THE EMOTIONAL BRAIN AND SLEEP
A review of the relationship between sleep and emotional brain functioning.

Bachelor Degree Project in Cognitive Neuroscience
Basic level 22.5 ECTS
Spring term 2018

Hanna Lindhe

Supervisor: Petri Kajonius
Examiner: Katja Valli
Abstract

Why do we need to sleep? Not only is getting enough sleep important for our overall health and well-being, it is perhaps of utmost importance for normal brain functioning. Scientific findings derived from studying sleep deprivation suggests that sleep also plays an important role in our emotional functioning, which has led researchers to propose a causal and intimate relationship between sleep and emotional brain functioning. Without sleep it seems as our emotional processing become impaired in various ways. Along with advances in cognitive neuroscience, it is now possible to characterize mechanisms underlying emotional brain processes. In pursuit of the possible functions of sleep, researchers have also proposed that rapid eye movement sleep, might support a process of affective brain homeostasis and recalibration that optimally prepares the organism for next-day social and emotional functioning. This thesis reviews current behavioral and neurophysiological evidence focused on the relationship between sleep and emotional brain functioning, and the role of rapid eye movement sleep in emotional processing.

*Keywords:* sleep, emotion, sleep deprivation, rapid eye movement sleep
## Table of Content

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prologue</td>
<td>4</td>
</tr>
<tr>
<td>Introduction</td>
<td>4</td>
</tr>
<tr>
<td>Clarifying the Terminology</td>
<td>7</td>
</tr>
<tr>
<td>Glossary of Terms</td>
<td>7</td>
</tr>
<tr>
<td>The Basics of Sleep</td>
<td>8</td>
</tr>
<tr>
<td>Circadian Regulation</td>
<td>10</td>
</tr>
<tr>
<td>Neurobiology of the Sleeping Brain</td>
<td>11</td>
</tr>
<tr>
<td>Neurochemicals Involved in Sleep</td>
<td>12</td>
</tr>
<tr>
<td>Neural Correlates of Sleep</td>
<td>14</td>
</tr>
<tr>
<td>Healthy Sleep</td>
<td>16</td>
</tr>
<tr>
<td>Sleep Loss</td>
<td>18</td>
</tr>
<tr>
<td>Emotion</td>
<td>19</td>
</tr>
<tr>
<td>Concepts of Emotion</td>
<td>21</td>
</tr>
<tr>
<td>The Emotional Brain</td>
<td>22</td>
</tr>
<tr>
<td>The Role of Sleep in Emotional Brain Functioning</td>
<td>24</td>
</tr>
<tr>
<td>Observational Studies</td>
<td>25</td>
</tr>
<tr>
<td>Experimental Studies</td>
<td>26</td>
</tr>
<tr>
<td>Neurobiological Underpinnings of Sleep and the Emotional Brain</td>
<td>28</td>
</tr>
<tr>
<td>A Proposed Function of REM Sleep Homeostasis</td>
<td>31</td>
</tr>
<tr>
<td>Discussion</td>
<td>34</td>
</tr>
<tr>
<td>Conclusion</td>
<td>39</td>
</tr>
<tr>
<td>References</td>
<td>41</td>
</tr>
</tbody>
</table>
Prologue

“Sleep that knits up the ravelled sleave of care
The death of each day’s life, sore labour’s bath
Balm of hurt minds, great nature’s second course,
Chief nourisher in life’s feast. (Shakespeare, Ed., 1877, 2.2.21-23)

My interest in sleep and emotion originates from memories from my childhood – trying to wake up my older brother with poor results. Usually, we would take turns in the family of who was supposed to wake him up – yes, it was that bad. I always had a bad feeling in the stomach when it was my turn considering I never knew what reaction I would get. There were so many feelings in this young boy who, if he could, probably would have slept the entire afternoon. I often wondered, and still do, what it was that made us so different from each other concerning our sleep habits. This trip down memory lane gave rise to many questions. Why do people need different amounts of sleep to function? What happens to our emotional responses when we miss out on sleep? What happens in the brain when we are sleep deprived? An adaptation of these questions is what I aim to answer in the scope of this thesis.

Introduction

Sleep is such a naturally occurring phenomenon in all mammals that it is rarely questioned by the public. By contrast, sleep research has attempted to discover why we sleep for quite some time and are still trying to grasp its functions.

For decades sleep was thought of as a state where the brain was shut off and at rest without consciousness (Paterson, 2012). As cognitive neuroscience has evolved we now know that some areas in the brain are indeed highly active during sleep, especially in one of the stages which are characterized by more activity than in waking. The scientific interest in
sleep emerged already in medieval times (Espie & Morin, 2012). However, it was not until the 17th century that a first theory of sleep mechanisms, by Descartes, was described. Descartes proposed that the pineal gland helped maintain alertness and that a “loss of animal spirit” from the pineal gland caused the ventricles to collapse which induced sleep (Espie & Morin, 2012). In the early 1950’s the research area of sleep grew and became much larger as set in motion by the discovery of rapid eye movement (REM) sleep by Aserinsky and Kleitman (1953). Not too long thereafter several major discoveries were made and already in the mid-1960's several features of sleep were sufficiently established that a standardized manual for sleep scoring were published (Espie & Morin, 2012). As 20 years went by the two-process model by Borbély (1982), the most widely known theory of sleep regulation was proposed. The two-process model demonstrates the interaction between homeostatic and circadian functions (Espie & Morin, 2012), which essentially is the control and maintenance of sleep and wakefulness during the period of approximately 24 hours. Much has happened since then and sleep research is now a vast field with several subdivisions and various areas of interest.

Human sleep is, based on the American Academy of Sleep Medicine scoring manual (Berry et al., 2012), subdivided into two categories - REM sleep and non-rapid eye movement sleep (NREM). Collecting certain physiological and neural activity during the stages of sleep have made it possible to distinguish between the two as well as further investigating their possible functions. As neuroscience continuously attempts to discover the functions of sleep, an increased amount of research on the subject has been published and it seems as scientists are finally beginning to grasp the mechanisms and functions of sleep. Heretofore, the most widely accepted cognitive theory regarding sleep and cognition is that sleep plays an important role in memory and learning (Gazzaniga, Ivry, & Mangun, 2013). The most common way of investigating the role of sleep in behavior and cognition has been
through sleep deprivation, hence many of the studies reviewed in this thesis will be under such conditions.

In our modern-day society, it seems as sleeping is all too often compromised due to not having enough time. Insufficient sleep has become a public health problem and is associated with impairments in both physical and mental health (Strine & Chapman, 2005). Consistent with this, research findings suggest that good sleep is important for normal brain function, well-being and overall health (Buysse, 2014). Findings derived from studying sleep deprivation have led to the emergence of a new area aimed at investigating a relationship between sleep and emotional brain functioning. Fortunately for such investigations, advances in cognitive neuroscience have made it possible to characterize potential mechanisms underlying emotional brain processes (Goldstein & Walker, 2014). These advances have paved the way for a proposed intimate and causal relationship between sleep and emotional brain functioning. As this sleep - emotion research area has progressed several review papers have been published regarding this relationship, accompanied by interesting frameworks (e.g. Goldstein & Walker, 2014; Palmer & Alfano, 2017). One of these two frameworks, namely the REM sleep homeostasis framework (Goldstein & Walker, 2014), will be discussed later.

As previously mentioned scientists are beginning to grasp the functions of sleep, especially regarding the influence of sleep on emotional brain functioning. Growing support for such functions comes from both behavioral and neuroimaging studies on sleep and emotion (Killgore, Balkin, Yarnell, & Capaldi, 2017; Krause et al., 2017; Motomura et al., 2013; Yoo, Gujar, Hu, Jolesz, & Walker, 2007).

The aim of this thesis is to review the proposed relationship between sleep and emotional brain functioning as well as a proposed function of REM sleep. Along with reviewing emerging evidence that either further illustrates the relationship or lack thereof. The following question is aimed to be answered;
What is the role of sleep in emotional brain functioning?
What is the role of REM sleep in emotional processing?

The focus will therefore mainly lie on the suggested influence of sleep on emotional brain functioning and a proposed function of REM sleep. To answer this question the thesis will provide a basic overview of sleep and emotion, followed by data suggesting their neural basis. These data will be implicated in theories on and scientific evidence for the relationship between sleep and emotional brain functioning and, REM sleep and emotional processing.

The upcoming section will provide a glossary of terms to avoid confusion of concepts.

**Clarifying the Terminology**

The sleep – emotion area struggles with consensus, which is highly noticeable as researchers use the concepts of emotion differently. All too often emotions, mood and affect, and emotion regulation and affect regulation are used interchangeably which is problematic. Poor delineation and insufficient distinctions among emotional processes cause conceptual and empirical challenges in the field which is unfortunate as many of the concepts are overlapping while others are somewhat distinct. It is therefore necessary to present a glossary of the terms and how they will be used in this thesis as an attempt to eliminate any possible confusion. Nevertheless, it should be noted that it might not be possible to circumvent the obvious conceptual difficulties that exist in the field.

**Glossary of Terms**

- **Affect** – an umbrella term often used interchangeably for describing emotions, mood, and positive- or negative affect
• *Emotion* – a process triggered by internal or external stimulation which includes changes in multiple systems; subjective, physiological, behavioral, relational (short-lived event, response to external stimulus)

• *Emotion regulation* (i.e. *affect regulation*) – the process by which people influence the quality, intensity, and duration of emotions

• *Emotion-related brain areas* – specific brain areas that have been suggested to underly emotional behavior

• *Emotional brain functioning* – functioning of the network of brain structures underlying emotional behavior

• *Emotional dysregulation* – when an emotional response is poorly modulated thus not fitting the conventionally accepted range of emotive response (e.g. aggressive outburst, hypersensitivity)

• *Emotional processing* – how people processes emotions across process domains (e.g. affective reactivity, emotional discrimination, emotional expression)

• *Emotional reactivity* – threshold, peak intensity, rise time, and recovery time of a response to emotional stimulation

• *Mood* – emotional tone that is unprovoked by a stimulus which refers to a stable condition (sustained event, often internally generated)

• *The emotional brain* – a term for the network of brain structures (e.g. the limbic system) underlying emotional behavior

**The Basics of Sleep**

Sleep is a universally naturally reoccurring altered state of consciousness and we spend one third of our lives sleeping (Paterson, 2012). Electroencephalographic (EEG) studies on animals and humans have made it possible for the field of neuroscience to collect
Information on cortical and subcortical systems that generate the characteristic rhythms of sleep stages, and neuroimaging studies have allowed inferring which areas are active during sleep stages (Hobson & Pace-Schott, 2002). In humans, sleep is divided into two categories whose names are based on rapid eye movement or lack thereof – REM sleep and NREM sleep. NREM sleep is further subdivided into three (previously four) stages N1-N3 and they differ in depth of sleep (Berry et al., 2012). N1 being the lightest sleep stage and N3 or so-called slow wave sleep (SWS) being the deepest stage. REM sleep and NREM sleep have also been found to differ, among other things, in neurochemical- and neurological activity (Walker & van Der Helm, 2009).

When measuring stages of sleep in humans the used methodology is polysomnography (PSG), which is a combination of three techniques that record changes in brain waves with electroencephalography and at the same time measure eye movements with electrooculography and muscle activity with electromyography. Together these three collect and assemble physiological signs that serve as indicators for differentiating between the stages of sleep. It is possible to distinguish the four stages of sleep as they significantly differ in terms of physiological signs, EEG amplitude, and oscillations. The voltage amplitude is the size of the signal in EEG recordings and the frequency is the number of oscillations per seconds (Paterson, 2012). These frequencies are divided into bands of beta, alpha, theta, and delta thus making it easier for distinguishing between the stages.

PSG of NREM sleep shows that muscle tone is diminished and there are no eye movements or very slow eye movements (Peigneux, Urbain, & Schmitz, 2012). By contrast, PSG of REM sleep show loss of muscle tone and rapid eye movements. EEG measures of wakefulness show high-frequency waves and low amplitude as well as high muscle tone. When resting with eyes closed EEG shows alpha waves of low frequency and increased amplitude. The characteristics of N1 is theta waves and low frequency, and high amplitude.
N2 show delta range slow frequency along with periodical and transient events so-called spindles and k-complexes (negative sharp waves) with increased high amplitude. N3 show delta waves or slow waves with high amplitude. REM sleep shows mixed frequency and low amplitude, resembling N1, and saw-tooth waves in conjunction with rapid eye movements. These characteristics reflect changes within and between neuronal populations during the different stages of sleep.

Throughout the night REM sleep and NREM stages 1-3 fluctuates in cycles of an average 90 minutes, called an ultradian cycle. N3 or SWS occurs mostly at the beginning of the early sleep hours. The first REM sleep occurs after approximately 90-100 minutes after sleep onset but is brief and only last for about 5-10 minutes (Peigneux et al., 2012). However, the duration of REM sleep increases with each cycle and has its longest duration just before waking. Depending on for how long we sleep, the average amount of sleep cycles under normal nocturnal conditions is up to five completed cycles – that is if we sleep for 8 hours (Peigneux et al., 2012). REM sleep is often understood as the stage where we dream, although we dream in NREM sleep as well. REM sleep has been linked to such functions as consolidation of emotional memories (Palmer & Alfano, 2017). REM sleep has also quite recently been suggested to have a homeostatic and recalibrating function (Goldstein & Walker, 2014) which will be further explained in a subsequent section.

Circadian Regulation

Sleep is governed by a so-called circadian or internal clock that regulates our sleep-wake pattern during an approximate 24-hour period (van Someren & Cluydts, 2013). This is called the two-process model of sleep regulation and was proposed by Borbély in 1982 (Paterson, 2012). Since then it has been recognized as the conceptual framework for sleep regulation. The two-process model is based upon the interaction of a homeostatic process (Process S) and the process of a circadian pacemaker (Process C). The homeostatic process is
believed to function by accumulating a kind of “sleep pressure” throughout the day and in synchrony with sleep, it decreases. Adenosine has been suggested to be the main neurochemical linked to this sleep pressure (Peigneux et al., 2012). This circadian process does a tremendous job every day by trying to give us humans optimal conditions for optimal functioning throughout the day, such as making us more alert in the early hours of the day and tired in the evening (van Someren & Cluydts, 2013). What makes this circadian phenomenon so interesting is that even though we miss a night of sleep, the 24-hour process remains untouched. When we miss a night of sleep we still experience the same pattern of sleepiness and alertness as if we had slept (Dijk & Lazar, 2012).

The circadian pacemaker and the homeostatic process have both been linked to substrates of the hypothalamus – the suprachiasmatic nucleus (SCN) and the ventrolateral preoptic area (VLPO) (van Someren & Cluydts, 2013). Stripping away most details, the SCN works by using light information from the optic nerves to reset its inherent time inaccuracy to a 24-hour cycle and increased VLPO activity promotes sleep and inhibits locus coeruleus and raphe nuclei which are wake-promoting nuclei. Sleep is influenced by several systems such as the hypothalamus-pituitary-adrenal axis, the interaction with neuropeptides, steroids, and estrogens (van Someren & Cluydts, 2013) which is difficult to cover in the scope of this thesis. However, the neural correlates of sleep will be demonstrated in the upcoming section on the neurobiology of the sleeping brain.

**Neurobiology of the Sleeping Brain**

The neurobiological underpinnings of sleep and emotion have been investigated with different types of neuroimaging techniques such as positron emission tomography (PET), EEG and functional magnetic resonance imaging (fMRI).

As previously mentioned the sleeping brain has been an area of interest in the field of neuroscience for two decades which have led to a growing body of research in this area.
Several studies (e.g., Dang-Vu et al., 2010; Hobson & Pace-Schott, 2002; Schwartz & Maquet, 2002) have found various areas in the brain that are specifically active during REM sleep as compared to wakefulness. Brain activity during sleep stages is assumed to not be constant and homogeneous over time but structured by spontaneous transient and recurring neural states (Dang-Vu et al., 2010).

Sleep-wake cycles and alternations between sleep stages are assumed to be controlled, initiated and regulated by activity in specific neuronal populations (Peigneux et al., 2012). These populations are located in the basal forebrain and the hypothalamus which is two broad regions of the brain, and the brainstem and the pons where most sleep promoting/wake-promoting nuclei are. There are also several neurochemical systems that promote cerebral arousal and prevents the organism from falling asleep, such as acetylcholine, histamine, norepinephrine, serotonin, hypocretin/orexin, dopamine and glutamatergic cells. These specific wake-promoting cells have been found in the ascending reticular activating system (ARAS) in the reticular formation (Peigneux et al., 2012). Here, the focus will lie on cholinergic, noradrenergic, and serotonergic neurons because they are of the utmost importance in relation to the emotional brain.

**Neurochemicals Involved in Sleep**

Acetylcholine (ACh) is a neurotransmitter and neuromodulator involved in muscle action, learning and memory (Devi & Fricker, 2013). It has great importance for the autonomic nervous system and is a major neurotransmitter in motor neurons of the spinal cord and parasympathetic nervous system. Motor neurons, neurons within the brainstem and neurons in the basal forebrain (BF) produce ACh in the central nervous system and project to many areas of the brain. Cholinergic (exhibiting or stimulating the activity of ACh) cells have been found to occupy brain areas involved in the main arousal systems in the brainstem and forebrain - pedunculopontine tegmentum (PPT) and laterodorsal tegmentum (LDT) of the
brainstem and the anterior hypothalamus of the BF (Peigneux et al., 2012). Cholinergic cells in PPT nucleus have been found to act by activating several cortical areas that promote wakefulness and cholinergic cells in BF receive and project input from other wake-promoting systems to the cerebral cortex. During REM sleep and wakefulness activation in these neuronal populations have been found to be high and by contrast, during NREM the activity has been found to be strongly diminished (e.g., Datta & MacLean, 2007). It has been suggested that ACh is involved in the generation of desynchronized EEG pattern in REM sleep and wake (Peigneux, et al., 2012). ACh has also been suggested to have great importance for long-term consolidation of emotional learning (McGaugh, 2004).

Norepinephrine synthesizing noradrenergic cells have been found to be located in the locus coeruleus of the pons (e.g., Berridge & Waterhouse, 2003) and project directly to the cerebral cortex, hippocampus, amygdala and other subcortical areas such as the thalamus, hypothalamus, and BF. Noradrenergic neurons are suggested to slow down the initial phase of SWS as well as impact the loss of muscle tone during sleep (e.g., Datta & MacLean, 2007).

Serotonergic synthesizing cells have been found to be located in the raphe nuclei within the brain stem in the reticular formation (Devi & Fricker, 2013). They have been found to project widely throughout the brain and are suggested to be largely involved in modulating sleep-wake cycles and mood. These serotonergic cells are similar to noradrenergic cells as they project almost to the same brain areas (e.g., Morgane, Galler, & Mokler, 2005). Serotonin has been suggested to play a role in maintaining arousal and regulation muscle tone as well as some phasic events of REM sleep (Peigneux et al., 2012).

Adenosine has been suggested to play a role in the homeostatic regulation of sleep as the concentration of adenosine in the basal forebrain cholinergic region has been observed to change with sleep duration and depth. Adenosine is an endogenous compound involved in
biochemical and neuromodulatory processes (Krause et al., 2017). During sleep loss, the levels of extracellular adenosine have been found to accumulate and in synchrony, it declines during recovery sleep (e.g., Schwartz & Kilduff, 2015). This accumulation of adenosine has been suggested to downregulate specific dopamine receptors thus leading to an imbalance of dopamine receptor availability and increasing approach-driven and reward-driven behavior (Krause et al., 2017). These neurochemical changes during sleep are followed by changes in activity in specific brain regions, these will be further examined below.

**Neural Correlates of Sleep**

Using PET, REM sleep have consistently been associated with a neuroanatomical pattern of increased activity in several regions; the PPT, thalamus, BF, cerebellum, caudate nucleus, amygdala, hippocampus, hypothalamus, anterior cingulate cortex (ACC), motor cortex, parahippocampal gyurs and associative posterior temporo-occipital regions (e.g., Braun et al., 1997; Maquet et al., 1996; Schwartz & Maquet, 2002). By contrast, decreases in regional cerebral blood flow have been found in frontal and parietal regions such as prefrontal cortex (middle, inferior, dorsolateral, orbitofrontal cortices), posterior cingulate gyrus, precuneus and inferior parietal cortex, as well as in primary sensory areas (e.g., Braun et al., 1997; Maquet et al., 1996, 2000; Schwartz & Maquet, 2002), indicating that activity in these regions is strongly diminished during REM sleep. In consistency with earlier studies, an fMRI study by Dang-Vu et al. (2010) also suggest that during REM sleep there is a global level of activity not significantly different from wakefulness, and enhanced activity in comparison to waking state specifically in areas of pontine tegmentum, BF, thalamus, amygdala, hippocampus, ACC and temporo occipital areas. The researchers also found the similar decreases during REM sleep in the dorsolateral prefrontal cortex, posterior cingulate gyrus, and inferior parietal cortex.
PET studies during NREM sleep have consistently found decreases in regional CBF and oxygen consumption, as compared to wakefulness or REM sleep. These decreases have been found to occur in subcortical and cortical regions encompassing the dorsal pons and the mesencephalon, cerebellum, thalamus, basal ganglia, BF, anterior hypothalamus, neocortical-prefrontal-, anterior cingulate cortex, precuneus and mesial parts of the temporal lobe (e.g., Braun et al., 1997; Maquet et al., 1997; Maquet, 2000).

Dang-Vu et al. (2010) have made similar observations with fMRI during NREM sleep suggesting that there is a decrease of activity in brainstem, thalamus and several cortical areas including the medial prefrontal cortex (mPFC). The researchers also observed significantly less activation in NREM sleep as compared to REM sleep with reduced activity in regional subcortical areas such as the brainstem, basal ganglia, BF, and cortical regions such as prefrontal cortex (PFC), anterior cingulate cortex (ACC) and prenucleus. These structures are involved in arousal and awakening as well as wakefulness (Peigneux et al., 2012).

During NREM sleep subcortical cholinergic systems in the brainstem and forebrain have been found to become markedly less active along with the activity of serotonergic- and noradrenergic neurons being reduced relative to waking levels (Goldstein & Walker, 2014). These aminergic populations are instead inhibited during REM sleep along with more activity in cholinergic systems in comparison with wake which results in a brain state lacking aminergic modulation and an acetylcholine domination instead (Walker & van Der Helm, 2009).

Scientists are eager to further investigate the function and dynamics of sleep which have generated many reviews on the topic (Goldstein & Walker, 2014; Krause et al., 2017; Palmer & Alfano, 2017; Vandekerckhove & Cluydts, 2010; Walker & van Der Helm, 2009). Interestingly, a new area has taken form on the relationship between sleep and emotional brain functioning and vice versa. The regulation of emotions seems to be particularly
sensitive to the effects of sleep deprivation (Hall, Levenson, & Hasler, 2012) which brings us to the topic of healthy sleep and effects of sleep loss.

Healthy Sleep

How many hours of sleep do we need to function properly? The National Sleep Foundation revisited their sleep recommendations in 2015 which included a new recommendation of prolonged sleep in all the previous age subsections except for adults (Hirshkowitz et al., 2015). They also added new categories of young adults (18-25 y) and older adults (65+ y). According to the recommendations, 7-9 hours of sleep for an adult (25-64 y) is optimal to stay healthy, but the reality is that many people sleep less than 7 hours each night. A study by Liu et al. (2014) shows that approximately 65% of the American population sleeps 7 hours or less. Although 7-9 hours of sleep is suggested for all individuals (Hirshkowitz et al., 2015), there are natural variability of individuals (Paterson, 2012). Two of these are called chronotype (morningness/eveningness) and somnotype (sleepability). Somnotype refers to individual differences in homeostatic regulation of sleep which is the individual need, duration and propensity of sleep such as natural short and long sleepers (Putilov, Verevkin, & Donskaya, 2013). Chronotype instead refers to individual differences in regulation of circadian cycles and is often referred to as either morning larks or night owls (Korf & von Gall, 2013). Hence, the preferred timing for sleep and waking, either waking up early in the morning and going to bed early in the evening or waking up late and going to bed late at night rather than evening (Dijk & Lazar, 2012). Interestingly, individuals with an eveningness tendency as compared to a morningness tendency are more likely to develop depression (Eidelman, Gershon, McGlinchey, & Harvey, 2012). As society is largely based on daytime work and schools, it is perhaps not strange to wonder how this would affect an eveningness person. It seems as it is especially persons with the extreme chronotypes such as extreme morningness or eveningness that suffer the most, and these have been linked with
mood disorders (Eidelman et al., 2012). One could imagine that morning types probably get enough sleep, whereas evening types probably does not. A study investigating mood changes after sleep deprivation in morningness-eveningness chronotypes in healthy individuals (Selvi, Gulec, Agargun, & Besiroglu, 2007) showed that the effect of sleep deprivation on mood in normal subjects is related to their circadian preferences and they therefore suggest that adjusting work schedule with morningness of eveningness preferences would improve mood alterations.

Problems with sleep need to be investigated already in childhood. The sleep literature has shed light upon how important sleep is for children and adolescents in several areas such as the learning environment. A quantitative meta-analysis (Dewald, Meijer, Oort, Kerkhof, & Bögels, 2010) examined 26 studies assessing the relationship between three different sleep domains and children and adolescents’ school performance. The meta-analysis found that all three sleep variables; sleep quality, sleep duration, and sleepiness, were significantly however modestly related to school performance. The meta-analysis yielded small overall effect-sizes in all three domains; sleepiness showed the strongest relation to school performance, followed by sleep quality and sleep duration. Dewald et al. (2010) emphasizes the importance of educating schools, parents, adolescents, and children about the effects of insufficient sleep on school performance. A study by Owens, Belon, and Moss (2010) showed that delaying school start time for students in 9-12 grade (14-18 years) was associated with significant improvements in alertness, mood, and health. Owens (2014) points out the severity of chronic sleep loss in adolescence and the threat that it poses for academic success, health, the safety of her nation's youth as well as the importance of this public health issue. Hopefully, more countries and nations will follow this new advice and thus prevent our future generation from the detrimental effects of sleep deprivation. Identifying emotion-related processes as explanatory mechanisms linking sleep and psychological risk is important for the future of
predicting mental wellbeing and the evolution of affective disorders. New research on sleep deprivation (Krause et al., 2017) stresses the importance of health policies to start giving sleep-recommendations in line with the new evidence to aid the current sleep-loss epidemic in industrialized nations.

**Sleep Loss**

Sleep loss, sleep restriction or sleep deprivation is the condition of not getting enough sleep. Studies examining the effects of sleep deprivation are often based on either partial (1-6h), acute (24-48h) or chronic (24h+) sleep deprivation. The sleep-deprived brain has been investigated for decades and in relation to various topics such as e.g. attention, memory, fear conditioning, emotions, psychopathology and performance (e.g. Lowe, Safati, & Hall, 2017; Pilcher & Huffcutt, 1996). Not getting enough sleep has been linked to several outcomes of emotion dysregulation, such as increases in the experience of negative emotions, lower occurrence of positive emotions and changes in how these emotions are understood, expressed and modified. Several studies (Gross & Jazieri, 2014; Harvey, 2011) demonstrate this pattern and suggests that inadequate or disrupted sleep may serve as a risk factor for psychiatric disorders such as anxiety and mood disorders. Unfortunately, in our modern-day society sleep does not seem to be something that we prioritize which probably has contributed to the ongoing epidemic of sleep loss (Liu et al., 2014) which will be further illustrated in the upcoming section. The ability to study different conditions of sleep deprivation has an important role for further investigating potential mechanisms underlying emotional brain processes. Hobson and Pace-Schott (2002) found that slow wave activity (SWA) induced by sleep deprivation were prominent in frontal brain areas and have therefore suggested that SWA might indicate an especially high need for recovery sleep in the region of the brain that is the seat of executive function and working memory. Sleep deprivation studies will be reviewed in an upcoming section.
Emotion

Although people have been asking the question of what an emotion is for several thousand years, it is still surprisingly unclear (Gazzaniga et al., 2013). There is no universal definition of emotion. However, researchers do agree upon three components that emotion consist of – a physiological reaction to a stimulus, a behavioral response and a feeling. Ochsner & Gross (2005, p. 242) suggests “that emotions are valenced responses to external stimuli and/or internal mental representations that;

- involve changes across multiple response systems
- are distinct from moods
- can be either unlearned responses to stimuli with intrinsic affective properties or learned responses to stimuli with acquired emotional value
- can involve multiple types of appraisal processes that assess the significance of stimuli to current goals, that
- depend upon different neural systems “

To reiterate, every emotion consists of a physiological reaction, a behavioral response, and a feeling. Emotion generation is the term for the specific timing of that physiological reaction, behavioral reaction and experiential feeling (i.e. how emotions arise and unfold) (Gazzaniga et al., 2013).

Many parts of the nervous system are involved in emotions - the sensory systems, memory systems, and the autonomic nervous system. Emotions arise from the appraised significance of stimuli that is in line with our goals and needs, by these systems. Such appraisal involves attention processes, evaluation processes, and response processes. These processes influence the type of emotions one has, when one has them, and how one express and experience them. The influence of such processes is called emotion regulation. These
processes can be altered by strategies aimed at regulating emotions and intervene at multiple points in the emotion generation. Naturally, there are several theories on emotion generation. This thesis will solemnly focus on the process model. The process model is the most widely cited framework for organizing emotion regulation strategies (Paterson, 2012).

In 1998, Gross described his view of emotion regulation as the process model (Gross, 2014). By outlining several steps of the emotion regulation processes, the process model consists of the five following steps; situation selection, situation modification, attentional deployment, cognitive change and response modulation.

Gross (2014) proposed that emotion regulation is distinct from emotion generation, whereas the latter arises from encountering, attending to, and appraising an emotion-eliciting stimulus and lastly generating an emotional response. According to this view, emotion regulation instead involves actions influencing what emotions we have and how and when we have them. According to Gross (2014), emotion regulation can depend on the context, take many different forms such as regulating one's own emotions (intrinsic) or regulating someone else's emotions (extrinsic). Awareness, goals, and strategies are important and common factors for adaptive regulation. Emotional awareness affects the range of available strategies as well as the flexibility in how we use them, in other words, it might help us act differently given the situation. The emotion regulation goal increases or decreases the magnitude or intensity, in line with what we aim to achieve, of the emotional experience, expression or physiology. The strategies then have the most obvious cause by specifying the means of emotion regulation, as they are executed in accordance with achieving such goals. The five steps mentioned above are divided based on their primary effect in the emotion-generative process. Situation selection determines if we encounter a specific situation thus generating an emotion that is either desired or not. Situation modification does precisely what it sounds like, attempt to alter external features of the environment to influence the emotions.
Attentional deployment is the direct or indirect way of altering the emotional response followed by a cognitive change which aims to revise the meaning of the situation that also influence the emotions. Lastly, response modulation is the efforts influencing experiential, behavioral, or physiological responses that one has to the emotion-eliciting situation (Gross, 2014). Depending on what we do or feel from day to day minute by minute we move from left to right in this cycle and engage in various combinations of behaviors and strategies rather than just one. It is important to note that these strategies are not always optimal and can be maladaptive as well as adaptive (Gross & Jazaieri, 2014).

**Concepts of Emotion**

Gross (2015) makes the distinction between affect, moods, emotions and emotion regulation by sub diving them into categories rather than defining them on their own. According to Gross’s view, affect is an umbrella term for physiological states that involve valuation - stress responses, emotions, and moods. These affective states are alike as they are quite short-lived ones and they come and go as they please. Stress responses typically occur in response to an inability of managing situational demands while emotions are assigned to specific negative or positive affective states. Moods are more diffuse and tend to be longer lasting than both stress responses and emotions.

Again, emotions are hard to define, and conceptualizations in the field differ in approaches such as basic emotion approaches, appraisal approaches, psychological construction approaches and social contract approaches. However, these approaches seem to agree upon certain aspects of emotions; “Emotions involve loosely coupled changes in the domains of subjective experience, behavior, and peripheral physiology”, “emotions unfold over time” and “emotions can be either helpful or harmful, depending on the context” (Gross, 2015 p. 3-4). These are the core aspects of emotions.
Continuing with Gross’s (2015) view, affect regulation is another umbrella term. Gross subdivides affect regulation into coping, emotion regulation and mood regulation. Coping overlaps with emotion regulation, although they differ in a time span. Coping has a longer temporality than emotion regulation and predominated emphasis on alleviating stress responses whereas emotion regulation rather attempts to influence our emotions – which emotions we have, and when and how we experience or express them. Lastly, mood regulation aims at altering subjective feeling states (Gross, 2015). Emotions give rise to impulses to act in different ways that change the experience or behavior, and these changes are associated with autonomic and neuroendocrine responses that both anticipate emotion-related behaviors and follow them (Gross, 2014).

Emotional reactions to stimuli are often characterized by the factors valance and arousal. Valance can be either pleasant or unpleasant, or, good or bad whereas arousal is either high or low thus defining the intensity of the internal emotional response (Gazzaniga et al., 2013). It is common among researchers to use these dimensions in search of the neural correlates of different emotional reactions elicited by stimuli. The network of brain structures involved in emotional behavior is called the emotional brain.

The Emotional Brain

The scientific study of the neural bases of emotions has addressed the question of which brain systems underlie emotions for the past 40 years (for a historical overview, see; Dalgleish, 2004). The emotional brain is a term used for describing the network of brain structures underlying emotional behavior, often associated with the limbic system (Gazzaniga et al., 2013). The limbic system consists of the cingulate gyrus, corpus callosum, thalamus, hippocampus, amygdala, hypothalamus and the orbitofrontal cortex. Progress in measuring brain responses to emotional stimulus has helped researchers in finding additional emotional networks that include the somatosensory cortex, the higher order sensory cortices, the insula.
and the mPFC including the orbitofrontal cortex (OFC), ventral striatum and ACC (Gazzaniga et al., 2013). Emotion is not associated with a specific neural circuit; different neural systems are rather involved in different emotional behaviors.

The amygdala is a small almond-shaped bilateral structure in the medial temporal lobe and has been found to be important for emotion processing in general because of its many connections to several other brain regions. The amygdala is called “the veritable godfather” of the forebrain as it is its most connected structure (Gazzaniga et al., 2013). Due to the extensive connections to and from the amygdala, it has been possible to investigate and infer its important roles learning, memory and attention in response to emotionally significant stimuli. The amygdala contains receptors for various neurotransmitters such as norepinephrine, dopamine, serotonin, and acetylcholine, among others. The amygdala has been suggested to be involved in attention, perception, value representation and decision making due to its involvement in determining what a stimulus is and what is to be done about it. The role of the amygdala in emotion is however still controversial (Gazzaniga et al., 2013).

Functional neuroimaging in the context of emotion regulation studies have shown increased activity in the PFC and decreased activity in the amygdala when emotion is down-regulated (Hajcak, Dunning, Foti, & Weinberg, 2014). Neuroimaging studies examining the effects of different stimulus contexts in a form of emotion regulation strategy called reappraisal, a cognitively oriented form of emotion regulation in which a person tries to think about a situation in a way that alters the emotional response (Gross, 2014), have shown to engage dorsomedial, dorsolateral, and ventrolateral prefrontal cortex, as well as temporal and parietal cortex (Gross, 2015). This network has also been suggested to either upregulate or downregulate the activity of emotion-generative systems including the amygdala and ventral striatum depending on the context. A meta-analysis by Buhle et al. (2014) describes that existing reappraisal models broadly agree upon that reappraisal recruits frontal and parietal
control regions to modulate emotional responding in the amygdala via either control regions
that engage the ventromedial prefrontal cortex (vmPFC) that in turn modulates amygdala
responses or that control regions modulate semantic representations in lateral temporal cortex
that indirectly influence emotion-related responses in the amygdala.

Studies on emotional processing have found significant activity in the amygdala, OFC
and the insula which suggests an involvement of these brain areas in different forms of
emotional processing (Gross, 2014), this involvement will be illustrated in an upcoming
section.

**The Role of Sleep in Emotional Brain Functioning**

Everyone has probably felt the backlash of a lack of sleep and how it might impact
our mood, appetite and executive function. Have you ever had an irrational emotional
response as a consequence of sleeping too little or too much? Luckily, there are logical
explanations for such phenomena. Sleep abnormalities are characteristic of most psychiatric
disorders and disrupted sleep has been suggested to be a robust risk factor for psychiatric
conditions (Palmer & Alfano, 2017). The section below is aimed at presenting scientific
findings on the relationship between sleep and emotional brain functioning.

Insufficient sleep has been associated with subjective reports of irritability, emotional
volatility, amplification of negative emotions and increases of stress, anxiety, and anger
(Goldstein & Walker, 2014). Emotional dysregulation often follows from sleep deprivation, a
pattern that has been observed in studies assessing both objective physiological and neural
measurements of affect (Gujar, Yoo, Hu, & Walker, 2011; Motomura et al., 2013; Yoo et al.,
2007). Sleep abnormalities have also been suggested to influence our mood and emotion
regulation (Baum et al., 2014).
Observational Studies

Observational studies from infancy to adulthood have consistently suggested that sleep affects emotions (Hall et al., 2012). A study on napping behaviors in children (ages 3-5) by Ward, Gay, Anders, Alkon, and Lee (2007) found that children with disruptive behavior and problems with settling down at naptime, called problem nappers, showed greater levels of negative affect as well as decreased effortful control. Similarly, a prospective study (Touchette, Petit, Tremblay, & Montplaisir, 2009) on children of ages 5 months to 6 years reported an elevated risk of hyperactivity and impulsivity in children who slept poorly and less than 9 hours a night until the ages of 3.5 years. A new study by Palmer, Oosterhoff, Bower, Kaplow, and Alfano (2018) utilized data from a nationally representative sample from the United States - the National Comorbidity Survey-Adolescent Supplement (N=10,148; age range 13-18 years). The adolescents that reported sleep problems were also more likely to fulfill the criteria of a mood or anxiety disorder (Palmer et al., 2018). There were also tendencies of poorer emotion regulation strategy use in these participants. These findings suggest that poor emotion regulation strategies may be a factor that contributes to sleep-based psychiatric risk. Sleep disturbances are evident in several psychiatric conditions such as mood- and anxiety disorders, major depression and post-traumatic stress disorder (PTSD) (Goldstein & Walker, 2014). Investigations of such disorders have helped scientists to begin to understand and give plausible explanations for the prevalent relationship between sleep abnormalities and such disorders (Harvey, 2011). Sleep disturbances are a risk factor for adolescents and adults with mood disorders, which can give rise to new episodes, cause relapse and also have an adverse impact on emotion regulation which is critical for cognitive functioning. In line with such assumptions, levels of trait anxiety have been found to predict inter-individual differences in anticipated exaggerated response after sleep deprivation and especially high trait anxiety has been correlated with severe increases of anticipatory
reactivity (Goldstein et al., 2013). Anticipation is an adaptive process that prepares us for possible threatening events. Excessive anticipatory responding and hyper-reactivity in the amygdala have consistently been found in anxiety disorders (Goldstein et al., 2013). Hence, it is suggested that insufficient sleep might amplify the impact of a trait vulnerability factor. In line with this, Cellini, Duggan, and Sarlo (2017) have suggested that level of neuroticism is a predictor of sleep quality. By contrast, a large study of 929 subjects (Ackermann, Hartmann, Papassotiropoulos, de Quervain, & Rasch, 2015) on how interindividual differences in sleep are related to memory performance assessed with neutral and emotional pictures, showed no association between individual differences in sleep parameters and memory consolidation for emotional and neutral stimuli. These contradictory results indicate that sample size might be a critical aspect that require close examination.

**Experimental Studies**

Insufficient sleep has consistently been linked to impairments of emotional processing such as amplified negative emotions and lower thresholds for stress (Goldstein & Walker, 2014). One night of experimentally controlled sleep loss has been found to lower the threshold for experiencing an event as stressful (Minkel et al., 2012), as well as enhance impulsivity towards negative stimuli (Anderson & Platten, 2011). Similarly, a study by Gujar et al. (2011) investigating responses to emotional picture stimuli after one night of sleep deprivation showed increased reactivity in regions of dopaminergic mesolimbic systems in response to pleasure-evoking emotional picture stimuli. This enhanced sensitivity has been associated with decreases in functional connectivity in regions of the medial and orbital prefrontal cortex. These findings suggest that insufficient sleep might cause a state of emotional imbalance that arises from an exaggeration of subcortical limbic and striatal responses to affective stimuli and impoverished PFC activity and/or connectivity (Goldstein & Walker, 2014). Studies focusing on the effects of sleep deprivation on emotional
processing have shown amygdala hyper-reactivity and changes in functional connectivity (Motomura et al., 2013; Yoo et al., 2007). By contrast, studies on the role of sleep on emotional reactivity has also shown minor/no changes (Baran, Pace-Schott, Ericson, & Spencer, 2012; Groch, Wilhelm, Diekelmann, & Born, 2013). Baran et al. found that 12 h of daytime wakefulness were associated with attenuation of negative ratings of emotional pictures, whereas a period of 12 h including sleep was associated with relative maintenance of the initial negative ratings. Hence, a picture that was first considered as highly negative was then rated substantially less negative after 12 h awake, however only slightly less negative following sleep. In line with this, Groch et al. (2013) found that REM sleep contributed to the consolidation of emotional contents in memory. However, they also found that valence and arousal ratings of emotional pictures were not affected by REM-rich or SWS-rich sleep which, according to Groch et al. (2013), suggests that sleep does not modify emotional reactivity that is associated with such images. Hence, that the affective tone is preserved rather than reduced by the processing of emotional memories during REM sleep.

In 2018 a new and very interesting study by Zhang, Lau, and Hsiao was published, aimed at investigating how sleep deprivation impacts the brain network of emotional functioning. It is the first study to examine sleep deprivation impacts on emotional functioning using EEG in a resting state. Zhang et al. (2018) suggest that emotional regulatory processes might be compromised during the resting state after sleep deprivation. Left lateralization of alpha power has been associated with poorer emotional processing and depressive and anxiety disorders, and theta/beta ratio has been associated with weak inhibition control to emotional faces. They found marginally heightened left frontal alpha asymmetry and heightened theta/beta ratio which implies that sleep loss not only has a negative impact in the functioning of this network upon stimuli but also its default functional state without external stimuli (i.e. during a resting state) (Zhang et al., 2018).
Another new study by Killgore et al. (2017) aimed at investigating the effect of sleep deprivation on emotional recognition showed that sleep deprivation significantly reduced the accuracy of identifying facial expressions of happiness and sadness, interestingly these reductions were abolished by recovery sleep. In line with this, a study by Crönlein, Langguth, Eichhammer, and Busch (2016) showed similar results of impairments in patients with sleep disorders. Insomnia and sleep apnea reduced the recognition of both happy and sad faces but not for anger, anxiety, fear or disgust. These results suggest that sleep deprivation mainly affects subtle facial cues of happiness and sadness while the identification of other more primitive survival-oriented faces seems relatively unchanged by sleep deprivation (Killgore et al., 2017). By contrast, a large study of about 500 subjects (Holding et al., 2017) investigating the role of sleep in emotion recognition found no effects of subjective sleep quality or sleep deprivation on facial emotion identification abilities. In comparison with previous studies they found no distinctive impairment following sleep loss in the specific emotions such as sadness, anger and happiness, disgust, or fear. These contradictory results also indicate the importance of sample size.

**Neurobiological Underpinnings of Sleep and the Emotional Brain**

The relationship between sleep and emotion regulation seem to be supported by their neurobiological underpinnings given that they both are believed to involve the same brain regions (Palmer & Alfano, 2017). These regions are the subcortical brain structures of the limbic system such as the amygdala and prefrontal cortex. Neuroimaging studies in humans have shown that the sleep-deprived brain give rise to several maladaptive changes in behaviors (Krause et al., 2017).

Sleep deprivation has been suggested to be linked with a failure of normal regulation of the amygdala, which causes increased activation of the amygdala and a loss of functional connectivity with the mPFC (Vandekerckhove & Cluydts, 2010). The decrease in prefrontal
activation along with increased attention and reactivity towards negative stimuli have been suggested to result in emotion regulatory functions becoming dysfunctional (Vandekerckhove & Cluydts, 2010).

A fMRI study by Yoo et al. (2007) investigated the impact of sleep deprivation on emotional brain reactivity and functional connectivity. The participants in the study were divided into a control group (rested) and a sleep deprivation group (34 hours), they were then tested in an emotional stimulus viewing task in an event-related fMRI design during scanning. Both groups showed significant amygdala activation in response to increasingly negative picture stimuli, however, a 60% amplification in reactivity was found in the sleep deprivation group as compared with the control group. The results also showed an increase in amygdala connectivity in the deprivation group with autonomic-activating centers of the brainstem, including locus coeruleus and midbrain, which suggests a hyper-limbic response by the amygdala towards negative emotional stimuli when sleep deprived. Such an increase in limbic activity was also suggested to be associated with loss of functional connectivity with the MPFC in the sleep-deprivation group which suggests a failure of top-down, prefrontal control. Yoo et al. (2007 p. R878) therefore suggest that "a night of sleep may 'reset' the correct brain reactivity to next-day emotional challenges by maintaining the functional integrity of this MPFC-amygdala circuit, and thus govern appropriate behavioral repertoires". Another fMRI study (Motomura et al., 2013) investigated 14 healthy adult men in a sleep restriction condition of 5 days (4 h for time in bed) and a control group (8 h for time in bed) in an emotional face viewing task. In response to the facial expression of fear, participants showed increased activity of left amygdala as unchanged by a happy expression. A significant decrease in functional connectivity between the amygdala and ventral anterior cingulate cortex (vACC) was also found in the deprivation group in proportion to the degree of sleep debt, along with significant correlation with amygdala and deterioration of subjective
mood states (Motomura et al., 2013). This study suggests global problems with monitoring and regulatory control. Sleep deprivation has also been suggested to alter the brain anticipation of cued emotional experiences (Krause et al., 2017). Similarly, this pattern has been found under conditions of one night of sleep deprivation, which caused elevated cue-evoked activity in the amygdala, anterior insula and ACC in anticipation of impending emotional picture slides (Goldstein et al., 2013).

Not only have sleep and emotion been suggested to involve the same brain regions, they have also been suggested to share the same neurochemicals (Goldstein & Walker, 2014). The neurochemicals involved in REM-sleep also govern emotion regulation, such as noradrenaline. During REM sleep noradrenaline reduces to the lowest concentration levels compared to NREM or waking (Ouyang, Hellman, Abel, & Thomas, 2004) during a period of 24 hours (Goldstein & Walker, 2014). Noradrenaline is associated with several arousal-related emotional processes within the brain and the body which makes this relationship appropriate (Goldstein & Walker, 2014). Furthermore, dysfunctional ranges of noradrenaline have been associated with PTSD and major depression.

Alterations and abnormalities of REM-sleep has been related to variables that are associated with the affective state of individuals during the day (Vandekerckhove & Cluydts, 2010). Different studies of sleep deprivation on individuals suffering from depression, alcohol abuse, and suicidal attempts have all shown similar patterns of increases in REM sleep activity, especially REM sleep duration. REM sleep have particularly been suggested as the affective part of sleep that impacts next day mood and emotion because of observing "a hyper limbic and hypoactive dorsolateral prefrontal functioning in combination with a normal functioning of the mPFC, probably adaptive in coping with the continuous stream of emotional events we experience" (Vandekerckhove & Cluydts, 2010, p. 219).
As the research area of sleep and emotion have grown, an interesting framework has been proposed on the relationship between the two.

**A Proposed Function of REM Sleep Homeostasis**

The outlined neurobiological features of sleep and emotional brain functioning in previous sections have intrigued researchers to further investigate REM sleep and its possible functions. REM sleep has been hypothesized to have an emotional homeostatic function. The basis for such assumptions lies mostly in neuroimaging findings showing alterations of functional brain activity and brain neurochemistry during REM sleep.

Goldstein and Walker (2014) have proposed a framework in which REM-sleep supports a process of an affective brain homeostasis that optimally primes the organism for next-day social and emotional functioning. This framework consists of two so-called brain benefits of REM sleep – emotional memory resolution and emotion recalibration.

The proposed function of emotional memory resolution is based upon what happens after emotional events and the aiding of reprocessing prior affective experiences. Such experiences seem to be easier to recall due to emotional tone and specifically as adrenergic and peripheral autonomic reactions are elicited as the experience occurs (McGaugh, 2004). These experiences have been found to cause activation of hormonal- and brain systems that regulate consolidation of new memories. Critical interactions among many neuromodulatory systems occur in the basolateral region of the amygdala and these effects have been suggested to be integrated through common actions on noradrenergic and cholinergic activation within the basolateral amygdala. Such activation is proposed to regulate memory consolidation as the amygdala projects to many other brain regions involved in processing memories. Hence, emotionally exciting experiences are believed to be well remembered through activation in these systems (McGaugh, 2004). Goldstein and Walker (2014) propose that REM sleep helps us forget this emotional tone of a memory although consolidating the memory of the
experience due to its neurophysiological, neuroanatomical and neurochemical properties. Roughly explained it is these changes during REM sleep that support their framework; the increased activity within limbic and paralimbic structures (e.g., Braun et al., 1997) that supports an ability of reactivation and reprocessing previous affective memories. Second, the neurophysiological signature of REM sleep suggesting emotional memory representation across anatomical networks (Goldstein & Walker, 2014) and lastly, that these interactions take place in a brain low in aminergic neurochemical concentration (Hobson & Pace-Schott, 2002) and specifically so, the suppression of noradrenergic input from the locus coeruleus (e.g., Itoi & Sugimoto, 2010; Ramos & Arnsten, 2007). Goldstein and Walker (2014) argue that this biological condition is aimed at strengthening and consolidating the core of emotional experiences as well as depotentiating the autonomic arousing charge of the experience.

The second benefit is said to recalibrate and restore next-day emotional brain sensitivity through REM sleep – an overnight resetting function within key brainstem, limbic and prefrontal brain regions. Evidence for this hypothesized benefit derives from three key points of neurochemical features during waking and sleeping. Goldstein and Walker (2014) propose that REM sleep might serve as a noradrenergic "housekeeper" by reducing and restoring concentration of noradrenaline to baseline each day (Mallick & Singh, 2011). During REM sleep, compared to wake or NREM, there is a lack of locus coeruleus firing which manifests in an absence of noradrenaline release (e.g., Ouyang et al., 2004). While sleep deprived the levels of noradrenaline exceed those of resting brain function (Mallick & Singh, 2011), thus suggesting a relationship between quantity or quality of REM sleep and a decrease in noradrenaline (Goldstein & Walker, 2014). Experimental manipulations similar to sleep loss impair the salience sensitivity and specificity of locus coeruleus responding (Mallick & Singh, 2011). While sleep deprived, the brain might become hypervigilant, thus
less able to discriminate salient from non-salient stimuli. Noradrenaline neurons within the locus coeruleus display two modes of overall activity during wakefulness, these modes respond in a different manner depending on the condition. When locus coeruleus responds in a predominant phasic manner (i.e. selectively to salient stimuli within the environment) while maintaining a low level of baseline (tonic) ongoing activity (i.e. background "noise") (Mallick & Singh, 2011) the overall threshold of reactivity to external events is optimal along a gradient of potential emotional strengths (Goldstein & Walker, 2014). Under conditions such as e.g. stress – the activity in locus coeruleus shifts to high levels of persistent baseline tonic firing and elevated levels of noradrenaline which contributes to the phasic signals that respond to external emotional stimuli resulting in a poor signal to noise ratio within the system and therefore reduced specificity (Mallick & Singh, 2011). This increase in noradrenergic concentration is what Goldstein and Walker (2014) propose to be the cause of the hypervigilant brain state while sleep deprived, thus making the person far less able to discriminate salient from non-salient stimuli. When the noradrenergic activity is low and with low tonic/high phasic signaling the locus coeruleus facilitates selective amygdala responsiveness to salient stimuli in a phasic manner which is further enhanced on mPFC functioning (e.g., Ramos & Arnsten, 2007). This increase in mPFC engagement enables top-down control of the amygdala and prevents it from responding to non-salient stimuli. In line with this Goldstein and Walker (2014) suggest that REM sleep optimally restores the emotional salience sensitivity and specificity of this adrenergic locus coeruleus-mPFC-amygdala functional network. Decreased amygdala-PFC connectivity following sleep deprivation has been demonstrated by Yoo et al. (2007) and it seems as a night of sleep might serve as a restorative function of amygdala-mPFC connectivity (van Der Helm et al., 2011). By contrast, a study by Cunningham et al. (2014) suggested that sleep rather decreased emotional reactivity. Cunningham et al. found a general de-potentiation effect of sleep as
their study showed that reactivity to both negative and neutral objects of scenes decreased following sleep. They also found that the increased arousal responses to negative scenes at encoding were positively correlated with subsequent memory for the negative objects of scenes, but only for the subjects that had slept. Cunningham et al. (2014) therefore describe that although arousal responses are often thought to account for emotional enhancement in long-term memory, their findings suggest that both an arousal response at encoding, and a subsequent period of sleep, are needed to optimize selective emotional memory consolidation.

Discussion

The aim of this thesis was to review the relationship between sleep and emotional brain functioning as well as a proposed function of REM sleep, and possibly implicate new scientific findings that either further illustrate the relationship or lack thereof. To answer these questions and provide a basic understanding of the concepts, basic overviews of sleep and emotion, and data suggesting their neural basis was provided. Lastly, current scientific evidence for the relationship between sleep and emotional brain functioning, and a proposed function of REM sleep in emotional processing was provided.

The functions of sleep are still controversial (Tempesta, Socci, De Gennaro, & Ferrara, 2017). The most well-known cognitive function of sleep to this day is its important role for memory and learning (Gazzaniga et al., 2013). Specifically, the role of sleep in the process of memory consolidation and integration seems to be largely acknowledged, whereas the specific role of sleep in emotional processing still lacks consensus (Tempesta et al., 2017). Neuroimaging studies investigating possible effects of sleep deprivation have found increases and decreases of activity in emotion-related brain areas (e.g. Dang-Vu et al., 2010; Gujar et al., 2011; Schwartz & Maquet, 2002; Yoo et al., 2007) thus suggesting that sleep might influence emotional brain functioning. Studies have also shown that the suggested
neurochemicals involved in sleep such as noradrenaline (Hobson & Pace-Schott, 2002; Ouyang et al., 2004; Mallick & Singh, 2011) are also involved in arousal-related emotion processes and has therefore been suggested to have a causal relationship (Goldstein & Walker, 2014). Nevertheless, correlation does not imply causation.

The scientific study of emotion still lacks a universal definition of emotion. However, the network of brain structures believed to underly emotional behavior seem to be largely acknowledged and are commonly termed as the emotional brain (Gazzaniga et al., 2013). These emotional brain networks have been suggested to involve the cingulate gyrus, the corpus callosum, the thalamus, the hippocampus, the amygdala, the hypothalamus and the OFC (i.e. the limbic system) as well as the somatosensory cortex, the higher order sensory cortices, the insula and the mPFC including the OFC, ventral striatum and ACC. Neuroimaging studies have consistently found increased activity in such areas during emotional processing (Gazzaniga et al., 2013).

The sleep – emotion area is still in its infancy, which becomes highly noticeable as the most common terms such as emotion, affect and emotion regulation are used differently by researchers in the field. Surprisingly, it is more rare than usual that the authors define the usage of concepts regarding emotion although these challenges seem to be well known. Specifically, the study of emotion regulation in relation to sleep does have contextual and empirical challenges to overcome such as overlapping constructs of emotion and lack of distinction regarding these concepts. These challenges become very apparent when attempting to provide a somewhat clear and unambiguous review of the current scientific evidence for the proposed intimate and causal relationship between sleep and emotional brain functioning. What this clearly points out is how important it is for the field to overcome these contextual challenges thus enabling researchers to draw unambiguous conclusions. Plenty of research in the sleep – emotion area has mainly focused on the influence of sleep in emotions
and mood rather than emotional processing. However, there seems to have been a shift of focus quite recently and since the beginning of the 21st century, many studies and papers published on the subject have made efforts to further investigate the specific influence of sleep in emotional processing.

There is a wide variety of methods used for studying the relationship between sleep and emotional brain functioning, and it is not uncommon to use different experimental paradigms that range from conditions of partial sleep restriction to total sleep deprivation, as well as various designs for examining the influence of specific sleep stages in emotional processing. These designs differ in e.g. tasks of viewing pictures or movies with aversive or neutral stimuli, as well as tasks of facial recognition or facial expression. They also differ in time span regarding the duration of sleep deprivation, as the conditions are measured for either one or several nights. Lastly, regarding ecological validity, studies vary in environments of either sleep labs or at the home of the subjects. These methodological differences can lead to large discrepancies regarding sleep duration, the timing of sleep and timing of testing sessions, which can result in difficulties comparing results.

Insufficient sleep has consistently been associated with increases in irritability, emotional volatility, negative emotions, stress, anxiety, and anger (Goldstein & Walker, 2014). Furthermore, sleep abnormalities are characteristic of most psychiatric disorders (Palmer & Alfano, 2017). It is therefore not surprising that several researchers have suggested an intimate and causal relationship between sleep and emotional brain functioning. At a first glance, there seems to be a growing body of evidence supporting this relationship (Goldstein & Walker, 2014). However, there are contradictive results which seem to unnoted by the most prominent researchers in the field. For instance, some studies on the role of sleep in emotional reactivity have both showed no minor/no changes (Baran et al., 2012; Groch et al., 2013) as well as decreased activity (Cunningham et al., 2014). However, evidence from
fMRI studies (Yoo et al., 2007) does show a strong convergence in connectivity in amygdala and vmPFC as well as the hippocampus (Tempesta et al., 2017). The inconsistency across studies of emotional reactivity (Baran et al., 2012; Cunningham et al., 2014; Groch et al., 2013) might be related to the characteristics of the experimentally elicited emotional experience, particularly due to emotional intensity. It is possible that the emotional stimuli used in studies measuring emotional reactivity are too weak to give rise to a sufficiently arousing emotional reaction or salient experience, thus leading to negative findings.

Another problematic aspect of how the research often is conducted is the lack of focus on other factors included in sleep deprivation, such as extended wakefulness. As Krause et al. (2017, p. 404) point out “it is therefore insufficient only to develop an understanding of the functional benefits of sleep and then to reverse- infer an understanding of the neural and behavioral changes that would be expected following a lack thereof”. An awareness of such factors would provide great insights and importance for the possibility of avoiding confounding variables.

REM sleep has been proposed to have a homeostatic and recalibrating function (Goldstein & Walker, 2014). However, the role of REM sleep in modulating next day emotional functioning is still controversial. Although there are several studies indicating that lack of sleep significantly influences emotional reactivity. It could be argued that distinct emotionally arousing events, characterized by different levels of intensity, are subjected to different temporal dynamics of processing. Therefore, whereas one night of sleep may be sufficient to adaptively process stimuli of low-intensity such as in experimental studies, stimuli of high intensity, and traumatic memories might require additional sleep to be processed successfully. One-night designs that dominate research on the relationship between sleep and emotional brain functioning might be insufficient to capture the critical modulating role of sleep in emotional processing over time. Similarly, restoring emotional brain
functioning to optimal baseline levels following sleep loss might require more than one recovery night (van Der Helm et al., 2011). Future research needs to investigate the influence of sleep and sleep deprivation on the processing of emotional stimuli across a broad range of emotional intensity as well as multiple nights in different populations, to possibly overcome these potential limitations.

According to Tempesta et al. (2017), sex differences can interact with a variety of cognitive and affective processes. Sex differences in functional asymmetry of limbic areas have been described in fMRI studies investigating emotional reactivity (e.g., Stevens & Hamann, 2012). Furthermore, females have been found to be specifically vulnerable to the detrimental effects of sleep loss (e.g., Killgore, Muckle, Grugle, Killgore, & Balkin, 2008) which highlights the importance of taking such factors into account. Tempesta et al. (2017) suggest that research on sleep and emotional regulation might be one of the research areas potentially most influenced by sex-related effects.

Levels of trait anxiety have been found to predict inter-individual differences in anticipated exaggerated response after sleep deprivation (Goldstein et al., 2013). In line with this, the trait factor neuroticism has also been suggested to be a predictor of sleep quality as high neuroticism scores have been associated with poor sleep quality (Cellini et al., 2017). These findings suggest that some individuals might be more vulnerable to the consequences of insufficient sleep. Tempesta et al. (2017) have suggested that trait-like features of sleep could have a substantial role in influencing some aspects of emotional processing such as emotional reactivity. Therefore, the field needs to closely explore such possible contribution of individual differences to the interaction between sleep and emotions. Such insights into how individual differences might influence the impact of sleep on emotional functioning could be of great relevance for future research.
Most importantly, research in this area commonly includes small samples sizes ranging from 20 to 50 participants which are problematic. A study of 929 subjects (Ackermann et al., 2015) showed no association between individual differences in sleep parameters and memory consolidation for emotional and neutral stimuli. In line with this, another study of about 500 subjects (Holding et al., 2017) found no effects of subjective sleep quality or sleep deprivation on facial emotion identification abilities. Tempesta et al. (2017) suggest that sample size could be a critical aspect of the sleep and emotions research in need of further examination. The risk of a positive publication bias could be limited if sample sizes were adequate and enable statistical power, which is of particular importance and perhaps underestimated in this research area.

Lastly, is it possible to save a nation of sleep-deprived humans? The study of the influence of sleep on emotional functioning is also of great importance for our public health. We have known for decades that sleeping is good for our health. Although, insufficient sleep has become a public health issue and there is now an ongoing epidemic of sleep loss in industrialized nations (Liu et al., 2014). Specifically, the sleep disorder insomnia is a major public health problem in a great desire of treatment as poor sleepers cost society as much as 10 times more than good sleepers (Daley et al., 2009). Identifying emotion-related processes as explanatory mechanisms linking sleep and psychological risk might have prognostic meaning in the future of predicting mental wellbeing and the evolution of affective disorders. New research on sleep deprivation (Krause et al., 2017) stresses the importance of health policies to start giving sleep-recommendations in line with the new evidence to aid the current sleep-loss epidemic in industrialized nations.

Conclusion

The idea of an intimate relationship between sleep and emotional brain functioning seems to be somewhat acknowledged, despite existing challenges in the field. By contrast, the
role of REM sleep in modulating next day emotional functioning seems to be controversial. However, research indicates that lack of sleep significantly influences emotional reactivity. The sleep–emotion area lacks consensus, and poor delineation and insufficient distinctions among emotional processes cause conceptual and empirical challenges in the field. Due to these challenges, it is difficult to comprehend and review the current scientific evidence for the proposed intimate and causal relationship between sleep and emotional brain functioning, and especially to draw any conclusions.

Future studies need to provide clear definitions of the constructs of emotion as well as clear distinctions between these concepts to overcome the existing conceptual and empirical challenges in the field. There is also a need to further investigate other factors that might have an influence on the relationship between sleep and emotional brain functioning such as sex differences and trait-like factors. Most importantly, future studies regarding sleep and emotional brain functioning need to have larger sample sizes, as adequate sample sizes and statistical power could limit the risk of a positive publication bias, which is of particular importance for this research area.
References


