power observed during phasic REM are linked to enhanced neuronal

activity and coordination throughout the hippocampus and with cortical areas (de Almeida-Filho et al., 2021, Montgomery et al., 2008). Taken together with further characteristics of phasic periods, PGO waves, and rapid eye movements, which have been related to vivid mental pictures, recent hypotheses have attempted to provide an explanatory framework for the mechanisms underlying the consolidation of emotional memories during REM sleep. The two microstates may represent alternating processes of (re)activation of emotional memories during phasic REM periods and integration/contextualization of those reactivated emotional memories promoted by interspersed tonic REM periods.

However, direct empirical evidence corroborating this notion is missing and to converge on an answer regarding the functional significance of the two microstates further research is needed. Furthermore, alluding to the "sequential hypothesis", given that natural REM sleep is always preceded by NREM sleep, it may serve a function in memory formation complementary to NREM sleep. Thus, sequences of REM and NREM sleep may be taken into consideration when investigating memory formation.

Delving deeper into the distinct sleep stages and the functional significance some features have been associated with, it becomes increasingly apparent that sleep is not solely crucial for physical, but also cognitive and psychological restoration and maintenance. As mentioned above, REM sleep in particular has gained a recent surge of interest for its role in emotional memory and emotional processing. The following chapter will address the evolving understanding of the relationship between sleep and emotions in greater detail.

2. Sleep and Emotional Reactivity

2.1. Emotions and Emotional Reactivity

While everyone might have an intuitive understanding of what an emotion is, the scientific literature has not yet converged on a single universally accepted definition of emotions, indicating just how complex and multifaceted of a phenomenon they are. Definitional approaches typically refer to a combination of subjective experiences, physiological as well as behavioural responses. For example, the American Psychological Association defines emotions as, "conscious mental reactions (such as anger or fear) subjectively experienced as strong feelings usually directed toward a specific object and typically accompanied by physiological and behavioural changes in the body" (*Emotions*, n.d.).

One way to classify the subjective experience of emotions is to evaluate them according to the basic components of the emotional process. This is done in dimensional approaches, one of which is the circumplex model of emotion (Russell & Carroll, 1999). The model posits that all emotions are derived from two fundamental neurophysiological systems, one associated with valence and the other with arousal (Tseng et al., 2013, Sharar et al., 2016).

Proceeding Darwin's theory of nature (Darwin, 1872), James postulated that it is alterations in the somatic/visceral and behavioural responses that give rise the subjective experience of emotions (James, 1884) and ignited a debate concerning the nature of emotions and their relationship with the patterns of the somatic/visceral activity that still remains today. Physiological responses are governed by the autonomic nervous system (ANS), which further subdivides into two branches: the sympathetic nervous system (excitatory function) and the parasympathetic nervous system (inhibitory function). These two nerve systems often work in antagonistic manner for maintaining physiological equilibrium. However, certain situations

require one system to dominate over the other. For example, when encountering a stressor, the sympathetic nervous system predominates, preparing the body for action, but once the stressor is dissipated, the parasympathetic system gains the upper hand and helps in order to regain a state of calmness.

Emotional reactivity is one aspect of emotions and is defined in terms of quality and intensity of emotions of response to affective stimuli (Wheeler et al., 1993). It is most often studied by means of exposing participants to images or video clips with positive, negative or neutral content whilst assessing their emotional reactivity via subjective ratings and/or physiological measurements of various kinds. Regarding subjective ratings, most studies evaluate the emotional response following the approach of circumplex model of emotion along two dimensions, i.e., valence and arousal (Russell & Carroll, 199). Valence refers to the extent to which emotions are categorized as pleasant or unpleasant, with neutral often considered as an intermediate value, whereas arousal ratings range from calm to excitement.

Emotional reactivity can be assessed with a number of different (neuro)physiological measurements, the most prevalent are heart rate deceleration (HRD), skin conductance response (SCR), and late positive potential (LPP). HRD is an autonomic orienting response that is characterized by an initial slowing of the heart rate in response to a stimulus, particularly to emotionally negative stimuli. HRD responses are highly automatic and predominately depend on subcortical structures as the amygdala (Kuniecki et al., 2002). The SCR is yet another autonomic measure, primarily under sympathetic control and reflects a state of arousal (Sequeira et al., 2009). The LPP represents a physiological increase in amplitude of the parietal event-related EEG response and is sensitive to emotional arousal (Bolinger et al., 2019; Cuthbert et al., 2000).

2.2. Emotional Reactivity and Sleep

Chances are, most of us have an intuitive understanding for how strongly connected sleep and our emotions can be. It's a connection, one has likely experienced firsthand, where just a single poor night of sleep can leave us feeling impatient, more irritable and susceptible to stressors, indicating a compromised emotional regulation. In turn, many can relate to the frustration of lying awake at night, unable to fall asleep due to stress and anxiety. Indeed, existing research suggests a bidirectional link between sleep and emotions, however putatively more complex and dynamic than initially assumed. It is well-documented that mood (i.e., lasting affective states not necessarily triggered by specific stimuli) is significantly affected by sleep loss, even more so than cognitive or motor performance (Pilcher & Huffcutt, 1996). Recently, there has been an increasing focus on the link between sleep and emotional reactivity, although the underlying mechanisms remain to be fully understood (Tempesta et al., 2018).

Early systematic examinations on the effects of sleep on emotional reactivity were conducted through studies employing sleep deprivation, referring to prolonged sleepless periods, or sleep restriction paradigms, which restrict sleep to a limited time rendering it insufficient. Nonetheless, the two terms are often used without distinction. Those paradigms have also been adapted in order to evaluate the contribution of different sleep stages, e.g., deprivation of NREM versus REM sleep stages, or including early or late-night sleep deprivation to examine the effect of distinct sleep stages. In addition to deprivation studies, numerous researchers have sought to examine the connection by assessing emotional reactivity directly after nighttime sleep or daytime naps.

Emotional reactivity is typically assessed comparing reactivity to the same or novel emotional stimuli of participants after a period of sleep versus an equivalent period of waking using subjective and/or objective measures. Generally, an attenuated emotional reactivity

(rather than an enhanced or maintained response) is considered more adaptive, a notion derived from studies with healthy individuals during wakefulness. Repeated exposure to emotional, and especially negatively valanced, stimuli has been associated with increased attenuation to the material (Minkel et al., 2011, Baran et al., 2012).

Regarding the role of sleep, two different hypotheses have been proposed to explain the impact of sleep on emotional processing. The currently leading theory in the field is known as the "Sleep to Forget and Sleep to Remember" (SFSR) hypothesis and was initially put forward by Walker and van der Helm (2009). The theory posits that when emotional memories are consolidated during sleep, the emotional tone of the experience gets reduced while the content gets preserved. In contrast, Baran et al. (2012) hypothesized in what is known as the "Emotional Salience Consolidation" (ESC) theory that while sleep improves the consolidation of emotional memories, it maintains likewise their emotional valence. Both theories assign a crucial role to REM sleep in emotional processing. However, empirical studies do not exclusively favour one hypothesis over the other as studies yielded contrasting results, indicating that the actual impact of sleep on emotional reactivity may be more intricate and influenced by various factors.

2.2.1. Deprivation Studies

The prevalent notion derived from sleep deprivation studies is that without adequate sleep negative emotional reactivity in response to adverse experiences is significantly enhanced, while positive reactions to positive events are often attenuated. Zohar et al. (2005) used data from medical residents who were monitored every 6 months for 5-7 days over the course two years to investigate the effects of sleep disruptions on emotional reactivity to daytime work events. Not only did the authors observe an enhancement in negative emotions following sleep loss, but even a reduction in positive emotions. Nonetheless, these results

have to be interpreted with caution as negative mood represents a common confound of sleep loss. A study with a cohort of university students by Tempesta and coworkers (2010) indeed accounted for the negative mood which accompanies sleep loss. In line with the results obtained by Zohar et al. (2005), following a night of sleep deprivation, subjects rated neutral pictures more negatively compared to well-rested subjects. Nevertheless, this effect was not mediated by an increased negative mood.

Even though further studies corroborate the findings regarding an enhanced emotional reactivity in response to negative stimuli consequent to sleep loss (e.g., Altena et al., 2016), contrary results were reported by a significant minority of studies. When compared to a normal sleep condition, an extent period of sleep restriction (5h a night for 5 consecutive nights) entailed greater negative ratings of pleasant and neutral pictures, while there was no such effect for unpleasant pictures (Tempesta et al., 2020). Pilcher et al. (2015) reported an overall decrease in valence and arousal ratings following both partial and total sleep deprivation, with an interaction effect yielding greater effects for positive than negative events.

A recent systematic review and meta-analysis by Lipinska and coworkers (2022) included 24 studies which examined changes in emotional reactivity in response to affective stimuli (at least one comparison of negatively valences with neutral stimuli) before and after a period of waking versus sleep in healthy individuals. Results obtained by sub-group analysis contrasting subjects with full-night sleep to sleep-deprived subjects showed that sleep deprivation engendered significantly larger changes in response to positive stimuli, as they were characterized by more negative responses. Yet, it should be mentioned that the subgroup analysis regarding sleep deprivation only included three studies.

As noted by Tempesta et al. (2018), despite a small number of contrasting results, the existing body of research on sleep deprivation consistently supports the idea that substantially

impacts emotional appraisal, in that it entails a tendency to perceive neutral stimuli more negatively as well as a heightened emotional reaction to these stimuli.

2.2.2. Full Night Sleep and Daytime Nap Studies

Whereas sleep deprivation studies are generally characterized by a reduction in normal sleep time, there are also studies examining reactivity to emotional stimuli after regular overnight sleep or after a daytime nap. Studies yielded inconsistent findings, some in support of an attenuated emotional reactivity following sleep (e.g., Gujar et al., 2010), others observed increased (Wagner et al., 2001; Jones et al., 2018) or same level emotional reactivity (Baran et al., 2012).

Employing a full night sleep paradigm, Jones et al. (2018) assessed emotional reactivity following a full night sleep or an equivalent wake period. Data was collected from 40 young and 41 middle-aged adults, who had to rate negative and neutral pictures taken from the International Affective Picture System (IAPS) with respect to arousal and valence, both before and after the experimental manipulation (sleep or wake period). After a night of sleep relative to a day of wakefulness, subjects showed elevated self-reported negative affect in response to negative, but not neutral stimuli.

In contrast, another full night sleep study conducted by Baran et al. (2012) had 106 young adults rate negative and neutral pictures from the IAPS in terms of arousal and valence during two session, that were separated by 12h period of wakefulness or 12hr including an overnight sleep. Compared to the sleep condition, subjects perceived negative pictures less negative after the period of wakefulness, for neutral pictures however, this effect was not observed. Although not statistically significant, a similar trend was observed with respect to arousal ratings: the wake condition entailed a greater reduction in arousal ratings for negative pictures when compared to the sleep condition.

It is important to keep in mind that if sleep and wake groups are tested at different times of the day, it is possible that circadian factors rather than sleep have an impact on emotional reactivity and thus contribute to the inconsistency in the results observed. For instance, Hot et al. (2005) reported an attenuated reactivity to emotional stimuli in the morning and early afternoon, ensued by an increase peaking at 3:30 PM which then decreased again in the evening.

In order to account for circadian factors, paradigms using daytime naps may be more suited, as individuals both from sleep and wake groups can be tested at the same time of the day. Gujar et al. (2010) conducted a between subject study with 36 healthy young adults that were randomly designed to 90-min nap (monitored with Polysomnography) or wake condition. Participants had to perform an emotional face recognition task comprising 4 emotions, i.e., anger, fearfulness, happiness and sadness before and after experimental manipulation (sleep vs. waking). Results showed a significant increase in anger expression ratings for the wake relative to the sleep group, as well as an amplification of fear expression ratings, which were decreased in the nap group. With regards to positive emotions, the authors reported a significant increase in happiness expression ratings for the nap group.

Since subjective ratings rely on self-report, they are prone to various biases, e.g., subjects may respond in a manner that is perceived as socially acceptable (desirability bias) or tend to give consistent or extreme responses (response bias). Therefore, the co-registration of additional objective measures, such as physiological ones, is beneficial in order to the understanding the underlying neural and physiological processes that accompany emotions. Pace-Schott et al. (2011) reported a between-subjects study including 43 young adults that, during two afternoon sessions, viewed negative and neutral pictures from the IAPS. The second session included also novel pictures that were presented alongside previously seen pictures. The sessions were separated by a 2.5hr period in which participants were randomly

allocated to either a nap or wake condition. In addition to valence and arousal ratings, evoked skin conductance response (SCR), heart rate deceleration, and Corrugator supercilia electromyogram response (EMG) were measured. For subjective measures no group difference was found. With regard to physiological measures however, the nap group showed greater inter-session habituation in SCR and EMG (greater for negative pictures) plus a tendency for reduced inter-session habituation in HRD.

Another interesting finding was reported by Bolinger et al. (2019). 32 young adults were randomly assigned to either a sleep or wake group. During two sessions, separated by a 10hr period in which participants either slept or stayed awake, they were presented negative and neutral pictures (IAPS) and had to rate them for valence and arousal, whilst HRD, and late positive potential of the EEG were recorded. During the second session, participants were shown novel pictures alongside previously seen pictures. Sleep did not influence arousal or valence ratings. With regards to physiological measures, compared to staying awake, sleep led to a decrease in LPP emotional response, whereas the HRD remained the same. However, a follow-up assessment 10 days later yielded differing effects on valence ratings, HRD and LPP. Consequently, the authors proposed that sleep influences subjective ratings in the long term.

In summary, findings on the role of sleep on emotional reactivity have reported mixed and contrasting findings both for subjective ratings and for physiological measurements. Inconsistent results could be attributed to various factors, including differences in methodology (e.g., assessment of emotional reactivity, overnight sleep vs. daytime nap), moderating variables (e.g., circadian factors), and/or different factors that constitute sleep, e.g., how specific sleep stages contribute to emotional regulation. Given the assumed crucial involvement of REM sleep in emotional processing, taking into account the time spent in

various sleep stages could potentially clarify the complex relationship between sleep and emotional reactivity.

2.2.3. REM and Emotional Reactivity

Within the theoretical framework of the SFSR model, REM sleep in particular is central for the emotional memory processing (Walker & van der Helm, 2009). This idea is based on a REM sleep's unique neurobiological characteristics. Research using neuroimaging has demonstrated an increased activity in the amygdala and hippocampus during REM sleep for emotional stimuli relative to neutral stimuli (Dolcos et al., 2004, 2005; Kilpatrick & Cahill, 2003). Neurochemically, REM sleep is marked by heightened cholinergic activity (Vázquez & Baghdoyan, 2001) and a lack of aminergic activity (Pace-Schott & Hobson, 2002), particularly the absence of noradrenergic inputs from the locus coeruleus, a structure that has been associated with conditions characterized by high stress and anxiety disorders (Sullivan et al., 1999).

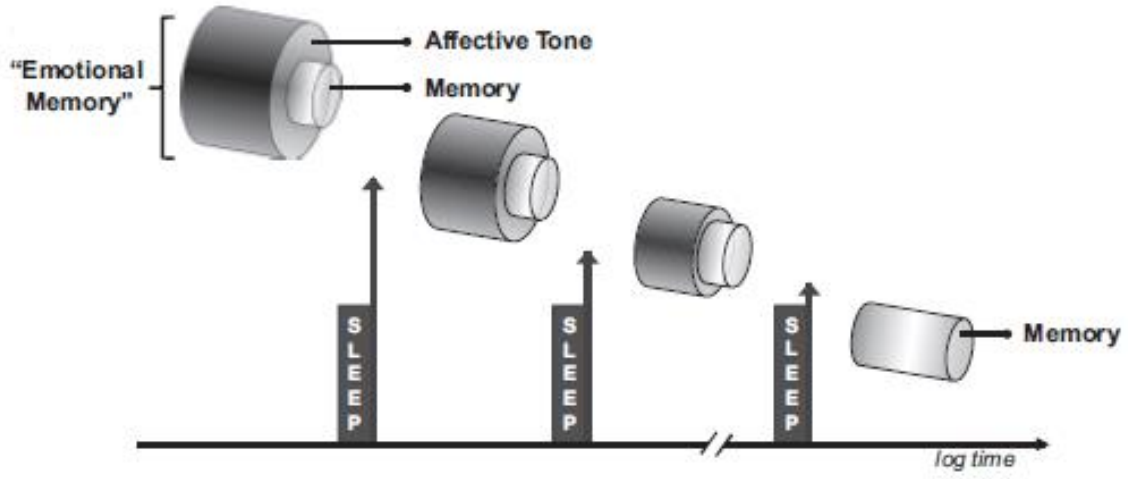

Figure 2.1. Visualization "Sleep to Remember, Sleep to Forget" (SFSR) Model. Repeated REM processing over several nights strengthens the content, while the affective charge reduces during recall. Taken from Walker & van der Helm (2009).

The SFSR theory holds that during REM sleep, the declarative component of the emotional experience is strengthened via reactivation of the same emotion-related brain areas, whilst the lack of aminergic neurotransmitters permits an attenuation of the affective tone as these experiences are processed in a state with less physiological arousal.

In contrast, the competing ESC model suggests that given REM sleep's high level of activity in brain regions associated with emotional processing, it might instead reinforce the salience of events, and thus increase emotional reactivity (Baran et al., 2012; Pace-Schott et al., 2011, Werner et al., 2015).

Evidence in favour of SFSR hypothesis was provided by Gujar et al. (2010). The authors further grouped participants of the nap group into those that reached REM sleep (n = 8) and those that did not (n = 10). The REM group showed a significant reduction in fear ratings and a significant increase in happy ratings, relative to the non-REM group. It was thus suggested that REM sleep in specific attenuates emotional reactivity.

Similarly, a full-night within-subject study by Rosales-Lagarde et al. (2012) investigated the effects of selective REM sleep deprivation on emotional reactivity. 20 young healthy males were randomly assigned to two groups: a REM sleep deprivation group (REM-D) and a NREM sleep interruption group (NREM-I). The REM-D group was woken up every time they entered REM sleep, the NREM-I group was awakened from stage 2, 3 or 4 or NREM sleep in order to control for non-specific effects of sleep disruption and fragmentation. In a within-subject experimental design subjects spent 4 consecutive nights at the laboratory, all participants completed a visual emotional reactivity task in a magnet resonance imaging (MRI) scanner before and 24 hours after the sleep manipulation. The REM-D group showed an increase in behavioural and neural activity in response to threatening stimuli. The opposite effect was found for the NREM-I group, i.e., behavioural

and neural activity were decreased in response to threatening stimuli. These findings suggests that the absence of REM sleep is associated with heightened emotional reactivity.

In contrast to the aforementioned results, the study by Baran et al. (2012) - mentioned in the previous section - represents one of the most influential studies in support of the idea that sleep, REM sleep in particular, in fact increases emotional reactivity. Additional analyses, investigating the correlation between REM sleep and valence ratings, revealed a significant negative correlation: Participants with more REM sleep showed less attenuation of negative reactivity. Consequently, the authors proposed that REM sleep rather preserves than de-potentiates emotional reactivity. very large same size

Similarly, the above-reported study by Pace-Schott et al. (2011) further assessed the relationship between REM sleep and emotional reactivity by means of physiological measures. The authors reported a greater inter-session habituation of EMG measures to negative stimuli correlated with SWS, whereas REM sleep was associated with less inter-session habituation of SCR in response to negative stimuli.

Lastly, Lara-Carrasco et al. (2009) evaluated emotional reactivity in 35 healthy subjects preceding and subsequent to either a night of late- night REM sleep deprivation (REMD) or a night of comparable interrupted sleep (control group). Subjects had to rate negative and neutral pictures for arousal and valence. It was found that late-night REM sleep deprivation enhanced emotional adaptation to negative pictures, manifesting itself in a decrease in subjective arousal rating for negative pictures. The authors suggested that REM sleep may augment morning reactivity in response to negative emotional stimuli.

To summarize, the increasing body of results indicates an influence of REM sleep on emotional reactivity. However, the existence and the exact nature of this influence remains a subject of debate and yet to be understood. As evidence for both, the SFSR and ESC model

exists, further research is necessary to examine how REM sleep in particular influences specific emotional processes and emotion regulation.

3. Method

3.1. Introduction and Hypotheses

The present study used preliminary data obtained in an ongoing larger project which, employing a nap paradigm, is investigating the influence of REM and NREM sleep on the consolidation of emotional memories and emotional reactivity in a between-subject design. The study at hand focused on emotional reactivity alone and, given the limited number of participants collected to date, examined the contribution of different sleep stages only in an exploratory manner.

As discussed in the previous chapter, an increasing body of findings indicates a pivotal role of sleep in emotional reactivity. Yet, the modulatory effect of sleep remains to be debated as empirical evidence has been highly varied both with regard to subjective ratings as well as physiological measurements. Contrasting findings might be due to differences in the methodological approach, sometimes considering subjective and physiological emotional reactivity disjointedly. The present study investigated emotional reactivity in response to neutral and negative stimuli before and after a daytime nap with both - subjective and physiological measures. Moreover, as evidence suggests that the effect of sleep emerges in the long-term (Bolinger et al., 2019), emotional reactivity was assessed again after two days for drawing inferences about the temporal unfolding of the effect. Participants were randomly assigned to either a NAP (REM vs NREM) or WAKE group and emotional reactivity was examined at three measurement time points, i.e., preceding the daytime nap (T0), subsequent to the daytime nap (T1), and after two days (T2).

The inherent variability in empirical observations poses a challenge to the formulation of a hypothesis. Nevertheless, the currently leading hypothesis by Walker & van der Helm (2009) proposes that sleep preserves the content of a memory whereas the emotional tone is

discharged. The subsequent hypotheses regarding emotional have been articulated within the theoretical framework of the SFSR hypothesis.

The first hypothesis concerned the change in emotional reactivity immediately following the daytime nap:

H1: Emotional reactivity for negative vs. neutral stimuli decreases from T0 to T1 for both subjective and cardiac measurements in the NAP group compared to the WAKE group.

Given that both groups have obtained sleep when assessed at measurement time point T2, hypothesis two predicted:

H2: Emotional reactivity for negative vs. neutral stimuli decreases from T0 to T2, with a greater reduction in the NAP group relative to the WAKE group for both subjective and cardiac measurements.

The SFSR framework assigns a pivotal role to REM sleep, in that the reactivation of emotion-related brain areas during REM sleep preserves the content while weakening the affective tone of the memory. Consequently, the exploratory analysis regarding the specific contribution of REM and NREM sleep was phrased as:

H1a): A greater reduction in emotional reactivity for negative vs. neutral stimuli assessed with both subjective and cardiac measures is expected from T1 to T0 for the REM group relative to the NREM and WAKE groups.

To assess whether the unfolding of the effect of REM sleep requires that a certain amount of time has passed the final hypothesis expected:

H2a): Emotional reactivity for negative vs. neutral stimuli decreases from T0 to T2, with the greatest reduction in the REM group compared to the NREM and WAKE group for both subjective and cardiac measurements.

3.2. Materials and Methods

3.2.1. Participants

Healthy adults (18-35 years) were recruited to participate in the study. Recruitment was done via online leaflet and by direct approach. Participation was voluntary and informed consent was provided by all participants right at the beginning of the experiment. Participants received no compensation in return. All participants were provided with a personal identification number to ensure privacy. Main demographic data (e.g. age, gender, working status) were collected and participants were screened for eventual clinical and subclinical problems. In particular, potential sleep difficulties, depression and anxiety symptoms, and excessive somnolence were assessed using the Pittsburgh Sleep Quality Index (PSQI; (Buysse et al., 1989), the Insomnia Severity Scale (ISS; Morin, 1993), the Depression, Anxiety and Stress Scale (DASS-21; Parkitny & McAuley, 2010), and the Epworth Sleepiness Scale (ESS; Johns, 1991). Additionally, the Morningness-Eveningness Questionnaire reduced version was administered to collect information on participants' circadian preferences. Finally, blood phobia was assessed using the Mutilation Questionnaire (MEQ-r; Kleinknecht & Thorndike, 1990). Questionnaires were to be completed up until one day before the experimental session. Study eligibility depended on the use of medications, the presence of somatic or psychiatric disease, and blood phobia. Participants were randomly assigned to NAP or WAKE conditions. The study protocol was approved by the Ethics Committee of the School of Psychology of the University of Padova.

3.2.2. Procedure

The experimental session spanned 3 days and was held at the Sleep Psychophysiological Laboratory of the University of Padova. After informed consent was given by the participant, the session began around 11:30 with the application of electrodes for

standard PSG recording, including ECG and EDA. First, participants completed computer-based, subjective measures regarding their level of sleepiness and anxiety. Next, participants performed the emotional reactivity assessment, which was followed by the encoding phase of the emotional memory assessment starting at around 12:00 (emotional reactivity, T0). This was proceeded by a short break of 10 min in which for the sleep group the application of EEG montage was commenced, while the WAKE group was engaged in a conversation with the examiner of the experiment. The break was ensued by the first memory recognition test (emotional memory, T0). After completion, the WAKE group was dismissed and continued with their normal activities, whereas the polysomnographic montage for the NAP group was finalized. At around 13:00 the NAP group took a nap of 60 min (90 min bed allotted), or 90 min (two hours in bed allotted) in a quiet and darkened room. Sleep was monitored in real-time by the examiner of the experiment to assess sleep onset and offset times. The manipulation of the nap duration allowed to have shorter naps in which the likelihood of REM sleep should be decreased, as well as longer naps in which the likelihood of REM sleep should be increased. Based on the duration of REM sleep, participants were further segregated into NREM and REM groups. According to McDevitt et al.'s (2014) rule, participants who napped for more than one minute were included in the REM group.

At 15:30, on the same day all participants performed first the emotional reactivity assessment and the second emotional memory assessment (T1). After 2 days, all groups again underwent the emotional reactivity assessment and the emotional memory assessment (T2).

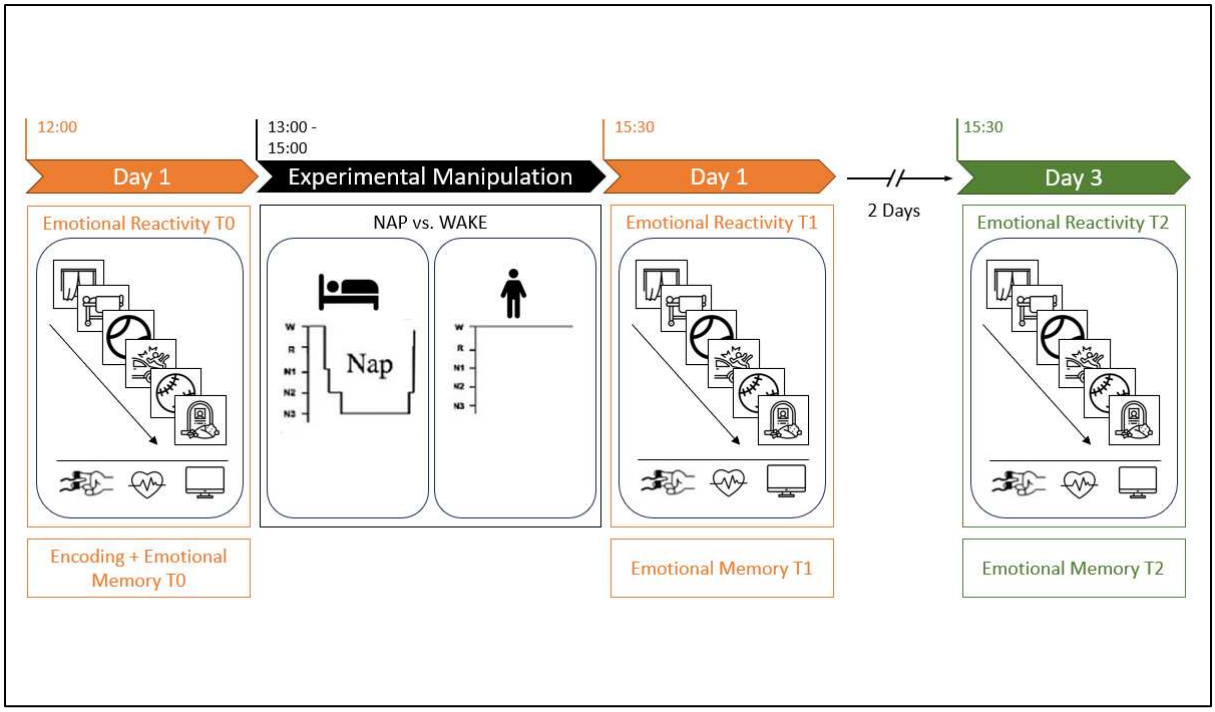

Figure 3.1. Timeline of the Experimental Procedure. On the first day at 12pm participants completed the initial emotional reactivity assessment, followed by the encoding phase of the emotional memory test and the first assessment of emotional memory (T0). The same day, following a nap or spending an equivalent period awake, participants performed the second emotional reactivity and emotional memory assessment (T1). Two days later, participants returned to the lab in order to perform the final assessment of both emotional reactivity and emotional memory (T2).

3.2.3. Emotional Reactivity Assessment

Participants' emotional reactivity was assessed through a series of 28 pictures. The stimuli included 14 unpleasant, high-arousal images (such as mutilations and threats) taken from the International Affective Picture System (Lang et al., 1997) and 14 neutral images (sport objects), all taken from Mnemonic Similarity Task (Stark et al., 2015). Each picture was displayed for 6 seconds, followed by a 4-second ISI, during which participants rated the subjective valence (pleasantness/unpleasantness) and arousal (activation/calmness) of the image using the Self-Assessment Manikin on a 9-point Likert scale. There was an ITI of 8-12 seconds before the start of the next trial. Electrodermal activity (EDA) and electrocardiogram (ECG) were recorded throughout the emotional reactivity assessment. The same procedure, including the order and pictures used, as well as the EEG and EDA recording, was employed

at both T1 and T2. The emotional reactivity assessment took approximately 10 minutes to complete.

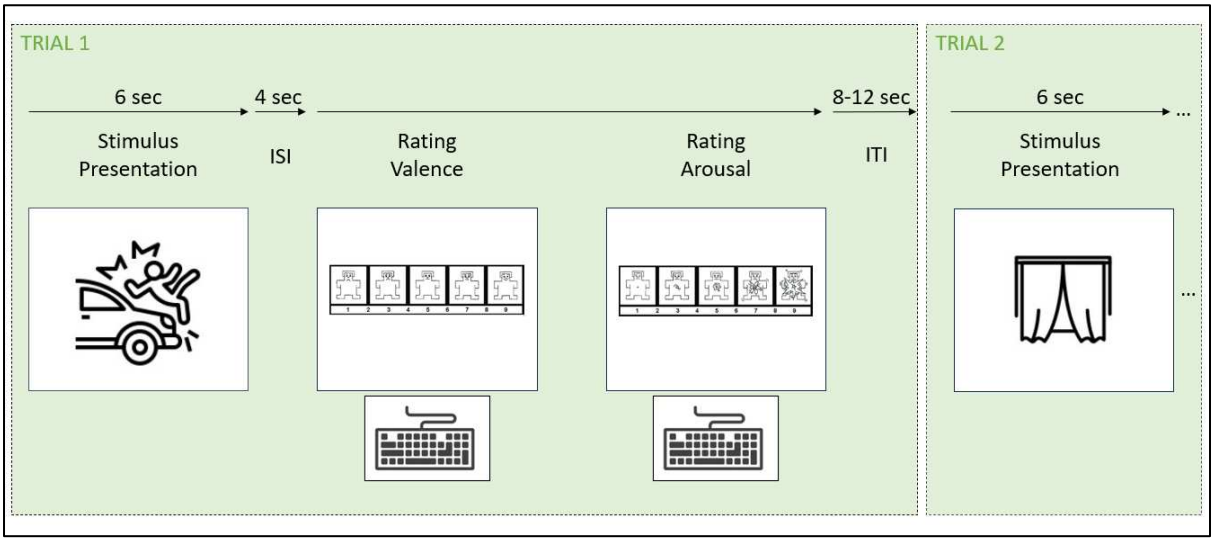

Figure 3.2. Schematic Illustration of Emotional Reactivity Assessment Trials. Participants were presented with negative and neutral stimuli for 6 seconds. This was followed by a 4-second ISI in which subjective valence and arousal ratings were obtained by keyboard entry. An 8-12-second ITI preceded the onset of the subsequent trial.

3.2.4. Emotional Memory Assessment

Emotional memory was evaluated with a picture recognition task. The task was devised to investigate the process of encoding negative and neutral images, as well as the extent to which this information was retained over time. The task comprised a series of 240 affective pictures drawn from the International Affective Picture System (Lang et al., 1997).

Encoding phase. During the encoding phase, participants were presented with a series of 120 images (60 negative and 60 neutral), each displayed for 3 seconds with a 1.5-second ISI.

Memory recognition test. Following the encoding phase, participants completed a memory recognition test (referred to as Immediate recognition, denoted as T0). The test consisted of the presentation of 80 pictures, half of which were already presented during the encoding phase (20 negative and 20 neutral), whereas the remaining 40 were entirely new

images (20 negative and 20 neutral). Each image was presented for 2.5 seconds, followed by a 0.5-second ISI. For each item participants had to indicate whether the picture displayed was "Old" or "New". This was done by responding to the question “Have you seen this picture before?” by means of clicking onto either an encircled “Yes” (lefthand screen) or “No” (righthand screen) response button via mouse click. The positioning of the “Yes” and “No” response buttons was the same for all participants and remained fixed throughout the test. The trial types were counterbalanced during the test.

During the second session (T1), participants had to complete the first delayed recognition test. T1 followed the same procedure as T0. It again comprised a total of 80 items – 40 repeated items and 40 novel items. Notably, the 40 repeated items in this test were distinct from those used during T0.

After 2 days participants underwent a third testing session (referred to as T3) consisting of a memory recognition test with 80 pictures. The test included the remaining 40 items originally presented during the encoding phase, as well as 40 new items (20 negative and 20 neutral). Memory performance was quantified using parameters from signal processing theory, including hit rate, false alarm rate, and d’. The execution of this task was carried out using Psychopy 2 (Peirce et al., 2019).

3.2.5. Electrophysiological Recording and Polysomnography

For examining participants’ sleep the signal obtained via the V-Amp (Brain Products mbH, Gilching, Germany) from 12 EEG channels Fpz, Fz, F3, F4, Cz, C3, C4, Pz, P3, P4, O1, O2 was used. The electrodes were positioned following the International 10-20 system. The signal was online referenced to FCz and offline to the contralateral mastoid (M1, M2). Bipolar electrooculographic and electromyographic recordings from electrodes in close proximity to the eyes and at the chin were applied in order to track participants’ eye

movement and measure muscle activity, respectively. Ag/AgCl electrodes were used and impedances for electrooculographic and electromyographic recordings were kept below 10 kΩ and 5 kΩ for the EEG recording. Signals were sampled at 500 Hz. To record ECG activity, three other electrodes were attached to the participants' chest. All electrodes of the PSG recording were applied following the guidelines of the American Academy of Sleep Medicine (Iber, 2007).

3.3. Behavioural analysis

As the thesis focused on the assessment of emotional reactivity, data from the emotional memory assessment will not be included in the analyses. Similarly, SCR measures will not be reported.

3.3.1. Cardiac Activity

To determine HDR, a slope-based detection algorithm in the open-source software EDFBrowser (https://www.teuniz.net/edfbrowser/) identified R components of the QRS complex. Trials were visually inspected by trained research assistants and corrected if needed.

HRD was assessed using the Matlab toolbox Kardia (Perakakis, Joffily, & Taylor, 2009). The temporal window for assessing the HRD in response to negative and neutral stimuli was set from -2 seconds pre-stimulus onset (epoch start) to 8 seconds (epoch end). The baseline was calculated as the mean of the two seconds before stimulus onset compared to the subsequent 8 seconds (6 seconds of stimulus presentation and + 2 seconds post-stimulus presentation). Absolute values of the cardiac frequency were obtained for baseline and the subsequent 8 seconds. Delta response was calculated as the change in absolute value

between two consecutive time points, e.g., the difference between the absolute cardiac frequency of second 1 and second 2.

3.4. Statistical Analysis

In total 46 participants took part in the study. Due to attrition, data from one participant had to be removed from further analysis. Therefore, data from 43 participants entered the main analysis. As five participants (23,81%) in the NAP group did not fall asleep during the experimental session, they were assigned to the WAKE group and their data was analysed accordingly. Demographic and psychological data from one participant was missing and therefore did not enter the analysis.

Emotional reactivity was examined based on both subjective and physiological measures. Subjective ratings assessed emotional reactivity based on the average of self-reported ratings on valence and arousal for each session (T0, T1, T2), whereas the delta response was used to evaluate emotional reactivity in terms of the physiological response to emotional stimuli. A decrease in emotional reactivity should reveal itself with respect to self-reports in less negative valence ratings as well as a decreased arousal rating. Physiologically, a decline in HRD was considered indicative of a weakened emotional reactivity.

3.4.1. Demographic and Sleep Measures

Participants' demographic (Age, Gender) and psychological measures on PSQI, ISI, DASS, ESS, and MEQ-r were analysed. For evaluating differences in the composition of NAP and WAKE groups with regard to gender, a $\chi 2$-test was conducted. T-tests for independent samples were used for analysing participants' psychological and demographic characteristics. Dependent variables of the psychological characteristics represented total

scores assessed with the PSQI, ISI, DASS, ESS, and MQ. Effect sizes were assessed with Cohen's d.

For participants in the NAP group, Time in Bed (TIB), Wake After Sleep Onset (WASO), Total Sleep Time (TST), as well as the distinct sleep stages were analysed descriptively. TIB referred to the total time a participant spent in bed during the sleep period. WASO corresponded to the total time spent awake after the initial sleep onset. TST is a measure representing the overall quantity of a participant's sleep by subtracting WASO from TIB.

3.4.2. Emotional Reactivity Assessment

3.4.2.1. Self-report and HRD

Valence (Va) and arousal (Ar) ratings were analysed in response to negative (Neg) and neutral (Neu) stimuli by running two separate linear mixed models (LMM). Subjective ratings (Val, Ar) were included as the dependent variable and session (T0, T1, T2), experimental condition (NAP vs. WAKE), stimulus valence (negative vs. neutral) and the interaction between time and experimental condition as fixed effects. Each subject was included as a random effect. The model specifications were as follows:

$$Val \sim 1 + Session + Group + Valence + Session{:}\,Group + Session{:}\,Valence + Group{:}\,Valence + Session{:}\,Group{:}\,Valence + (1|Subject)$$

$$Ar \sim 1 + Session + Group + Valence + Session{:}\,Group + Session{:}\,Valence + Group{:}\,Valence + Session{:}\,Group{:}\,Valence + (1|Subject)$$

Cardiac deceleration was assessed using a LMM with the session (T0, T1, T2), experimental condition (NAP vs. WAKE), time (i.e., 6 seconds post-stimulus presentation), stimulus valence (negative vs. neutral) and the interactions between time and experimental

condition as fixed effects. The variable "Subject" was included as a random effect. Delta response was included as the dependent variable. Thus, the model specification was as such:

$$Delta \sim Session + Group + Valence + Time + Session:Group + Session:Valence + Group:Valence + Session:Time + Group:Time + Valence:Time + Session:Group:Valence + Session:Group:Time + Session:Valence:Time + Group:Time:Valence + Group:Session:Valence:Time + (1|Subject)$$

Exploratory analysis concerning the role of REM sleep on emotional reactivity was assessed by subdividing the NAP group into REM and NREM sleep and using the same model specifications for self-report and cardiac measures as described above. Nonetheless, given the small sample size in those two groups as well as the large number of post-hoc tests, it is important to acknowledge that significant effects may be potentially due to chance and lack replicability.

The restricted maximum likelihood (REML) function was used for estimating model parameters. Holm test was used for post-hoc analyses. Results were considered significant only with a p-value < 0.05. All analyses were conducted with JAMOVI software (The jamovi project 2.3, 2022).

4. Results

4.1. Demographic and Psychological Measures

Demographic and psychological data from one participant was missing and therefore did not enter the analysis. The NAP group was composed of 10 females and 11 males, the WAKE group consisted of 23 females and 2 males. Assessed with a $\chi 2$-test, the gender of the NAP and the WAKE group was not equally distributed, $\chi 2$ (1, $N = 46$) = 11.1, $p = <.001$. The demographic and psychological characteristics of the participants are depicted in Table 4.1.1. Participants in the two groups were comparatively balanced with regard to data obtained on Age, PSQI, ISI, DASS and MEQ-r. However, the groups differed significantly in their ESS scores, $t(43)$, $p = .003$, Cohen's $d = .96$.

Table 4.1.

Demographic and Psychological Characteristics of Participants

	NAP (*n*=21)		WAKE (*n*=25)		*t*(43)	*p*	Cohen's *d*
	M	*SD*	*M*	*SD*			
Age	23.1	3.21	23.3	1.65	-0.199	0.843	-0.059
PSQI	4.71	2.47	5.75	2.66	-1.347	0.185	-0.403
ISI	5.33	3.47	6.92	5.50	-1.135	0.262	-0.339
DASS	15.1	11.4	19.0	12.6	-1.068	0.292	-0.319
ESS	8.38	3.56	4.96	3.59	3.203	0.003*	0.957
MEQ-r	16.3	3.59	15.5	3.30	0.805	0.425	0.241

Note. *M* computed on total scores on PSQI: *Pittsburg Sleep Quality Index;* ISI: *Insomnia Severity Scale;* DASS: *Depression Anxiety and Stress Scale;* ESS: *Epworth Sleepiness Scale; MEQ-r:* Mutilation Questionnaire. $H_a\ \mu_{Sleep} \neq \mu_{Wake}$. $^*p < .05$.

4.2. Sleep Measures

Sleep data for one participant were unavailable and were thus excluded from the descriptive analysis. Table 4.2 provides a comprehensive overview of sleep characteristics, dividing the NAP group into REM and NREM groups, with a total sample of 20 participants that were considered in the analysis. In total, participants spent 63 minutes asleep, during which 9 out of 20 participants entered REM sleep. Notably, the REM group exhibited longer durations of N2 and N3 sleep compared to their NREM counterparts. Confirming the experimental assumption that nap duration increases the likelihood for REM sleep to occur, participants who were subsequently assigned to the REM group showed greater Total Sleep Time (M = 82.3, SD = 12.5) compared to the NREM group (M = 48.0, SD = 21.3).

Table 4.2.

Sleep Characteristics according to REM and NREM Sleep

	REM (n=9)		NREM (n=11)		TOT (n=20)	
	M	*SD*	*M*	*SD*	*M*	*SD*
TIB (min)	94.1	5.22	84.8	12.5	88.96	10.71
WASO (TOT, min)	11.7	11.0	36.8	19.0	25.5	21.71
TST (min)	82.3	12.5	48.0	21.3	63.4	24.7
N1 (min, %)	9.33 (11.1%)	3.43	8.09 (25.1%)	4.53	8.65 (19.6%)	3.93
N2 (min, %)	37.7 (49.7%)	8.63	26.7 (67.9%)	16.8	31.65 (59.5%)	14.84
N3 (min, %)	18.7 (23.3%)	12.5	4.32 (6.93%)	6.86	10.8 (14.3%)	11.97
N2+N3 (min, %)	56.4	13.3	31.0	22.5	42.45	23.02
REM (min, %)	11.7 (14.8%)	4.94	-	-	5.25 (6.7%)	6.69

Note. *Reported values were calculated based on time in minutes*. TIB: *Time in Bed*; WASO (TOT): *Wake after Sleep Onset*. TST: *Total Sleep Time.*

4.3. Self-report Measures

4.3.1. Valence Ratings

The fixed effects and random effects together explained 85.9% over the total (expected) variance of the valence ratings.

Generally, participants rated negative stimuli more negatively (M = 1.88, SD = 0.91) than neutral stimuli (M = 5.18, SD = 0.75). This difference was reflected in a significant main effect of Valence ($F_{1,\ 218.1}$ = 1502.46, p = <.001). Likewise, the main effect of Group was statistically significant ($F_{1,\ 44.1}$ = 4.76, p = .035), with the NAP group rating the emotional stimuli less negatively (M = 3.72, SD = 1.88) compared to the WAKE group (M = 3.38, SD = 1.82). Neither the main effect of Session (p = .595) nor the two-way interactions of Session*Group (p = .549), Session*Valence (p = .785) or Group*Valence (p = .124) reached statistical significance. Similarly, the three-way interaction of Session*Valence*Group yielded a p-value of .371 and was therefore non-significant.

Although neither of the two-way nor the three-way interaction was statistically significant, the data was further explored with a post-hoc analysis. While the subsequent post-hoc analysis has been adjusted for multiple comparisons, it has to be interpreted with caution, as significant results could arise by chance due to the large number of tests and given that the interactions were non-significant. The post-hoc analysis revealed a significant two-way interaction between the NAP and WAKE group for negative stimuli (t_{73} = 2.65, p_{holm} = 0.020). Figure 4.1 visualizes the direction of this effect: Considering all three sessions together, participants from the NAP group rated negative pictures less negatively than the WAKE group, most apparently at T2.

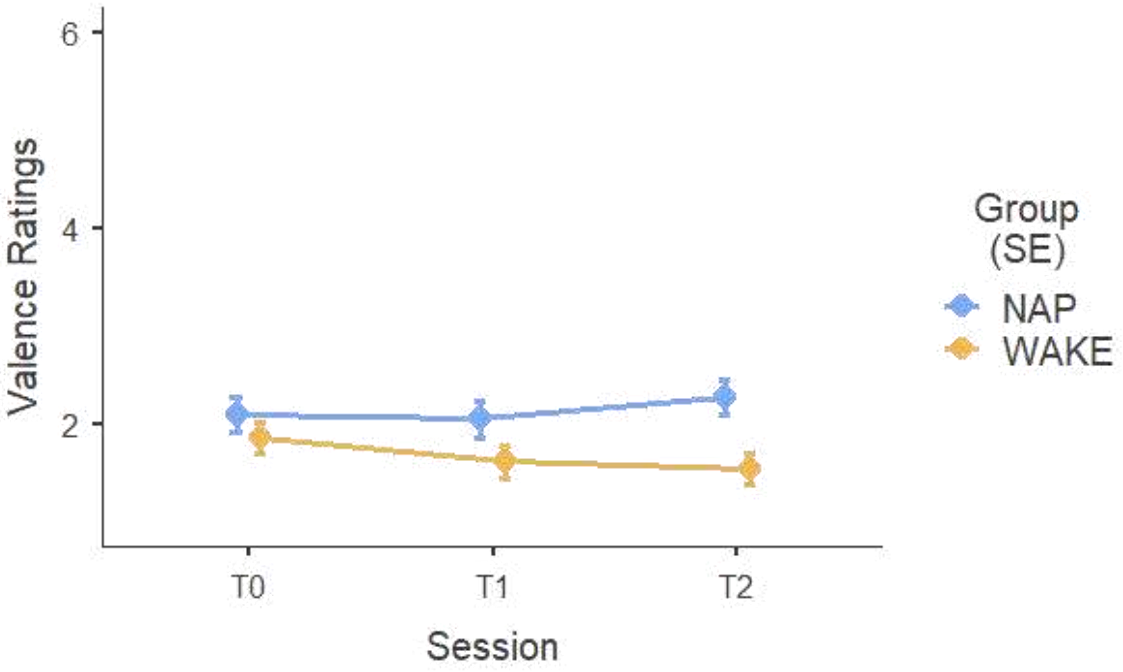

Figure 2.1. Valence Ratings in Response to Negative Stimuli between NAP and WAKE Group. Valence Ratings: Lower values correspond to greater negativity. Initially, participants provided similar valence ratings in response to negative stimuli at T0. Following the daytime nap, the NAP group's ratings remained constant at T1 compared to T0, whereas the participants who stayed awake rated negative stimuli slightly more negatively. Tested again after two days, the divergence in the valence ratings between the two groups became more pronounced at T2, primarily driven by NAP participants rating negative pictures less negatively compared to the WAKE group. Error bars represent standard error of the mean.

To explore the influence of distinct sleep stages on those results, further analysis dividing the NAP group into a REM (n=9) and NREM (n=11) group was carried out. A visual inspection of valence ratings in response to negative stimuli in Figure 4.2. indicated a difference emerging between the NREM and REM group from T1 to two days following the experimental manipulation at T2. Whereas neither of the three-way interactions between the REM, NREM and WAKE group at T1 was significant, the interaction between the REM and WAKE group in response to negative stimuli at T2 was ($t_{191} = 3.60$, $p_{holm} = 0.029$): The REM group displayed ratings less negatively compared to the WAKE group.

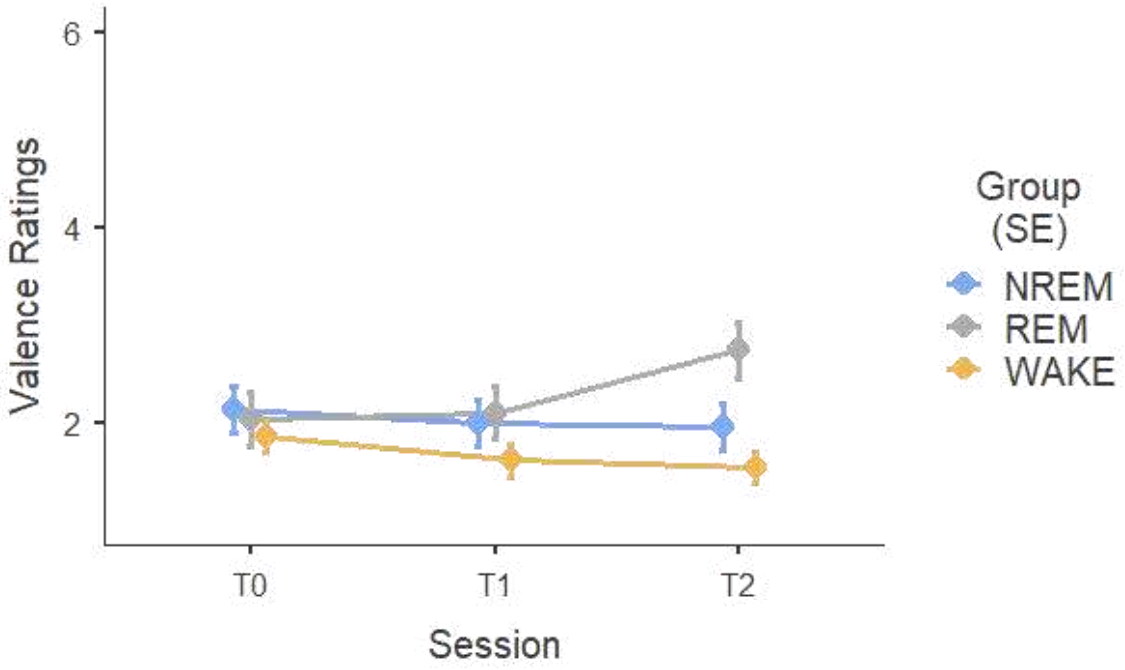

Figure 4.2. Valence Ratings in Response to Negative Stimuli between REM, NREM and WAKE Group. In the beginning, participants assigned comparable valence rating to negative stimuli at T0. After the experimental manipulation, the REM and NREM group's ratings remained constant at T1 compared to T0, in contrast to the WAKE group. However, a difference in valence ratings between the REM and NREM as well as the WAKE group became apparent: The REM group rated negative stimuli less negatively compared to the NREM and WAKE group. Valence Ratings: Lower values correspond to greater negativity. Error bars represent standard error of the mean.

4.3.2. Arousal Ratings

With regard to arousal ratings, 81.10% over the total (expected) variance was accounted for by the fixed and random effects together.

The main effect of Valence was statistically significant ($F_{1,\ 217.9} = 955.57$, $p = <.001$), with participants in both groups indicating less arousal ($M = 2.03$, $SD = 1.28$) when exposed to neutral stimuli relative to negative stimuli ($M = 6.54$, $SD = 1.70$). Arousal ratings between the NAP and WAKE group ($F_{1,\ 44} = 0.52$, $p = .477$), as well as between the sessions ($F_{2,\ 217.9} = 0.18$, $p = 0.834$), did not differ significantly from each other. A significant interaction between the Group levels and Valence levels could be observed ($F_{1,\ 217.9} = 18.15$, $p = <.001$). However, the variation in arousal ratings could neither be explained by either of the other two-way interactions, nor the three-way interaction between Group*Session*Valence.

Post-hoc tests regarding the two-way interaction of Group*Valence revealed a significant effect comparing the arousal ratings of the NAP and WAKE group in response to negative stimuli ($t_{65.1}$ = -2.47, p_{holm} = 0.033). This effect is illustrated in Figure 4.3.: the WAKE group reported greater arousal (M = 6.92, SD = 1.42) relative to the NAP group (M = 6.08, SD = 1.91) when presented with negative stimuli.

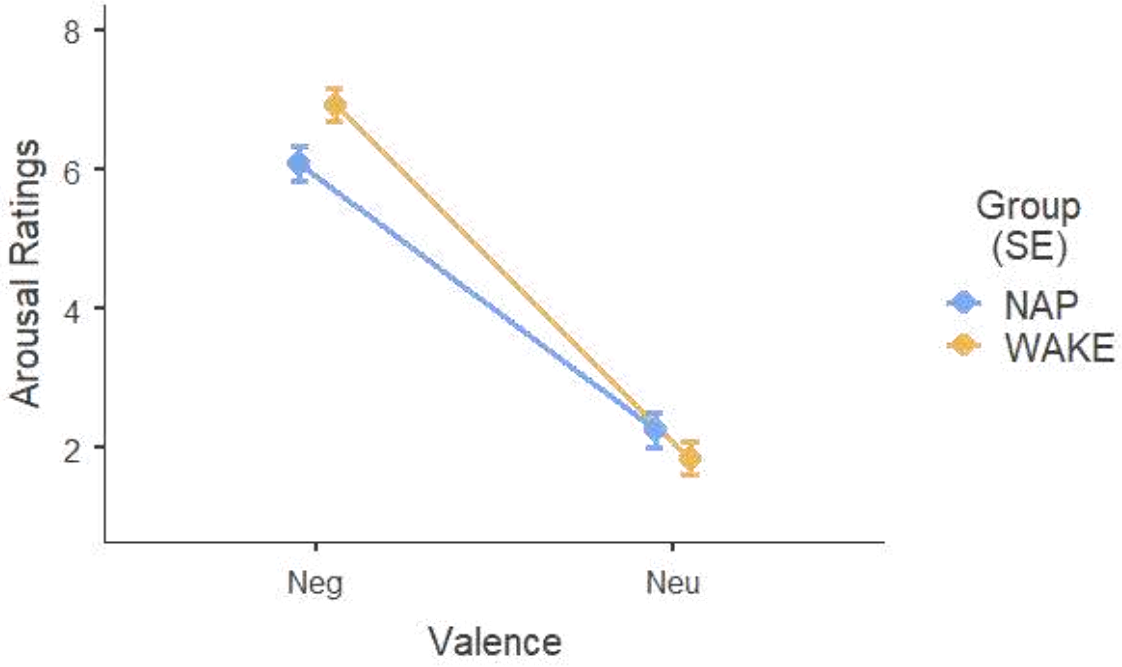

Figure 4.3. Arousal Ratings between NAP and WAKE Group. Negative stimuli (NEG) were rated as more activating than neutral images (NEU) by both groups. The WAKE group indicated more arousal in response to negative stimuli relative to the NAP group. Error bars correspond to the standard error of the mean.

The exploratory analysis including the differentiation between participants who entered REM sleep (n=9) and those who remained in NREM (n=11) only replicated the significant effects mentioned above. REM and NREM group showed nearly identical trends in arousal ratings across the three sessions.

4.4. Heart Rate Deceleration

Fixed and random effects of the LMM conducted for HRD could explain 26.8% over the total (expected) variance. Averaged across the 6-second time window, participants

showed greater HRD when exposed to negative (M = -1.15, SD = 2.45) relative to neutral stimuli (M = 0.44, SD = 2.25). This difference was found to be statistically significant ($F_{1, 1515.3}$ = 211.94, p = <.001). Likewise, the main effect Session reached significance ($F_{1, 43.3}$ = 26.79, p = <.001). With regard to the two-way interactions, Time*Valence was observed to be statistically significant ($F_{5, 1515.3}$ = 6.83, p = <.001). The direction of this effect is illustrated in Figure 4.4.: negative stimuli evoked a greater heart rate deceleration compared to neutral stimuli.

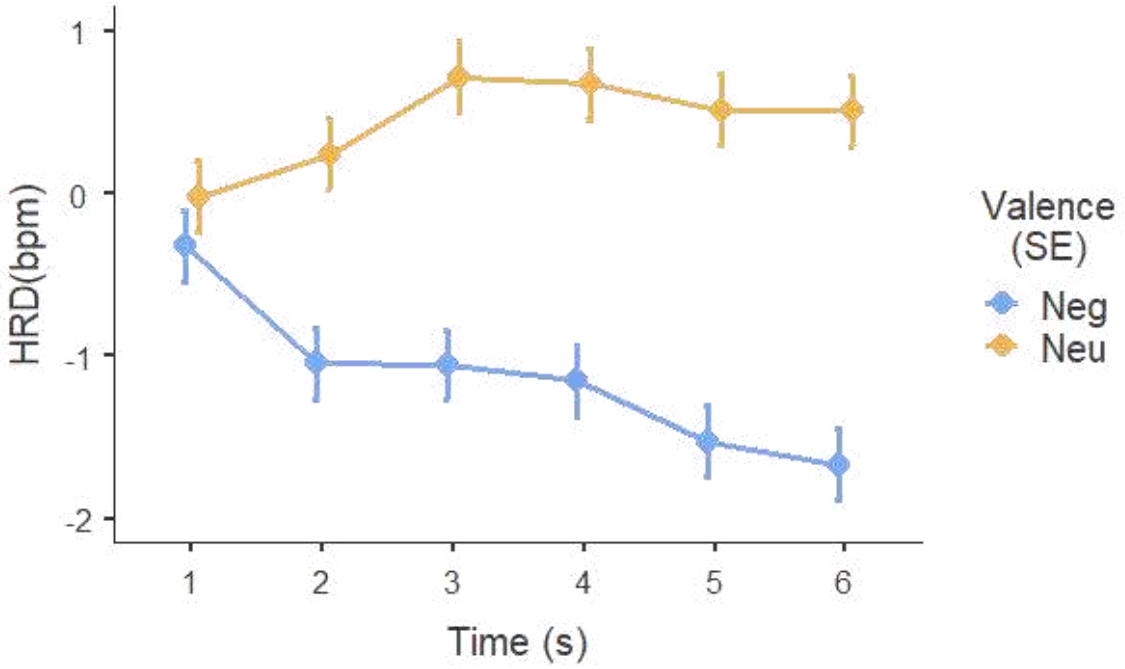

Figure 4.4. Heart Rate Deceleration (HRD) in Response to Negative and Neutral Stimuli. Heart rate deceleration was greater when participants were present with negative stimuli (NEG) relative to neutral stimuli (NEU). Error bars represent the standard error of the mean.

Additionally, a significant two-way interaction of Group*Valence ($F_{1, 1515.3}$ = 6.04, p = 0.014), as well as Session*Valence ($F_{2, 1515.3}$ = 8.28, p = <.001) was observed, signifying a difference in HRD for negative compared to neutral stimuli based on group membership, and test session, respectively. A significant effect was also found for the three-way interaction of Group*Session*Valence ($F_{2, 1515.3}$ = 5.90, p = 0.003). Post-hoc analysis regarding the three-

way interaction revealed significant decrease in HRD from T0 to T1 for negative stimuli in the NAP group (t_{1516} = -5.07, p_{holm} = <0.001). Illustrated by Figure 4.5., HRD in the NAP was more pronounced at T0 compared to the assessment after two days at T2 when presented with negative stimuli (t_{1516} = -1.58, p_{holm} = <0.001).

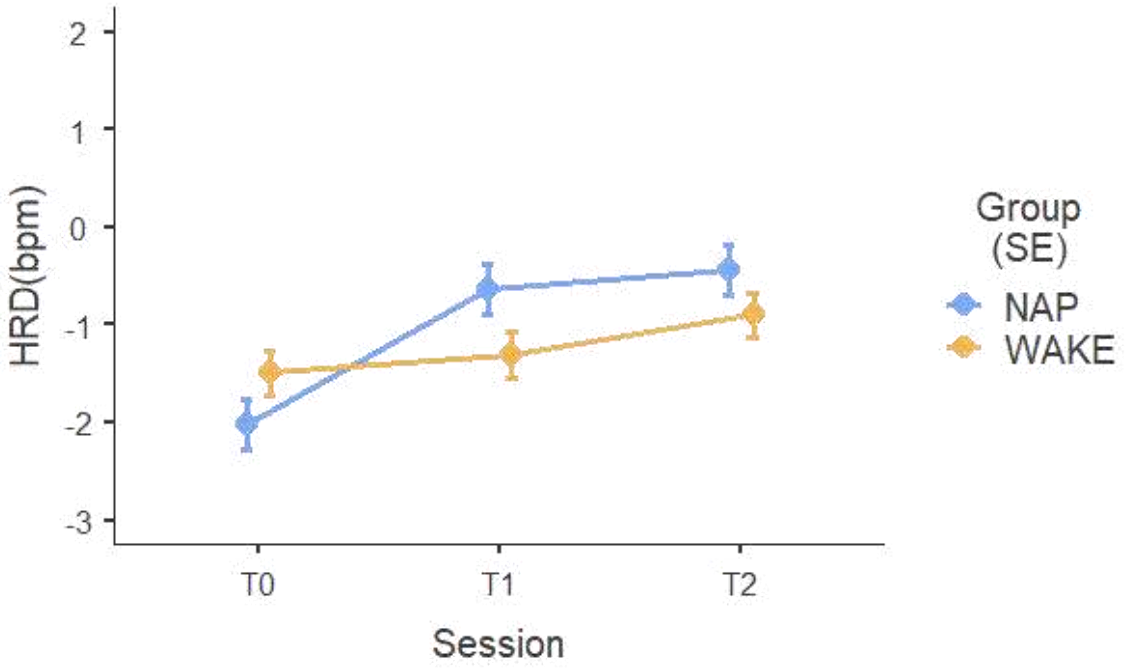

Figure 4.5. Heart Rate Deceleration (HRD) for Negative Stimuli across T0, T1 and T2. The graph displayed the HRD in both the NAP and WAKE group, averaged across the 6-second exposure to negative stimuli during the three test sessions. The NAP group showed a decrease in HRD from T0 to T1, that then remained consistent from T1 to T2. Conversely, HRD in the WAKE group remained relatively stable across all three assessments. *Bpm:* beats per minute. Error bars correspond to the standard error of the mean.

Interestingly, Figure 4.6. demonstrates that, when presented with neutral pictures, the WAKE group displayed a significant decline in HRD from T0 to T1 (t_{1519} = -5.32, p_{holm} = <0.001), and an increase in HRD from T1 to T2 (t_{1516} = 3.64, p_{holm} = 0.011).

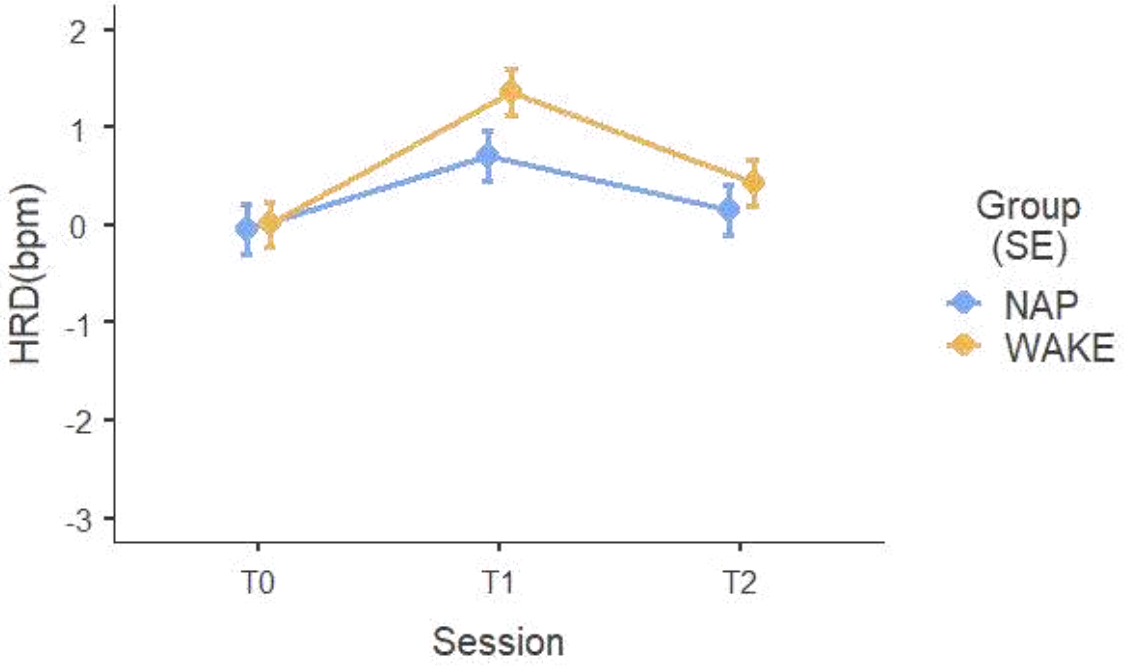

Figure 4.6. Heart Rate Deceleration (HRD) for Neutral Stimuli across T0, T1 and T2. The graph illustrates the HRD in both the NAP and WAKE, group averaged across the 6 seconds of exposure to neutral stimuli during the three test sessions. The WAKE group exhibited a decline in HRD from T0 to T1, which then increased again from T1 to T2. In contrast, HRD in the NAP group remained relatively consistent throughout all three assessments. *Bpm:* beats per minute. Error bars represent the standard error of the mean.

In addition to a replication of the abovementioned significant effects, the exploratory analysis concerning the specific effect of REM and NREM sleep showed a significant two-way interaction of Group*Session ($F_{4,\ 1486.1} = 3.28$, $p = 0.011$). A visual inspection of three-way interaction of Group*Session*Valence indicated different changes in HRD in the three groups in response to negative and neutral stimuli across the testing sessions. Exposure to negative stimuli evoked a trend of HRD in the REM group which rather resembled the one seen in participants of the WAKE group than the NREM group (see Figure 4.7.).

Conversely, when presented with neutral stimuli the REM group displayed no apparent change in HRD, neither from T0 to T1, nor from T0 to T2 (see Figure 4.8.). The direction of changes in HRD was similar in the NREM and WAKE group.

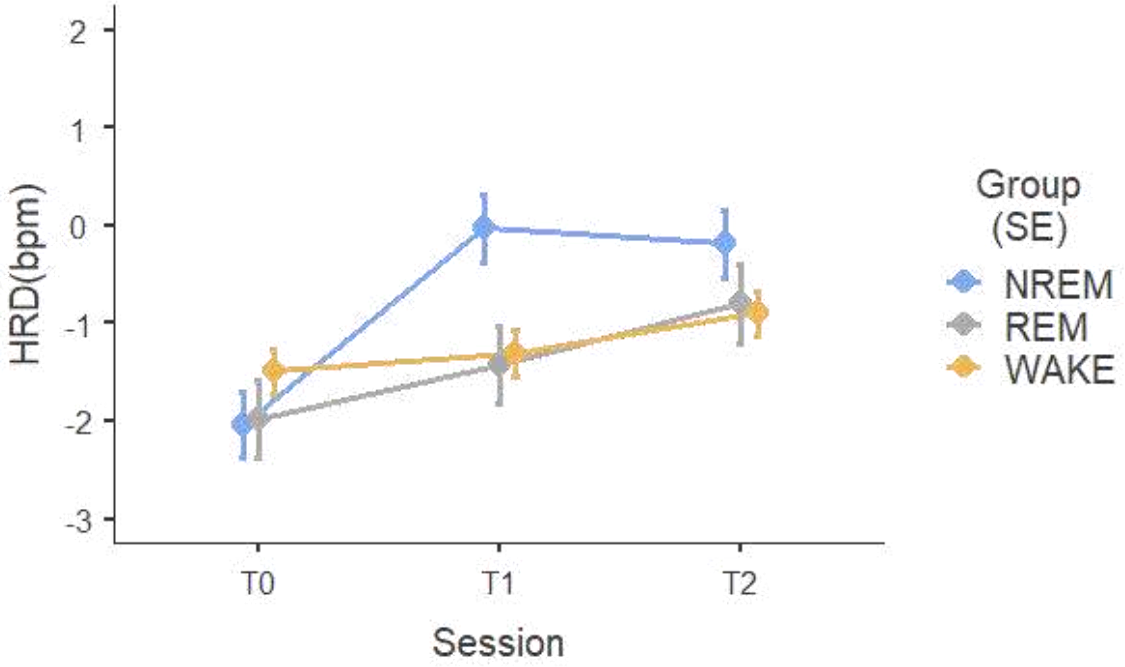

Figure 4.7. Heart Rate Deceleration (HRD) for Negative Stimuli across T0, T1 and T2. The graph displayed the HRD in the REM, NREM and WAKE group, averaged across the 6-second exposure to negative stimuli during the three test sessions. The REM group displayed a decrease in HRD similar to the WAKE group. Bpm: beats per minute. Error bars correspond to the standard error of the mean.

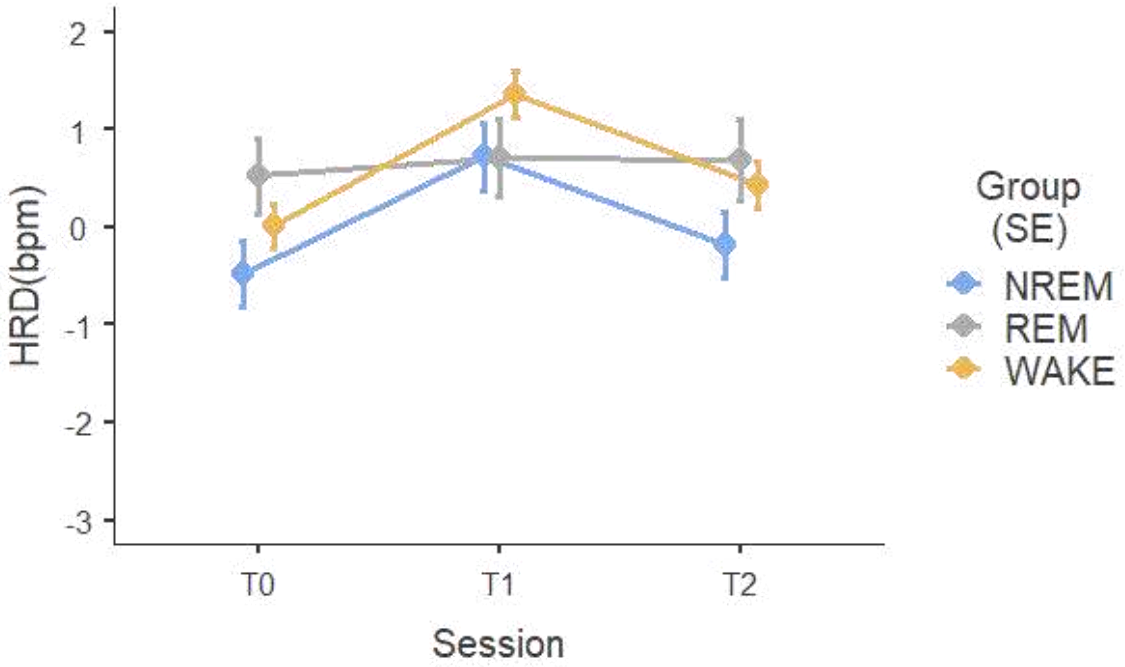

Figure 4.8. Heart Rate Deceleration (HRD) for Neutral Stimuli across T0, T1 and T2. The graph illustrates the HRD in the REM, NREM and WAKE, group averaged across the 6 seconds of exposure to neutral stimuli during the three test sessions. HRD in the REM group remained relatively stable throughout all three assessments. *Bpm:* beats per minute. Error bars represent the standard error of the mean.

5. Discussion

5.1. Key Findings and Hypotheses

The present study sought to explore the influence of sleep on emotional reactivity to both neutral and negative stimuli. Consistent with the theoretical framework of the SFSR model (Walker & van der Helm, 2009), *H1* predicted that*: Emotional reactivity for negative vs. neutral stimuli decreases from T0 to T1 for both subjective and cardiac measurements in the NAP group compared to the WAKE group.* With regard to self-reports a decrease in emotional reactivity was anticipated to manifest as less negative valence ratings and lower arousal ratings, and, physiologically, as a decrease in heart rate deceleration (HRD). A significant main effect of Valence observed in all three measures confirmed the validity of the assumed differentiation between negative and neutral stimuli. However, as hypothesized by *H1*, the expected change in emotional reactivity immediately following the daytime nap was only found in the physiological measure: the NAP group exhibited a significant decrease in HRD from T0 to T1 in response to negative stimuli, while the HRD in the WAKE group remained stable. Conversely, the WAKE group displayed a greater decline in HRD from T0 to T1 to neutral stimuli relative to the NAP group.

To explore the by the SFSR proposed pivotal role of REM sleep in emotional processing, the alternative hypothesis *H1a)* posited: *A greater reduction in emotional reactivity for negative vs. neutral stimuli assessed with both subjective and cardiac measures is expected from T1 to T0 for the REM group relative to the NREM and WAKE groups.* Neither the results based on self-reports nor the physiological data were indicative for expected effect. In terms of self-reports, the REM group closely resembled the NREM group. Visually, a distinct pattern of change in HRD was evident for the REM group, differing from that of the NREM and WAKE group. However, the direction was not in alignment with *H1a)*. The NREM showed the greatest decrease in HRD to negative stimuli from T0 to T1, while

the decline for both REM and WAKE group was very small. In contrast, in response to neutral stimuli, there was no apparent change in HRD for the REM group.

To assess whether the effect of sleep on emotional reactivity emerges in the long-term *H2* predicted that: *Emotional reactivity for negative vs. neutral stimuli decreases from T0 to T2, with a greater reduction in the NAP group relative to the WAKE group for both subjective and cardiac measurements*. The results from neither self-reports nor cardiac measures corroborated *H2*. There was no statistically significant difference in terms of valence and arousal ratings. In the NAP group no further decrease in HRD in response to negative stimuli was detectable, as HRD at T2 remained at the same level as T1. In contrast, HRD to negative stimuli in the WAKE group remained relatively stable throughout all three assessments. Intriguingly, the WAKE group displayed a significant increase in HRD from T1 to T2, engendering a HRD similar to the one observed at T0.

Regarding the particular influence of REM sleep, *H2a)* stated that: *Emotional reactivity for negative vs. neutral stimuli decreases from T0 to T2, with the greatest reduction in the REM group compared to the NREM and WAKE group for both subjective and cardiac measurements*. Solely the findings based on valence ratings were indicative for the effect assumed by *H2a)*. When presented with negative stimuli, the REM group showed the least negative valence ratings followed by the NREM and then the WAKE group when assessed two days after the daytime nap. Nonetheless, no distinct trend for the REM group was discernible for arousal ratings, similarly the by *H2a)* expected effect was not supported by the results obtained with HRD.

5.2. Interpretation and Comparison with Existing Literature

The observed results suggest a different temporal unfolding of the effect of a daytime nap depending on whether emotional reactivity is assessed physiologically, by means of cardiac measures, or subjectively.

Physiologically, the findings replicated the commonly observed phenomenon that exposure to negative stimuli engenders an increase in HRD (Bradley & Lang, 2009). However, at odds with the results by Bolinger et al. (2019) that initially motivated the exploration of the temporal unfolding of the effect of sleep, one intervention of daytime nap was observed to already evoke the maximal depotentiation of HRD, as assessment two days following the experimental manipulation did not result in any further decrease. Given that the same effect was not observable in the WAKE group, this finding cannot be attributed to the mere passage of time. REM sleep did not appear to be particularly involved in mediating this effect, as the greatest decrease in HRD was found for NREM group. However, the involvement of distinct sleep stages requires further investigation given the small sample size in NREM and REM group.

Conversely to the observable effect in physiological measures immediately following the experimental manipulation, an indication for a change in self-reported measures was only noticeable two days subsequent to when participants were instructed to take a daytime nap, and solely for valence ratings. An explanation for why the same effect did not reveal itself when arousal was examined might be the methodological assessment of the former. Although non-verbally mediated, the 9-point Likert scale of the Self-Assessment Manikin might not permit a sufficient interoceptive discrimination of subtle somatovisceral changes. Indeed Grob & Schimmack (2000) as well as Strongman (2003) suggested that the arousal dimension of the circumplex model of emotion is insufficiently defined as more attention has been given to the dimension of valence and its assessment.

Nonetheless, the emerging effect of sleep with regard to valence ratings two days following the assigned daytime nap implies that when emotional reactivity assessment involves also cognitive processes, more iterations of (overnight) sleep cycles may be required for the beneficial influence of sleep to occur. These observations align with those by Bolinger et al. (2019) in which a follow-up assessment 10 days later yielded differing effects on valence ratings. Similarly, albeit investigating emotional memory instead of emotional reactivity, the study by Wagner et al. (2001) suggested that changes may be more easily noted after several nights of sleep. Moreover, the authors attributed an essential role to the presence of REM sleep. Likewise in this study, the presence of REM sleep directly following the exposure to negative stimuli seemed to particularly impact the effect of sleep. A differential effect became apparent for the REM group, as they rated negative stimuli less negatively compared to the NREM and WAKE group.

The discrepancy in the temporal unfolding of the effect in physiological and self-report measures might be due to differences in the extent to which the physiological and cognitive processes are involved: HRD represents an orienting response governed by the autonomic nervous system and subcortical structures (Kuniecki et al., 2002), whereas subjective ratings also require cognitive processes, as they arise from both physiological and slower cognitive appraisal of the physiological response (Schachter & Singer, 1962). It could be possible, that while the cognitive reappraisal of the emotional event requires more iterations and especially reactivation during REM sleep, the effect of the physiological response already emerges after a single intervention of daytime sleep. Nevertheless, it should be emphasized that the present data on REM and NREM sleep is preliminary, engendering further research evermore necessary to disambiguate the specific contribution of sleep, and of different sleep states.

Finally, even though the hypotheses were phrased within the framework of SRSF hypothesis (Walker & van der Helm, 2009), the results observed were rather but not strictly in favour of the theoretical model. This might be due to the models lacking specificity in terms of the temporal unfolding of the effect as well as the specific measurement employed to assess emotional reactivity. Walker & van der Helm (2009) only offered a vague description regarding the temporal revelation of the effect in that if the emotional tone is not removed from the memory content during the first night of sleep, a subsequent attempt to discharge the affective tone is commenced in the ensuing nights. Results obtained by previous (see Bolinger et al., 2019, Wanger et al., 2001) as well as this study encourage a refinement of the assumptions made in the model, otherwise it defies any attempt to corroborate or falsify it. Additional assumptions could potentially concern the specific measurements of emotional reactivity and their involvement of autonomic and cognitive processes.

5.3. Limitations

The present study represents an experimental manipulation of sleep relative to an equivalent period of wakefulness, and thus allows an evaluation of the causal relationship between daytime nap and emotional reactivity in a controlled laboratory setting. As the study employed a daytime nap paradigm, observed findings are less likely to be confounded by effects of the circadian rhythm or other external factors, e.g., cortisol levels, levels of alertness, light exposure or meal timing, as subjects were tested at the same time of the day.

However, the presented results have be considered in light of some limitations. Firstly, it should be emphasized that the results obtained may be restricted to daytime nap rather than overnight sleep, due to qualitative and quantitative differences between the two. While overnight sleep is governed by a consistent and predictable schedule, naps are less regular and pose the risk to disrupt the regular sleep-wake cycle. Typically, overnight sleep

exceeds daytime naps in duration, therefore allowing for several sleep cycles including multiple successions of NREM and REM sleep, whereas daytime naps usually comprise less and lighter stages of sleep. Nonetheless, owing to a successful experimental manipulation participants included in the NAP group were able to enter REM sleep. Yet, the discussion of distinct sleep stages has to be taken with the caveat of daytime naps limitation in REM cycles and above all a limited sample size for the REM and NREM group.

Additional limitations pertain to the measurement and equally the induction of emotional responses. For science as a field, there is a strong inclination toward parsimony, although the natural world often presents a much more complex picture. This raises the question of whether assessing emotions based on two dimensions can account for the whole range of emotions a person can possibly experience. Even the authors that devised the circumplex model of emotions acknowledged a lacking adequate explanation for how certain negative emotions, i.e., fear, jealousy, anger and shame differ from each other (Russell, 2003). Russell (2003) himself proposed a solution by adding the attributional stage of perception in order to take discrete emotional episodes into consideration, while Lindquist (2013) suggested the addition of conceptualization. Furthermore, the differentiation between types of emotions beyond valence is also questionable, as the vulnerable dimension of the model is arguably arousal. Suggested by Grob & Schimmack (2000) as well as Strongman (2003) too much attention has been given to valence and too little to arousal, contributing to an insufficient definition of the dimension.

However, despite the criticism voiced, the circumplex model of emotions (Russell & Carroll, 1999) should not be dismissed as evidence to support it exists. Yet, given the model's popularity a more precise definition of the dimension of arousal might prove beneficial for future studies. In addition to the measurement of emotional reactivity, the induction of the emotional response itself might also be made subject of debate. The stimuli utilized in this

study may lack adaptive valence. Hence, future studies could benefit from the incorporation of more ecologically relevant stimuli, such as videos or virtual reality experiences.

Further limitations regard the study's sample as well as the composition of NAP and WAKE group. Since the study mostly consisted of university students it might not be a representative sample of the broader population, due to differences in age, education and socio-economic background. This can limit the external validity of the study as its findings may not be applicable to non-student populations. An additional bias may be introduced by the attrition of roughly 23% participants of the NAP group, as dropout patterns could lead to less comparable groups due to the loss of certain characteristics. Yet, the analysis of demographic and psychological data comparing the NAP and WAKE group revealed only one significant difference on the Epworth Sleepiness Scale (ESS), which measures the propensity to fall asleep in various daily life situation in order to differentiate people with excessive daytime sleepiness from awake individuals. Lastly, threatening to the validity and generalizability of the results is a potential gender bias, as gender was not equally distributed across the WAKE and NAP group, with a higher proportion of females in the WAKE compared to the NAP group. Indeed, differences concerning behavioural and physiological emotional reactivity between gender have been reported (Filkowski et al., 2017), potentially rendering gender a confounding variable. However, it should be noted that the data obtained is preliminary, and subsequent data collection for the larger project it was embedded in will ensure a balanced representation of gender within the groups.

6. Conclusion

The present study investigated the influence of sleep on emotional reactivity in response to neutral and negative stimuli. The results suggest a different temporal unfolding of the effect for physiological and subjective assessment. Maximal decrease in emotional reactivity, indicated physiologically by a decreased HRD, was observed immediately following one intervention of daytime napping, with a crucial role for NREM sleep in this effect. When emotional reactivity assessment involves also cognitive processes, such as in subjective measures, more iterations of (overnight) sleep cycles may be required for the beneficial influence of REM sleep to occur. However, due to the data at hand being preliminary, the specific involvement of sleep stages and their contributions to emotional reactivity warrants further exploration, using adequate sample sizes. The results emphasize the need for a specification of the SFSR hypothesis as well as a refinement of the circumplex model's dimension, particularly arousal, to adequately evaluate the involvement of sleep as well as sufficiently capture emotional experiences, respectively. The use of more ecologically relevant stimuli such as movies or virtual reality experiences in future study is advisable.

In conclusion, our research highlights the complex relationship between sleep and emotional reactivity and emphasizes the need for further investigations to elucidate the underlying mechanisms. While the study offers valuable insights, it likewise opens the door for continued exploration and refinement in this field of study.

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