Does sleep affect socio-emotional functioning?

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Abstract

In the first chapter I compare and contrast the extant literature on sleep loss and insomnia, including theories as to how insomnia develops and the role of the circadian and homeostatic systems. In Chapter 2 I summarize the extant literature on sleep, emotion perception, and social task performance, and review the relevant emotion literature. I then critically appraise this literature and suggest future directions for this field. In Chapter 3 I pilot an emotion recognition task among students, including measures of sleep and empathy. Results suggest that the previous nights' sleep, as well as depression scores, are significant predictors of happiness recognition. In Chapter 4 I assess emotion recognition in insomnia using dynamic stimuli, and results suggest that insomnia disorder impairs the categorization accuracy of high intensity expressions of sadness and low intensity expressions of surprise. Sleep diary parameters were also found to be significant predictors of happiness recognition on both accuracy and reaction time measures. I then assess how normal sleepers perform with these stimuli in Chapter 5, testing subjects at different times since waking. Chapter 5 Experiment One suggests that the early group are more sensitive towards several temporal parameters, with no effects on emotion recognition. Chapter 5 Experiment Two suggests that normal sleepers tested early are less sensitive towards mid-intensity expressions of anger and sadness, with effects on intensity recognition. These results are interpreted in the context of differences with the two late-tested groups. Chapter 6 extends these results to static stimuli, with results suggesting that the early group tend to make more errors when categorizing happy faces. Chapter 7 returns to the daytime impairments in insomnia disorder, suggesting that theory of mind task performance is altered when reaction times are measured. As a result of issues raised in this thesis Chapter 8 systematically reviews the literature on how normal sleepers are screened for participation in research studies, suggesting future criteria. Chapter 9 summarizes these results in the context of hyperarousal and the etiology of insomnia disorder.

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For my parents.

List of Abbreviations

AC – Alert Cognition
ACC – Anterior Cingulate Cortex
a.m. – ante meridiem
ANOVA – Analysis Of Variance
ANS – autonomic nervous system
AU – action units
B – unstandardized coefficients of the regression equation
BMI – Body Mass Index
c.f. – confer
CNS – Central Nervous System
Cm – centimetres
CRSD – Circadian Rhythm Sleep Disorders
CVT – Cardiac Vagal Tone
DISS – Daytime Impact of Sleep Scale
DISS-AC - Daytime Impact of Sleep Scale – Alert Cognition
DISS- PA - Daytime Impact of Sleep Scale – Positive Affect
DISS-NA - Daytime Impact of Sleep Scale – Negative Affect
DISS-S/F - Daytime Impact of Sleep Scale –Sleepiness Fatigue
DSM – IV - Diagnostic and Statistical Manula of Mental Disorders Four
DSM-IV-TR - Diagnostic and Statistical Manula of Mental Disorders Four – to be revised
DV – dependent variable
ECG – electrocardiogram
ECI – emotional context insensitivity
e.g. - exempli gratia
EEG - Electroncephalography
EMG – electromyography
Et al - et alii
EQ-I – emotional quotient inventory
ERP – event-related potential
F - Female
FACS – Facial Action Coding System
FB – False Belief
FIRST – Ford Insomnia Response to Stress Test
fMRI – functional magnetic resonance imaging
FNE – Fear of Negative Evaluation
GVAS – Global Vigor and Affect Schedule
HADS – Hospital Anxiety and Depression Scale
HADS-A - Hospital Anxiety and Depression Scale – Anxiety subscale
HADS-D - Hospital Anxiety and Depression Scale – Depression subscale
HPA axis – hypothalamic–pituitary–adrenal axis
HER – high emotional reactivity
ICD-10 – International Classification of Diseases
ICSD – International Classification of Sleep Disorders
IAPS - International Affective Picture Series
i.e. – id est
INS – Insomnia disorder
IRI – Interpersonal Reactivity Index
IRI – EC - Interpersonal Reactivity Index – Empathic Concern
IRI-FS - Interpersonal Reactivity Index – Fantasy Scale
IRI – PD - Interpersonal Reactivity Index – Personal Distress
IRI-PT - Interpersonal Reactivity Index – Perspective Taking
ISI – Insomnia Severity Index
IV – independent variable
KSS – Karolinska Sleepiness Scale
LER – low emotional reactivity

M - Male
MEQ – Morningness-Eveningness Questionnaire
MFFT – Matching Familiar Figures Test
Ms – milliseconds
MSCEIT – Mayer-Salovey-Caruso emotional intelligence test
MSLT – multiple sleep latency test
N - Number
NA – Negative Affect
NPR – nociceptive pain reflex
NS – Normal sleepers
OFC – Orbitofrontal Cortex
PA – Positive Affect
PET – positron emission tomography
PI - psychophysiological insomnia
PLMS – Periodic Limb Movements
p.m. – post meridiem
PNS –peripheral nervous system
PSQI – Pittsburgh Sleep Quality Index
PSG – polysomnography
QUAL – Quality
RDC – Research Diagnostic Criteria
REM sleep – rapid eye movement sleep
REM-D – rapid eye movement sleep
REST – Rested/restored
RLS – Restless Legs Syndrome
RT – Reaction Time
SCR – skin conductance response
SD – Standard deviation
SE – Sleep Efficiency
SE – Standard error
SF – Sleepiness/Fatigue
SOL – Sleep Onset Latency
STQ – Sleep Timing Questionnaire
TAS-20 – Toronot Alexithymia Scale 20 items
TB – True Belief
TiB – Time in Bed
TST – Total Sleep Time
TSW – Time Since Waking
UGSC – University of Glasgow Sleep Centre
WAKE – number of night-time awakenings
WASO – Wake-time After Sleep Onset

Chapter One

Sleep Loss and Insomnia

Abstract

Sleep loss and insomnia are often assumed to be similar. In the current chapter we review the literature on sleep loss and insomnia, and their effects on daytime functioning. Both sleep loss and insomnia are associated with effects on daytime functioning, although objectively impairments in task performance have been harder to identify in insomnia. This is surprising in light of the subjective complaints of people with insomnia, and the importance of distress in its diagnosis. We then summarize the literature on how insomnia develops, identifying ways in which sleep loss and insomnia are different. In particular, arousal and hyperarousal seem to play a key role in insomnia disorder, and vulnerability towards stress-induced sleep disruption seems to be important, alongside other relevant predisposing, precipitating, and perpetuating factors. We conclude with a brief review of the contributions of homeostatic and circadian contributions towards insomnia, in keeping with an understanding of insomnia whereby this disorder is placed in the context of normal sleep.

Sleep Loss and Insomnia

What is sleep loss?

Sleep deprivation refers to a period of continual wakefulness over at least 24 hours. Sleep restriction has been defined as "a reduction in sleep time below an individual's usual baseline, or the amount of sleep needed on a regular basis to maintain optimal performance" (Reynolds and Banks, 2010). While sleep deprivation studies last for a couple of days, sleep restriction can be chronic, and results in sleep debt. This sleep debt arises when an individuals' sleep need is not being met. Sleep loss will be used as a general term to describe both sleep restriction and sleep deprivation.

Sleep need has been defined as "the daily amount of sleep that allows a subject to be fully awake and able to sustain normal levels of performance during the daytime" (Ferrara and De Gennaro, 2001). With regards to sleep architecture, slow-wave sleep can be used as an indicator of sleep need (Greene and Frank, 2010). On average, 7.5 hours of sleep per night is common (Ferrara and De Gennaro, 2001). However, among students, a quarter report less than 6.5 hours of sleep on average (Lund et al., 2010). Some factors which affect sleep need have been identified as gender and age, as well as preferred sleep duration and sleep-ability (Ferrara and De Gennaro, 2001; see also Anderson and Horne 2008; Kronholm et al., 2006).

Behaviourally, a large weekend-weeknight time-in-bed difference could indicate chronic sleep debt (Monk et al., 2000). Furthermore, individual differences in sleep timing preference could modulate total sleep time. For example, individual differences in diurnal preference could contribute towards sleep loss, with evening-types more likely to be sleep deprived during the working week (Wittmann et al., 2006). To be specific, an evening type individual would choose to stay up late most nights, reflecting their sleep timing preference. However, the normal working week would mean that they would be unable to sleep longer the next day, and so their sleep opportunity would be curtailed. Morning types would show the opposite pattern, and might be expected to be sleep deprived at the weekend in order to meet social demands.

How does sleep loss affect daytime functioning?

In the general population, sleep loss could be a matter of public health (Ferrara and De Gennaro, 2001; Luyster et al., 2012; Meerlo, Sgoifo and Suchecki, 2008). Even in healthy subjects, sleep restriction, total sleep deprivation, and sleep disruption affect cognitive

functioning, sleepiness, and mood (Reynolds and Banks, 2010; Pilcher and Huffcutt, 1996; Dinges et al., 1997; Barnett and Cooper, 2008; Caldwell et al., 2004; Rose et al., 2008; Haack and Mullington, 2005; Drury, Ferguson and Thomas, 2012; see also Magee et al., 2009). Complex and socially-relevant decision making is also affected by sleep loss (Killgore et al., 2008; Killgore et al., 2007; McKenna et al., 2007). The rate of sleep loss could be a factor in how sleep loss is perceived and its impact on performance (Drake et al., 2001). Specifically, a gradual reduction in sleep time has been found to be less impairing (Drake et al., 2001). Moreover, some individuals are more affected by sleep loss than others, and subjective and objective measures of the effects of sleep loss form different factors (Van Dongen et al., 2004).

As sleep loss affects performance, there has been interest in identifying factors which contribute to this variability. Briefly, subjective and objective reactions to sleep loss may be predictable within individuals (Rupp, Wesensten, and Balkin, 2012; Van Dongen et al., 2004), although these measures could be somewhat dissociable (Leproult et al., 2003). Trait factors like extraversion-introversion, in interaction with the environment, also affect performance (e.g. Rupp, Killgore, and Balkin, 2010). Following total sleep deprivation, a socially-enriched environment affects the performance of extroverts to a greater extent than introverts (Rupp, Killgore, and Balkin, 2010). The circadian clock gene PER3 seems to contribute to some of this variance in sleep and performance, and response to sleep loss (Viola et al., 2007; Groeger et al., 2008; Viola et al., 2008). The subjective perception of good sleep quality might differ somewhat from the amount of sleep needed to maintain functioning (Ferrara and De Gennaro, 2001), and individuals may not always be able to judge their sleep need well (Van Dongen et al., 2003).

Individuals with insomnia disorder also report effects of sleep loss, although there are some differences between this and the sleep loss experienced by normal sleeper controls.

What is insomnia?

The research diagnostic criteria (Edinger et al., 2004) suggest that insomnia disorder comprises three components. Firstly, an individual must report problems initiating or maintaining sleep, early morning awakenings, or sleep that is consistently of poor quality or not restorative. Secondly, the sleep difficulty must occur despite adequate opportunity for sleep. Finally, the sleep difficulty must be associated with daytime impairment in functioning (i.e. fatigue/malaise; cognitive impairment; impairments at

work; mood disturbance; daytime sleepiness; proneness for accidents; tension headaches or gastrointestinal symptoms; or concerns about sleep).

These three components are comparable to those found in other diagnostic systems, such as the revision of DSM-5 (2014). In this, insomnia disorder is defined as a complaint about sleep quality or quantity, which is associated with impairments in daytime functioning, and occurs despite adequate opportunity for sleep. Additional specifications are given as to the frequency of sleep disruption, which must occur at least three nights a week for at least three months. The ICSD-R (2005) definition of insomnia includes similar criteria, with additional specifications as to insomnia severity, such as minor daytime functioning impairments in mild insomnia.

Across diagnostic systems therefore, there is consensus that insomnia disorder comprises the components of sleep disruption, sleep opportunity, and daytime effects. In addition to these criteria for insomnia disorder, some additional specifications are given for primary insomnia (Edinger et al., 2004). In particular, any current or past mental or psychiatric disorder, or sleep-disruptive medical conditions, must show temporal independence from the onset of the insomnia disorder. In addition, the insomnia is not caused by another sleep disorder, circadian rhythm disorder, or sleep/wake cycle, or by the use, abuse, or withdrawal of any substance or psychoactive medications. Furthermore, the insomnia disorder must have been present for one month (ICSD-R, 2005).

Suggestions have been made as to how to increase the clinical utility of an insomnia diagnosis (Edinger, 2004). For example, cluster analysis of insomnia symptoms suggest that diagnostic systems might not reflect how symptoms group together, in a data-driven approach to classification (Edinger et al., 2004). Furthermore, the different goals of researchers and clinicians can also affect how insomnia is defined. For example, clinicians are biased against missing a diagnosis, whereas false positives present a greater problem for researchers (Edinger, 2004).

How does insomnia affect daytime functioning?

Adverse sleep-related daytime effects are a feature of an insomnia diagnosis, which can affect functioning either socially or occupationally, and be cognitive or emotional in nature (Edinger et al., 2004). Such daytime effects are found with problems of sleep onset and sleep maintenance, and non-restorative sleep (Roth et al., 2010). Differences between good sleepers and poor sleepers on daytime mood have been reported, with

poor sleep quality and short total sleep time associated with worse daytime mood (Ong et al., 2011). In a study of patients and controls, a sleep complaint has been linked to two distinct factors, named insomnia and lassitude (Koffel and Watson, 2009; see also Neu et al., 2010). In general, fatigue, or lassitude, seems to be an adverse daytime effect of sleep disruption. In turn, anxiety and depression also affect sleep (Mayers et al., 2009; Ramsawh et al., 2009), and such results suggest that mood and sleep are interlinked.

In primary insomnia, specific complaints reported are difficulty with concentration, attention, and memory, in addition to pain, anxiety and fatigue, and these problems are present even after controlling for comorbidities (Kyle, Morgan, and Espie, 2010). However, comorbidities are common in insomnia, and insomnia associated with a mental disorder has been linked to more daytime effects (Sanchez-Ortuno, Edinger, and Wyatt, 2011). Brain imaging results support alterations in wake-time functioning among people with insomnia, who show abnormal resting state connectivity of the emotional brain (Huang et al., 2012).

The effects of insomnia on quality of life have been investigated by Kyle, Espie, and Morgan, (2010). They identified three areas of functioning which were affected by this disorder. Firstly, daily life was reported to be a struggle, with physical, emotional and cognitive effects reported. Secondly, people with insomnia reported feeling isolated from others, which was thought to be driven by a perceived lack of understanding, fear of labelling and skepticism, and their use of well-developed coping strategies. Thirdly, insomnia was reported to obstruct their desired, ideal self. In particular, people with insomnia perceived their disorder as inhibiting their ability to perform desired roles, such as at work, and expected lifestyle improvements with its removal. For example, insomnia was found to impact on social interactions, with participants reporting that they excluded themselves or avoiding committing to social interactions due to the unpredictability of sleep and its effects on functioning.

How does insomnia develop?

The experience of insomnia symptoms is common, and not all poor sleepers go on to develop insomnia disorder. Of baseline good sleepers with no prior lifetime insomnia episodes, 29% develop insomnia symptoms over a year, and 4% develop insomnia syndrome (LeBlanc et al., 2009). Among those with insomnia symptoms at baseline, 21% continue to have insomnia symptoms at follow-up, while 12% remit to normal sleep (Morin et al., 2009). However, odds ratios indicate that subjects with insomnia

symptoms at baseline are three times more likely to remit to normal sleep than develop insomnia syndrome (Morin et al., 2009). In order to try to explain how insomnia develops and perpetuates in some individuals but not others, several theories have been developed.

The neurocognitive model suggests that acute stress precedes acute insomnia, which is maintained by maladaptive coping strategies (Perlis et al., 1997). This creates conditioned (cortical) arousal to the sleep environment, which contributes to somatic arousal, and this in turn feeds in to cortical arousal (Perlis et al., 1997). Cortical arousal is also thought to affect cognition. Furthermore, this model acknowledges the interactions of insomnia with health, and the presence of insomnia is thought to contribute towards the development of illness. In turn, vulnerabilities towards, or the presence of, another disorder (whether psychiatric or physical), also interact with cognitive alterations. Three types of cognitive alterations have been identified within this theory, which affect sensory processing, information processing, and memory (Perlis et al., 1997). Sensory processing alterations affect sleep onset latency and night-time awakenings, and information processing alterations contribute to the objective/subjective sleep discrepancy. Thirdly, alterations in long term memory formation lead to an overestimation of wakefulness, which is relevant to sleep state misperception.

The 'misperception' of sleep in insomnia patients reflects their experience of this disorder, and may be the result of a dissociation between sleep and arousal systems (Riemann et al., 2010). Although people vary in their estimation of sleep time (Vanale et al., 2000), normal sleepers often over-estimate sleep time (Lauderdale et al., 2008), while those with insomnia tend to underestimate time asleep (Means et al., 2003). REM sleep instability may contribute to this effect in sleep maintenance insomnia (Riemann et al., 2012). In healthy subjects, a greater amount of slow-wave sleep has been linked to longer perceived sleep times (Aritake-Okada et al., 2009), and this sleep stage has been linked to the response to treatment in insomnia (Krystal and Edinger, 2010; see also Dijk, 2010).

The hyperarousal theory of insomnia suggests that insomnia is a disorder of 24-hour 'hyper-arousal', and this hyperarousal is directly relevant for the risk of depression and cardiovascular diseases (Bonnet and Arand, 2010). The evidence for this has been reviewed by Bonnet and Arand (2010), Riemann et al. (2010), and Bonnet and Arand (1997). Such hyperarousal is thought to be present at night and during the day, and can be measured physiologically via hormones and metabolism, body temperature, EEG,

cardiovascular activity, and sleepiness assessed via the Multiple Sleep Latency Test, or MSLT (Bonnet and Arand, 2010). These reviews suggest that an inappropriate activation of the central nervous system affects sleep-promoting systems, creating a dysregulation of the processes involved in sleep (Bonnet and Arand, 2010).

Other researchers have focussed on cognitive processes which contribute towards the development and maintenance of insomnia. Lundh and Broman (2000), describe how sleep-interpreting and sleep-interfering processes could contribute to insomnia. Sleep-interpreting processes are defined as those psychological processes which are directly relevant to how an individual defines an insomnia complaint. Such appraisals of sleep and daytime functioning include attributions, sleep-related beliefs, and perfectionism, and these affect the interpretation of sleep. Sleep interfering processes are those which affect sleep and arousal levels, such as stressful life events, depression, and worries. Levels of arousal are affected by stimulus-arousal associations, interpersonal relationships, behavioural and cognitive strategies, and arousability. Sleep-related appraisals and arousal interact with each other to result in the insomnia complaint (Lundh and Broman, 2000).

The cognitive model of the maintenance of insomnia suggests that arousal and distress contribute to perceived deficits of sleep and functioning, leading to a vicious cycle which perpetuates the insomnia disorder (Harvey, 2002). Furthermore, the stages of the cognitive model of insomnia can be initiated at any point (Harvey, 2002). In this theory, excessively negatively toned cognitive activity leads to arousal and distress, which in turn leads to selective attention and monitoring, which perpetuates the excessively negatively toned cognitive activity. Examples of selective attention and monitoring include monitoring of bodily sensations at night-time for signs of falling asleep, night-time clock watching, noting how rested one feels upon awakening, and daytime monitoring of fatigue, performance, functioning, sleep, and mood (Harvey, 2002). Such selective attention leads to a distorted perception of the deficit in sleep or functioning in those with insomnia, and affects mood, culminating in a real deficit. The excessively negatively toned cognitive activity also contributes towards safety behaviours, which exacerbate beliefs, and these safety behavioural and sleep-related beliefs both affect mood.

Such safety behaviours are motivated by specific feared outcomes, such as not being able to sleep following an emotional upset, or the perceived inability to cope with too many demands (Harvey, 2002). Sleep-related beliefs can lead to safety behaviours such as alcohol consumption, or taking an easy day (Harvey, 2002). Alcohol consumption

before bed can aid sleep initiation, but disrupts sleep continuity, and the belief that alcohol improves the ability to sleep is therefore not disconfirmed. Similarly, taking an easy day leads to boredom, and more time to worry about sleep, with the effect of reinforcing the belief that one does not function as well following a night of poor sleep. The distorted perceptions can relate to the night-time sleep deficit or effects on performance or functioning during the daytime, and can lead to objective night-time or day-time deficits.

An attentional focus on sleep, as described in the cognitive model of insomnia, can lead to perceived daytime effects (Semler and Harvey, 2006), a process which is implicated in the attention-intention-effort pathway (Espie et al., 2006). In this theory, the normal automatic processes of sleep become the focus of attention, which paradoxically impairs sleep initiation. Following a stressful life event, the associated physiological and psychological effects of stress lead to both the inhibition of sleep-related de-arousal, and selective attention toward stressors (i.e. adjustment insomnia). This, in turn, perpetuates the selective attention towards stressors, although ordinarily normal sleep would ultimately recover from this disruption. However, in some individuals this stress-related arousal and sleep disruption contribute towards the inhibition of de-arousal, and causes insomnia symptoms. Insomnia symptoms also lead to an attentional shift towards sleep cues, and this attentional bias also contributes to insomnia symptoms.

The attentional shift contains three components, which comprise the attention-intention-effort pathway. Firstly, this shift is implicit, but becomes explicit, and leads to the explicit intention to sleep. In turn, this leads to sleep effort, which can be direct or indirect. The effect of the attention-intention-effort pathway in insomnia development is to impair the supposed “automaticity” of normal sleep, and contribute to sleep onset or maintenance insomnia. The selective attention phase is one of vigilance, in keeping with an acute stress response. The explicit intention phase then develops, and is predominated by planning. Finally, the sleep effort processes is an emotionally-driven performance mode, where action is taken and behaviours are initiated with the intention to aid sleep. It has also been hypothesized that normal sleepers can fall asleep before they intend to, and that the process of completing a sleep diary may itself affect sleep (Espie et al., 2006).

How are sleep loss and insomnia disorder different?

Objectively poor sleep and an insomnia complaint may be somewhat different (Rosa and Bonnet, 2000). Poor sleep quality is affected by a range of factors, including stress (c.f.

Van Reeth et al., 2000), and physical and mental health conditions (Fernandez-Mendoza et al., 2012; Ohayon, 2002). For example, among students, stress and noise are common reasons for poor sleep (Lund et al., 2010). However, while transient insomnia is common, only a subset of people go on to develop chronic insomnia, which could suggest a role of genetic factors (related to vulnerability for sustained hyperarousal) in triggering this disorder (Riemann et al., 2010, see also Bonnet and Arand 2010). In support of this, Waters et al. (1993) report that emotion, stress, and attentional factors have greater effects on the sleep of insomnia subjects than healthy controls. Some individual differences which could contribute to insomnia, such as perfectionism and arousability, have been described by Lundh and Broman, (2000).

A questionnaire has been developed to assess vulnerability to stress-related sleep disruption (Drake et al., 2004). This asks participants about the likelihood of experiencing sleep disruption following, for example, a stressful event, or before an important meeting, and has been linked to disruptions in objectively measured sleep (Drake et al., 2004). High scorers show evidence of emotional dysregulation (Fernandez-Mendoza et al., 2010), and poor sleep following caffeine intake (Drake et al., 2006). Moreover, vulnerability to the effects of stress on sleep can run in families (Drake et al., 2008), as can sleep architecture, and the spectral composition of sleep itself (Ambrosius et al., 2008; Landolt, 2011), with evidence of trait-like differences in this (Buckelmüller et al., 2006; Bonnet and Arand, 2003).

In the psychophysiological model of good sleep, acute stress, for example, can cause transient sleep disruption, which the sleep homeostatic and circadian processes can typically accommodate without the development of an insomnia problem (Espie, 2002). De-arousal, which can be cognitive and physiological, ordinarily interacts with stimulus control and daytime actions to result in sleep which is both automatic and resilient. Sleep homeostasis, circadian timing, and sleep quality also reinforce the maintenance of good sleep. In the model of Espie, (2002), the 'inhibition of de-arousal' is thought to be enough to result in poor sleep, although additional factors could contribute to the development of insomnia, as a disorder as such.

Another way in which sleep loss and insomnia could differ is in the effects on sleepiness, which is not experienced by people with insomnia despite their subjective sleep complaint (Riemann et al., 2010). Adequate sleep opportunity may be used to help differentiate sleep loss from an insomnia disorder. One way in which sleep opportunity has been assessed is via sleep efficiency measures. Sleep efficiency, defined as the percentage of time reported asleep, divided by the reported time in bed,

can be calculated from sleep diaries (Espie and Kyle, 2009). Poor sleep efficiency can arise from excessive time in bed, or staying in bed while awake; maladaptive strategies which are often used by people with insomnia (Perlis et al., 1997). For example, a normal sleeper could report a total sleep time of seven hours and a time in bed of seven and a half hours, which would give a sleep efficiency of 93%. However, a person with insomnia disorder would report a diminished total sleep time, for example of five hours and 45 minutes. Combined with a time in bed of nine hours, this would result in a sleep efficiency of 64%. Importantly however, the extent to which sleep duration is objectively impaired in insomnia has been subject to debate, and it has been suggested that there may be two distinct insomnia phenotypes which differ in their sleep duration (Vgontzas and Fernandez-Mendoza, 2013; Vgontzas et al., 2013). In contrast, sleep loss would be expected to affect total sleep time, which could be reduced to five hours, and time in bed could be correspondingly reduced, resulting in sleep efficiency close to 100%. Additional measures of sleep discontinuity include the number of night-time awakenings, the sleep onset latency, and the wake time after sleep onset. Importantly, while sleep loss would likely result in a sleep complaint, such individuals would not be expected to endorse an *insomnia* complaint, as they would not have internalized poor sleep into their self-concept as is found in insomnia disorder (c.f. Kyle, Espie and Morgan, 2010).

Insomnia: The role of sleep homeostatic and circadian processes

Both sleep homeostatic and circadian processes have been implicated in the etiology of insomnia (e.g. Richardson, 2007; Pigeon and Perlis, 2006; Benoit and Aguire, 1996; Lack and Wright, 2007; Espie, 2002). People with insomnia are often found to show little evidence of sleepiness, measured objectively, and do not seem to report increased sleepiness (Riemann et al., 2010), suggesting that the daytime effects may be more related to the fatigue experienced in insomnia (Kyle, Morgan, and Espie, 2010). Intriguingly, in contrast to typical insomnia, a category of individuals has been described who show the daytime effects of poor sleep, but who deny insomnia, despite objectively poor sleep (Trajonovi et al., 2007; Schneider-Helmert, 2007; Attarian, Duntley, and Brown, 2004). This ‘positive sleep state misperception’ causes excessive daytime sleepiness which subjects do not detect (Trajanovic et al., 2007), and could be linked to how night-time awakenings are perceived (Schneider-Helmert, 2007). Importantly, both daytime functioning and night-time sleep seem to improve with treatment (Schneider-Helmert, 2007). The construct of sleepiness could itself be comprised of several factors, such as sleep propensity, sleep need, and subjective sleepiness (Horne, 2010).

Sleep propensity has recently been modeled by Bes, Jobert and Schulz, (2009), in a model with two basic drives, one homeostatic, and the other circadian-based. They found two major peaks in sleep propensity as well as two major troughs, corresponding to increased sleep propensity at night and in the early afternoon, and decreased sleep propensity in the early morning and in the early evening. The circadian clock could contribute to sleep onset problems in insomnia, due to attempts to sleep during the evening wake-maintenance zone (Strogatz, Kronauer, and Czeisler, 1987; see also Czeisler et al., 1980) and an eveningness diurnal preference seems to increase the adverse effects of insomnia (Ong et al., 2007), with implications for treatment (Lack and Wright, 2007). It has been suggested that in healthy subjects increasing wakefulness and the circadian low-point are linked to difficulties awakening and a lack of feeling refreshed (following forced awakenings), in spite of good subjective sleep (Akerstedt et al., 1997). In healthy subjects, increasing time in bed reduces both sleep propensity (Rosenthal et al., 1993), and sleep efficiency (Levine et al., 1988). Furthermore, the relationship of an eveningness diurnal preference with poor sleep quality could suggest some genetic overlap of these components (Barclay et al., 2010).

Discussion

The literature suggests that both insomnia and sleep loss affect daytime functioning. However, the daytime effects of sleep loss are well documented, whereas reports of the daytime effects of insomnia have been dominated by its subjective experience. Vulnerability towards insomnia seems to be important as sleep disruption is common in the general population, and is something which most people have experienced at some point. The role of arousal, and predisposing, precipitating, and perpetuating factors seem to be important in the development of chronic insomnia; a process which is arrested in normal sleepers. How homeostatic and circadian processes interact to result in normal sleep is complex, and an understanding of normal sleep seems to be important in order to fully understand the etiology of insomnia disorder. Also important is the development and identification of sensitive tasks which are relevant for people with insomnia, and emotion tasks have recently been used by sleep researchers. These tasks and results will be reviewed in the context of the emotion science literature, with a view to informing the experiments within this thesis.

Chapter Two

Social Interactions, Emotion and Sleep: A Systematic Review and Research Agenda

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LB conceived the study, conducted the analysis and wrote the paper, with the other authors contributing minor suggestions primarily regarding style.

Abstract

Sleep and emotion are closely linked, however the effects of sleep on socio-emotional task performance have only recently been investigated. Sleep loss and insomnia have been found to affect emotional reactivity and social functioning, although results, taken together, are somewhat contradictory. Here we review this advancing literature, aiming to 1) systematically review the relevant literature on sleep and socio-emotional functioning, with reference to the extant literature on emotion and social interactions, 2) summarize results and outline ways in which emotion, social interactions, and sleep may interact, and 3) suggest key limitations and future directions for this field. From the reviewed literature, sleep deprivation is associated with diminished emotional expressivity and impaired emotion recognition, and this has particular relevance for social interactions. Sleep deprivation also increases emotional reactivity; results which are most apparent with neuro-imaging studies investigating amygdala activity and its prefrontal regulation. Evidence of emotional dysregulation in insomnia and poor sleep has also been reported. In general, limitations of this literature include how performance measures are linked to self-reports, and how results are linked to socio-emotional functioning. We conclude by suggesting some possible future directions for this field.

Social Interactions, Emotion and Sleep: A Systematic Review and Research Agenda

Introduction

Sleep is known to be important for health (Buysse, 2014), and the health risks associated with sleep disruption include cancer, metabolic disorders, and cardiovascular illness (Luyster et al., 2014). The relevance of sleep to psychiatric disorders has also been established (Benca et al., 1992; Baglioni et al., 2011). Poor sleep quality and insomnia are pertinent to emotion, and previous studies have investigated the effects of loneliness, complicated grief, hostility, and impulsivity on sleep (Baglioni et al., 2010). Sleep and emotion are closely linked, and the importance of this area has been increasingly recognized.

Recently, experimental paradigms with socio-emotional stimuli (i.e. emotional faces, voices, images, or movies) have been employed to investigate how sleep affects responses to stimuli. Emotion perception can therefore be defined as the sensory processing of emotional stimuli. Emotion is relevant to the meaning, or significance, which is given to events, and emotion, cognition, and motivation are interlinked (Lazarus, 1991). Specifically, emotion can be associated with approach or withdrawal behavioral states, or motivational states of reward and punishment (Adolphs, 2002). Affective states comprise a conscious emotional feeling, with associated autonomic, neuroendocrine, and somatomotor responses (Phillips et al., 2003). Emotional stimuli are therefore highly relevant for the well-being and survival of the perceiver (Brosch, Pourtois, Sander, 2010).

Following the perception of a stimulus, the emotional significance to the perceiver is appraised (Phillips et al., 2003). This initial appraisal leads to an affective state and corresponding behavior (Phillips et al., 2003), which is assumed to be proportionate to a situation (Brosch, Pourtois, Sander, 2010). Threatening stimuli can lead to the initiation of a behavioral response (Brosch, Pourtois, Sander, 2010), with the initial appraisal contributing towards the resultant response. This correspondence of an affective response, behavior, and context, is disrupted in psychopathological conditions (Beck, 1971). In particular, the conceptualization of an event is associated with an excessive or inappropriate emotional response (Beck, 1971). This is due to a greater contribution of internal processes, such as idiosyncratic interpretations of an event (Beck, 1971).

Emotion regulation also contributes towards emotion perception. Emotion regulation can be defined as the modification of an affective response by the recruitment of cognitive processes. Phillips et al. (2003) suggest that emotion regulation contributes towards the initial appraisal of stimuli, and to the affective state produced by stimuli appraisal. This implies that emotion regulation can affect the perception of emotional stimuli and the subsequent emotional state, suggesting two types of emotional regulation strategies. However, emotion is one of the most contentious areas within psychology (Gross, Sheppes, and Urry, 2011), and the validity of distinguishing between emotion generation and emotion regulation is disputed (Gross and Barrett, 2011). The relationships between mood and emotional stimuli are therefore likely to be complex.

When the sleep of healthy subjects has been manipulated, evidence of increased emotional reactivity has been found, although evidence of emotional "blunting" has also been reported. Experimental tasks have also been used with poor sleepers, with a view to understanding the role of emotion in insomnia. However, results to date have raised some important questions, such as the directionality of emotion effects (i.e. emotional reactivity vs. emotional blunting), the concordance between different output measures (e.g. behavioral responses, physiological activity, brain responses), and the relative contributions of emotional functioning and social performance to results. A working definition of key terms is provided in Table 1.

Table 1. Working definitions of key terms

Key terms	**Definition**
Affective states, emotional states	"A conscious emotional feeling, with associated autonomic, neuroendocrine, and somatomotor responses" (Phillips et al., 2003).
Affective instability	"Rapid oscillations of intense affect, with a difficulty in regulating these oscillations or their behavioral consequences" (Marwaha et al., 2013).
Emotional blunting; decreased emotional reactivity/lability	Stable and slowly changing emotions due to hypo-reactivity to emotional stimuli.
Emotion perception	The perceptual processing of emotional stimuli.
Emotion regulation	The modification of an affective response by the recruitment of cognitive processes.
Emotional reactivity/lability (increased)	"Unstable and rapidly changing emotions due to hyper-reactivity to emotional stimuli" (Fountoulakis, 2010).
Socio-emotional stimuli	Emotional faces, voices, or human-related images.

Furthermore, these issues have not been addressed in existing reviews. In general, reviews to date have summarized the literature on the bi-directional relationships of

sleep and emotions, with a focus on self-reported measures of mood (Kahn, Sheppes, Sadeh, 2013). The literature on sleep stages, especially rapid eye movement (REM) sleep, and their role in emotional memory and associated affective experiences have also been reviewed (Deliens, Gilson, and Peigneux, 2014; Vanderkerckhove and Cluydts, 2010; Goldstein and Walker, 2014). However, a limitation of these reviews is that results from experimental tasks with socially-relevant stimuli or outcome measures are largely considered within an emotional context, rather than as measures of social functioning. This is relevant to the complex interactions between mood, emotional stimuli, facial expressions, and social interactions, and we aim to address these issues in this review.

Given the increasing use of objective measures to investigate the relationships between sleep and emotion, it seems timely to review the sleep/emotion field with reference to the existing literature on emotion and social interactions. Furthermore, integrating these areas seems necessary in order to aid the interpretation of results, and advance this area. In particular, we start by systematically reviewing the sleep and emotion literature, including sleep studies which have investigated emotion with experimental tasks and/or objective outcome measures. We summarize results from these studies, placing results in the context of the relevant extant socio-emotional literature, and highlight some key methodological limitations. Lastly, we set out an agenda for future work in this area.

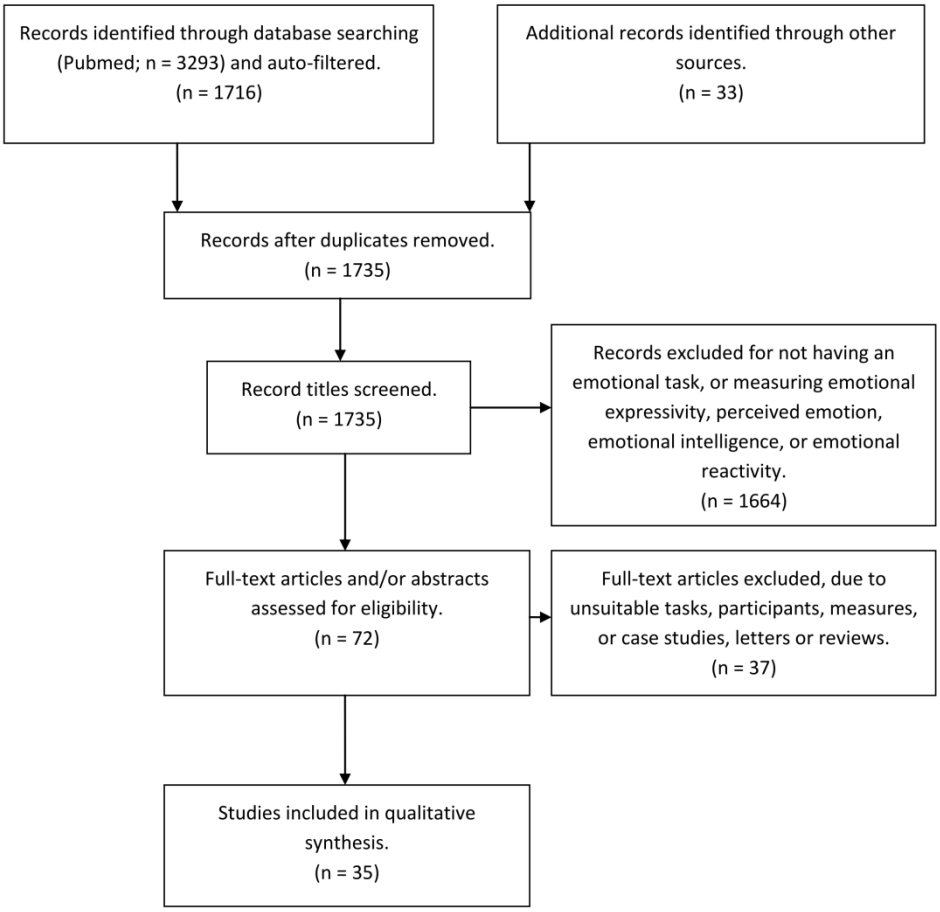
SLEEP AND EMOTION PERCEPTION

The effects of sleep on emotion perception have been increasingly studied, and we identified relevant papers in order to summarize this area, and this was done in two ways. Firstly, relevant papers were identified as published in an ongoing ad-hoc literature search. Secondly, relevant papers were identified via a systematic review of the literature. However, it should be noted that this systematic literature search was not linked to a quality rating of the papers, due to homogeneity in study designs and participants, dose of sleep deprivation, experimental tasks, and stimuli.

Papers were identified as indexed in PubMed (http://www.ncbi.nlm.nih.gov/pubmed/) via a search for the key terms relevant to sleep and emotion on 30th August 2014. The resultant 3293 items were then auto-filtered for article type, language, humans, and age. This resulted in 1716 items, and combined with those 33 papers previously identified resulted in 1735 unique papers.

We sought to identify those papers with an emotional task, and an associated neuroimaging, physiological, or behavioral dependent variable (DV). Emotional tasks were defined as those making use of social or emotional stimuli as independent variables (IV), such as emotional words, images, or faces. Also included were papers with measures of emotional expressivity, perceived emotion in sleep-deprived or insomnia individuals, measures of emotional intelligence in conjunction with an objective DV, and tasks of emotional reactivity in memory paradigms. Emotional reactivity is defined in Table 1 as "unstable and rapidly changing emotions due to hyper-reactivity to emotional stimuli" (Fountoulakis, 2010).

Figure 1. Outline of the literature search strategy



These 1735 titles were then manually screened to identify those papers meeting the above criteria, and supplemented by an auto-search to identify and review those papers with "emotion" in the title. In total 72 papers were identified and their abstracts screened, with the full text paper screened where there was ambiguity. Papers with self-reported measures of emotion alone were not included.

Excluded were tasks of attentional bias (n = 2), stress paradigms (n = 4), memory tasks (n = 5), priming task (n = 1), and other stimuli (food stimuli n = 1; humor n = 1), studies based on self-reports (n = 17), reviews (n = 1), letters (n = 1), case studies (n = 1), non-insomnia patients (n = 2), and a circadian study (n = 1). The resulting 35 papers were then organized by the use of neuroimaging, physiological, or behavioral measures, as well as studies of emotional expressivity, emotional intelligence, and rapid eye movement (REM) sleep, and are summarized in Table 2.

Table 2. Summary of papers included within the review.

	Author	Subjects	Design	Task	Stimuli	Measures	Result
1.	Baglioni et al. (i2014)	22 insomnia disorder, 38 healthy good sleepers.	Between-subjects.	Emotional images were shown in five blocks, repeated twice. Assessed habituation.	Emotional images. 10 neutral with low arousal levels. 10 negative stimuli with moderate arousal levels. 10 negative stimuli with high arousal levels. 10 insomnia-related negative stimuli with moderate arousal levels. 40 neutral stimuli with low arousal levels.	fMRI results. Recognition task.	Significant interactions of group and contrast (first presentation). Healthy good sleepers showed increased amygdala activation to negative stimuli versus neutral stimuli with similar arousal levels. There was no such difference in insomnia patients. Insomnia patients responded with increased activation to insomnia related stimuli compared to non-insomnia related stimuli. Healthy good sleepers showed the opposite pattern. Healthy good

							sleepers showed similar amygdala activation to the first and second presentations of neutral moderate, negative moderate, and sleep negative images. Insomnia patients displayed increased amygdala activation to the second presentation of neutral moderate images. Conversely, they showed decrease activation with regards to negative moderate and sleep negative. Healthy good sleepers also responded with increased amygdala activation towards negative stimuli compared to neutral, with no such effects in

							insomnia disorder.
2.	Cote et al. (2014)	49 healthy subjects.	Between-subjects. 24 subjects were sleep deprived.	Emotion face categorization.	Faces showing angry, sad, happy and fear, at different intensity levels (100%, 50%, 40% and 30%).	Behavioral; accuracy and reaction times. N170 and P1 ERP responses were analyzed.	Sleep deprived participants were less accurate towards sad faces and slower with the full expressions. Sleep deprived subjects were less accurate with morphed sad faces, and slower with morphed happy, sad, and angry faces. Sleep deprived subjects also had smaller P1 amplitudes and a larger N170 amplitude on the full face task. With morphed faces, sleep deprived subjects had a smaller P1 amplitude at 50% intensity, and main effects of sleep

							deprivation on N170 amplitude, with a larger amplitude in this group. There was also a significant three-way interaction on the N170.
3.	Kyle et al. (2014)	16 psychophysiological insomnia subjects; 15 controls.	Between-subjects.	Emotion face categorization and intensity judgments.	Faces showing anger, fear, happy and sad expressions.	Behavioral; accuracy and intensity judgments.	Lowered intensity ratings of sadness and fear in insomnia.
4.	DelVentura et al. (2014)	12 insomnia subjects; 13 controls.	Between-subjects.	Emotional images were shown, with intermittent startle probes or pain induction, followed by emotional ratings.	Emotional images depicting mutilation, neutral, and erotica.	Behavioral ratings, and physiological measures.	Insomnia subjects rated mutilation images as less unpleasant, and erotica images as less pleasant. Pain ratings were not modulated by image in insomnia. Strength of the relationship between NFR and pain was weaker in insomnia.
5.	Killgore, (2013)	65 healthy adults.	Between-subjects.	Emotional intelligence task and questionnaire. Sleep assessed via	Bar-On emotional intelligence inventory; Mayer-Salovey-	fMRI and subjective and objective emotional intelligence.	Less sleep was linked to lower subjective emotional intelligence. Sleep

				previous night's sleep duration.	Caruso intelligence test.		duration correlated negatively with prefrontal-amygdala connectivity.
6.	Weber et al. (2013)	55 healthy adults.	Between-subjects. Sleep assessed via habitual "sleep credit".	Emotional intelligence questionnaire.	Bar-On Emotional Intelligence Inventory (EQ-i).	fMRI and subjective emotional intelligence.	Sleep credit was correlated with greater grey-matter volume in two areas. EQ-i scores correlated with grey matter volume of the left grus rectus and superior and medial orbito-frontal gyrus. Grey matter volume in this area correlated with the interpersonal subscale of the EQ-i.
7.	Motomura et al. (2013)	14 healthy men.	Within-subjects. Sleep restricted to four hours of time in bed, with bed times four hours later than normal.	Presented with emotional faces under conscious and non-conscious viewing conditions.	Fearful, happy, or neutral faces.	fMRI, with button response to confirm wakefulness.	Significantly greater amygdala activation to fearful faces with sleep deprivation in the conscious condition. Significantly diminished

							amygdala – ventral anterior cingulate cortex connectivity with sleep deprivation, which also correlates with mood.
8.	Goldstein et al. (2013)	18 healthy adults.	Within-subjects. Sleep deprivation of one night.	Emotional anticipation task whereby subjects were presented with an anticipatory cue, followed by neutral, negative, or combination images, and response judgements.	Combination, negative and neutral images.	fMRI and behavioral (omitted trial and response times).	Sleep deprivation enhanced anticipatory brain activity. Sleep deprivation was linked to more omitted trials, but no effects on reaction times.
9.	Schwarz et al. (2013)	33 healthy subjects.	Within-subjects.	"Smile or "frown" to emotional images and faces.	Positive and negative scenes, and happy and angry faces.	Behavioral ratings (arousal and valence) and EMG responses.	No effects of sleep group on arousal and valence ratings. EMG responses were slower with sleep loss.
10.	Prather, Bogdan and Hariri, (2013)	299 participants.	Between-subjects. Sleep quality assessed via the PSQI.	Perceptual face-matching task.	Anger, fear, surprised and neutral faces.	fMRI.	Poor sleepers had a significant positive relationship between amygdala reactivity and measures of psychopathology,

							unlike good sleepers.
11.	Sundelin et al., (2013)	10 healthy adults were sleep deprived.	Within-subjects. Sleep deprivation period of 31 hours, preceded by a night of 5 hours of sleep.	Images were rated on cues related to fatigue, including sadness, dark circles under the eyes, pale skin, tense lips, and fatigue.	Photographs were taken of healthy adults when rested and following sleep deprivation.	40 observers rated faces for cues relevant to fatigue.	Sleep deprived individuals were perceived to be more sad and fatigued, with more swollen eyes, hanging eyelids, redder eyes, darker circles under the eyes, paler skin, more droopy corners of the mouth, and more wrinkles and fine lines around the mouth.
12.	Rosales-Lagarde et al. (2012)	20 healthy adults. 12 REM-deprived, 8 controls.	Between-subjects. REM sleep deprivation (REM-D), controls were awakened from other sleep stages.	Emotional reactivity task involved responding to image presentations with a button response.	Emotional images of negative and positive valence. High emotional reactivity (HER) images and low emotional reactivity (LER) images were	Behavioral responses (emotional reactivity and reaction times).	HER responses increased after REM-D, with no such effects in controls. Reaction times at second test were decreased in the REM-D group for LER responses.

					identified for each individual.		
13.	Baran et al. (2012)	106 healthy adults.	Between-subjects. Four groups, with testing before and after sleep, before and after wakefulness, morning testing, and evening testing.	Encoding phase with emotional judgments (arousal and valence), followed by a surprise recognition phase with emotional judgments (arousal and valence).	Emotional images (negative and neutral).	Behavioral arousal and valence, and memory.	Sleep group were more accurate with negative and neutral images, and made less false alarms. Higher valence ratings in the wake group than the sleep group, with ratings in the wake group becoming more neutral. Similar results were found with valence in the wake group, whose responses became more neutral.
14.	Minkel et al. (2012)	97 adult volunteers. Sleep was assessed via the PSQI.	Between-subjects.	Emotional regulation task, whereby emotional images are presented, with subjects cued to look at the stimulus, or decrease their emotional response, followed by ratings.	Neutral and negative images.	fMRI and behavioral (emotional reactions, neutral-negative).	Higher PSQI scores, and sleep medication use, were associated with less medial prefrontal cortex activity.

15.	Gujar et al. (2011)	36 healthy subjects.	Between-subjects.	Subjects were allocated to a nap/no nap group, and completed an emotion face task twice.	Emotion faces of angry, fear, happy and sad were morphed with neutral to create an emotional range.	PSG and behavioral intensity ratings.	In the no-nap group, anger ratings were significantly increased at the second test, a result not found in the nap group. Fear ratings were also significantly reduced in the nap group, and increased in the no-nap group. Happy faces showed increased ratings at re-test in the nap group, with no significant change in the no-nap group. The effects with happy and fear in the nap group were driven by those who had achieved REM sleep.
16.	Pace-Schott et al. (2011)	46 volunteers.	Between-subjects.	Subjects were allocated to a nap/no nap group, with the emotion task repeated twice.	Emotional images (negative and neutral).	Behavioural (valence and arousal), ECG, EMG, SCR.	Significant effects of nap/wake with physiological measures ("frowning" EMG

							and SCR).
17.	Van Der Helm et al. (2011)	34 healthy adults.	Between-subjects.	Participants rated the emotional intensity of images presented before and after sleep and wakefulness.	Emotional images, of positive and negative valence and low and high arousal.	fMRI and behavioral intensity ratings.	Amygdala reactivity decreased, and ventromedial prefrontal cortex activity increased following sleep. Amygdala activity increased, and ventromedial prefrontal cortex activity increased following an equivalent period of wakefulness. Emotional intensity ratings decreased in the sleep group and increased in the wake group. The extent of reduced prefrontal gamma during REM significantly predicted this effect.
18.	Anderson and Platten, (2011)	32 good sleepers, 16 per group.	Between-subjects. 36 hours of sleep	Emotional Go/NoGo task.	Neutral or emotional (positive or	Behavioral; hit rate and response times.	Faster incorrect responses and increased failure to

			deprivation.		negative) words.		inhibit a response.
19.	McGlinchey et al. (2011)	55 healthy participants, 17 adults.	Within-subjects. Sleep period the previous night of a maximum 2 hours.	Speak freely interview.	N/A	Computerized and human rater (eight raters) analysis of expressed emotion, computerized acoustic properties.	Increased negative expressed emotion, and alterations in vocal properties.
20.	Minkel et al. (2011)	23 subjects. 15 subjects were sleep deprived; 8 controls.	Between-subjects. Sleep deprivation period of one night.	Emotion-inducing films were shown, with responses recorded.	Movie clips to induce sadness and amusement.	Expressed emotion, facial movement analyzed via the FACES scoring system (two raters).	Sleep deprivation was linked to fewer facial movements to both types of movies.
21.	Gujar et al. (2011)	14 sleep deprived, 13 controls.	Between-subjects. Around 32 hours of sleep deprivation.	Viewing of increasingly pleasant images.	Emotional images raging from neutral (neutral valence, low arousal) to increasingly positive (positive valence, high arousal).	Emotional classification (pleasant/neutral). fMRI and behavioral response (pleasant/neutral).	With sleep deprivation, an increased activation e.g. in brain areas responsible for reward, and altered functional connectivity, and a greater tendency to categorize stimuli as pleasant.
22.	Tempesta et al. (2010)	40 subjects; 20 per group.	Between-subjects. One night of total sleep	Passive viewing followed by behavioral responses.	Neutral, pleasant, and unpleasant images.	Behavioral; arousal and valence judgments.	Increased unpleasant ratings of neutral stimuli following sleep

			deprivation.				deprivation.
23.	Baglioni et al. (2010)	18 good sleepers; 21 primary insomnia.	Between-subjects.	Passive viewing.	Neutral, negative, positive, sleep negative and sleep positive images.	Heart-rate, cardiac vagal tone, facial EMG, subjective valence and arousal ratings.	Insomnia subjects showed decreased sleep-positive tonic responses of the corrugator, unlike good sleepers. Insomnia subjects had enhanced CVT, and no differences in the valence ratings of sleep-negative and general negative stimuli; and rated all stimuli as more arousing.
24.	Chuah et al. (2010)	24 healthy subjects.	Within-subjects. 24 hours of sleep deprivation.	Delayed match-to-sample working memory task for faces, with emotional distracters.	Images were high arousal negative, low arousal neutral, or digitally scrambled versions.	Behavioral ratings and fMRI.	Sleep deprivation impaired working memory performance, but did not affect ratings of intensity or distractibility. Working memory decreases correlated with increased amygdala activation to distracters with sleep deprivation, and functional

							connectivity.
25.	Van Der Helm et al. (2010)	37 healthy subjects. 20 sleep deprived and 17 controls.	Between-subjects (total sleep deprivation vs. controls).	Intensity judgments of emotion faces.	Faces displaying anger, sad, and happiness morphed with neutral.	Behavioral intensity ratings.	Sleep deprived subjects made lower intensity ratings of happy and angry faces.
26.	Kuriyama, Soshi, and Kim, (2010)	28 healthy students. 14 sleep deprived, 14 controls.	Between-subjects. One night of sleep deprivation.	Passive viewing (memory encoding).	Movies of safe driving and of a motor vehicle accident.	Recognition accuracy (old-new) and fear rating, skin conductance response (SCR).	Sleep deprivation diminished the fear rating of SAFE movies. Sleep deprivation diminished the SCR in all contexts to incorrectly identified stimuli.
27.	Lara-Carrasco et al. (2009)	35 healthy subjects. 17 REM deprived, 18 controls.	Between-subjects (REM sleep deprivation vs. controls).	Arousal and valence ratings of emotional images at night and in morning.	Emotional images, including neutral and negative images.	Behavioral ratings (valence and arousal) and PSG.	Emotional adaptation (arousal) scores were significantly lower in the in those subjects with a high REM %. No effects were found with neutral images or valence ratings.
28.	Franzen et al. (2009)	30 healthy volunteers. 15 subjects per group.	Between-subjects. One night of total sleep deprivation.	Passive viewing task.	Emotional images (neutral, positive and negative).	Behavioral ratings of valence and arousal and pupillography.	Sleep deprived subjects showed anticipatory reactivity to negative blocks, and larger pupil

							diameters to negative images.
29.	Franzen et al. (2008)	29 healthy adult volunteers. 15 subjects per group. Between-subjects.	One night of total sleep deprivation.	Objective and subjective measures of sleepiness and mood.	Positive, negative, and neutral images.	Self-reports, behavioral, and physiological measures.	Distinction of subjective and objective measures was less clear following sleep deprivation.
30.	Huck et al. (2008)	54 healthy subjects.	All subjects sleep deprived, up to 44 hours of sleep deprivation. Between-groups comparisons. 3 drug conditions and a placebo group.	Recognition of angry, sad, happy, surprised, fearful and disgusted faces.	Ekman 60 Faces test, the Emotion Hexagon Test.	Behavioral; accuracy.	Sleep deprivation impairs recognition of complex emotions, which all drugs improve.
31.	Yoo et al. (2007)	14 sleep deprived, 12 controls.	Between-subjects comparisons. Around 35 hours of sleep deprivation.	Viewing of increasingly aversive images. Emotion classification response to verify wakefulness.	Emotional images, ranging from emotionally neutral to increasingly aversive.	fMRI and behavioral (unpleasant/neutral).	Sleep deprived subjects showed amygdala responses with impaired prefrontal connectivity.
32.	Pallesen et al. (2004)	36 cadets.	Within-subjects. Sleep deprivation	Delayed match to sample task with faces, presented to visual half-fields.	9 schematic facial expressions.	Behavioral; reaction times and accuracy.	Less accurate with sleep deprivation. Significant interaction of half

			period of 72 – 120 hours.				field with state on reaction times.
33.	Wagner, Fischer, and Born, (2002)	24 healthy subjects.	Within subjects.	Emotional judgments were made before and after sleep which was taken early or late at night.	Aversive images.	Behavioral ratings (arousal and valence).	Old-new valence ratings were more positive after early sleep and more negative after late sleep. Old-new arousal ratings were greater after sleep than wakefulness.
34.	Harrison and Horne, (1997)	9 healthy subjects.	Within subjects. One night of sleep deprivation (36 hours).	Word fluency task and short story oral reading.	N/A	10 trained raters.	Decreased word count during sleep deprivation; small increase on control nights (trial 3). Larger proportion of semantically-related words during the last two trials of sleep deprivation (trial 2 and 3). Diminished number of nouns and adjectives during trial 3 of sleep deprivation.

							Deterioration of intonation by day two of sleep deprivation, and sleep deprivation increased fatigue.
35.	Dushenko and Sterman, (1984)	10 healthy subjects.	Within subjects (sleep adaptation night and REM deprivation).	Left and right hemisphere tasks were presented to subjects tachistoscopically.	25 cartoon drawings of adult faces (extremely positive, mildly positive, neutral, mildly negative, extremely negative). 80 abstract nouns.	Same/different judgments (accuracy and reaction times).	There was a significant improvement in performance in the night 2 to night 3 left hemisphere first presentation of the facial stimuli. The left hemisphere first presentation of faces was linked to higher accuracy than the right hemisphere first presentation.

Abbreviations: electrocardiogram (ECG), electromyogram (EMG), emotional quotient inventory (EQ-i), event related potentials (ERPs), functional magnetic resonance imaging (fMRI), high emotional reactivity (HER), low emotional reactivity (LER), Nociceptive pain reflex (NPR), polysomnography (PSG), Pittsburgh sleep quality index (PSQI), rapid eye movement sleep (REM sleep), rapid eye movement deprivation (REM-D), skin conductance response (SCR).

EFFECTS OF SLEEP LOSS IN NORMAL SLEEPERS

Firstly, we review evidence for effects of sleep deprivation and sleep restriction on emotional responding. These studies of normal sleepers used neuroimaging, physiological and behavioral techniques. We then review those studies on emotional expressivity and emotional intelligence.

Neuroimaging studies

In 2007, Yoo et al. (2007) reported that 35 hours of sleep deprivation increases amygdala reactivity to a series of increasingly aversive images. In particular, they report that sleep deprivation was associated with 60% greater magnitude of activation of the amygdala, and a three-fold greater extent of activation of the amygdala volume between groups. Similar results were found when the upper and lower quartiles of the stimuli set were compared, suggesting that amygdala reactivity to the baseline condition was not responsible for these results. Diminished amygdala-prefrontal connectivity was also found after sleep deprivation, suggesting a lack of cognitive control over emotional brain areas. However, there was greater connectivity within brainstem areas which are involved in autonomic activation. Behavioral responses (unpleasant/neutral) were also recorded to verify wakefulness. Although no significant group differences were found in the use of these labels, sleep deprived subjects tended to rate stimuli as more negative ($p = 0.10$).

The same group has also reported hyper-reactivity to positive stimuli following sleep deprivation. Gujar et al. (2011) used a similar paradigm whereby increasingly arousing positive images were presented in an emotional gradient. A night of sleep deprivation (32 hours) increased activation in brain areas responsible for reward, such as the ventral tegmental area of the brain stem, as well as the amygdala and insula cortex between groups. Specifically, sleep deprivation increased functional connectivity within regions of the left amygdala and left anterior temporal pole, and connectivity of the insular cortex with several regions of the visual cortex was increased relative to control subjects. The bilateral amygdala showed increased reactivity, and decreased connectivity with the medial prefrontal cortex, bilateral orbitofrontal cortex, and left fusiform gyrus. The fusiform gyrus showed stronger connectivity with the left anterior temporal pole, and increased connectivity in the left superior insula and left lateral prefrontal cortex. There was also a significantly greater tendency of sleep deprived subjects to categorize stimuli as pleasant compared to neutral.

Using comparable tasks, Yoo et al. (2007) and Gujar et al. (2011) therefore report similar results with both negative-arousing and positive-arousing stimuli, i.e. increased

emotional brain activation and decreased functional connectivity with cognitive control regions. The test times of these studies are also comparable, with both studies testing subjects at 5pm. However, the presentation of stimuli in an increasingly arousing gradient could affect results. In particular, this approach seems likely to conflate the initial perception of stimuli with the capacity to regulate subsequent emotion. Specifically, antecedent-based methods of emotional regulation affect the perception of the emotion-inducing stimuli, whereas response-based methods of emotional regulation target the subsequent emotional response (Green and Malhi, 2006). Emotional regulation therefore contributes towards the appraisal of stimuli, as well as the subsequent emotional response and behaviour (Phillips et al., 2003). Both studies report evidence of impaired emotional regulation via connectivity measures, and the intensity of an emotional experience could modulate its ability to be regulated (Green and Malhi, 2006).

Sleep deprivation has also been studied in conjunction with a task of emotional distracters, in a counterbalanced crossover study (Chuah et al., 2010). In this delayed matching to sample task, task subjects were first instructed to remember 3 faces. After a 10 second delay subjects were asked to judge whether a newly presented face was old or new. In the interim, emotional images of highly arousing negative images, low arousal neutral scenes, or visual control images were presented as distracters. Following 24 hours of sleep deprivation, performance in this delayed-matching-to-sample task was significantly impaired in comparison to rested wakefulness (Chuah et al., 2010). When functional magnetic resonance imaging (fMRI) results were analyzed, amygdala activation to emotional distracters following sleep deprivation was found to correlate with working memory impairments. In particular, those with greater working memory impairments following sleep deprivation showed greater amygdala activation to emotionally distracting images (Chuah et al., 2010). The maintenance of performance was also positively associated with connectivity of the emotional brain and cognitive control areas. However functional connectivity between the amygdala and prefrontal cortices was impaired following sleep deprivation. Sleep deprivation was not found to affect behavioral ratings of emotional stimuli, with distractibility and emotional intensity assessed (Chuah et al., 2010). Importantly, this study suggests ways in which neural activation patterns and task performance are linked, indicating that increased amygdala reactivity to distracters and diminished prefrontal-amygdala connectivity correlates with working memory impairments. This could be relevant to increasing resilience to sleep deprivation.

The effects of 24 hours of sleep deprivation on the anticipation of emotional stimuli have also been studied in a within-subjects design (Goldstein et al., 2013). In this task, a cue predictive of aversive or neutral images, or an ambiguous cue, was presented in advance of an emotional image. Sleep deprivation enhanced anticipatory brain activation to all predictive cues in the amygdala region, without emotion-specific effects. There was also a significant interaction of cue type with sleep deprivation in the right anterior insula, which showed greater activation to emotional cues than ambiguous cues (Goldstein et al., 2013). Furthermore, trait anxiety was found to modulate these effects, with higher scores linked to greater amygdala activity. Identifying factors which increase vulnerability to sleep loss is important given the links of sleep and health (Buysse, 2014; Luyster et al., 2012; Benca et al., 1992; Baglioni et al., 2011) and amygdala reactivity could contribute to this. These results by Goldstein et al. (2013) appear broadly comparable to the effects of acute stress on amygdala activation (Van Marle et al., 2009). In particular, acute stress induction increases amygdala activation to all images, reducing specificity and increasing sensitivity. Such effects of sleep deprivation on amygdala reactivity could contribute towards the development of psychiatric disorders. Specifically, sleep disruption has been found to increase the likelihood of psychiatric disorders developing following a traumatic event (Bryant et al., 2010).

Emotional images have been used as stimuli in these four experiments. However other authors have used facial expressions of emotion in order to assess emotional responding. Facial stimuli are different from emotional images, in that facial expressions of emotion communicate information (Blair, 2003; Ekman, 1997) and serve social motives (Fridlund, 1991). These different types of emotional stimuli - words, images, and faces - have been compared, with different effects found on the speed of processing, extent of neural activation, and induced emotion. Specifically, compared to faces, images are more complex and novel, and may be more demanding to process, resulting in sustained patterns of activity (Britton et al., 2006). Emotional images also induce greater experienced emotion than emotional faces (Britton et al., 2006). However, brain responses to emotional faces are more pronounced in several brain areas (Britton et al., 2006). Distinctive universal facial expressions have been identified for anger, fear, sadness, enjoyment, and disgust, with weaker evidence found for other emotions (Ekman, 1993). In particular, anger/disgust and fear/surprise seem to be frequently confused, creating four emotional categories (Jack et al., 2014). These “basic” emotions can themselves be further classified according to their associated levels of arousal and valence, in a dimensional approach to emotion (Adolphs, 2002; Gerber et

al., 2008; Dailey et al., 2002). The following studies in this section have made use of emotional face stimuli.

Cote et al. (2014) measured the effects of sleep deprivation (31.5 hours of wakefulness) on emotion recognition, using behavioral measures in conjunction with event-related potentials (ERPs). When the behavioral response categorization labels were analyzed, sleep deprived subjects were significantly poorer at recognizing sad faces. These subjects were also slower to respond when the full emotional expressions were displayed. When images were morphed to vary the emotional intensity, sleep deprived subjects were less accurate at recognizing sad faces, and slower to recognize happy, sad, and angry faces. On the full-face task, sleep deprived subjects evidenced a smaller P1 amplitude and a larger N170 amplitude, with similar results found with morphed faces. At 50% morph level the P1 amplitude was also significantly smaller, and there was a significant three-way interaction for the N170 amplitude. These effects were linked to the increased perceptual difficulty of morphed faces, with a failure to make use of perceptual resources with subtle expressions of sadness. Neural reactivity increased for the threat-related emotions as they became more subtle. This suggests increased reactivity towards more ambiguous expressions with sleep loss, consistent with a behavioral study by Van Der Helm et al. (2010).

The effects of sleep restriction on emotion have also been investigated, and evidence for emotional reactivity has been found with facial stimuli. In the paradigm of Motomura et al. (2013), time in bed was restricted to four hours a day for five days. These authors report that sleep restriction increased emotional brain responses to negative face stimuli. Emotional faces showing happy, fearful, and neutral expressions were presented in two conditions. These stimuli were shown for 1000 ms in an “aware” condition, and 26 ms in an “unaware” condition, in a randomized crossover design. Significant differences in amygdala activation were found in the aware condition, when fearful and neutral faces were contrasted. This contrast was also associated with significantly impaired amygdala-anterior cingulate cortex connectivity (Motomura et al., 2013). When behavioral responses were analyzed, there were no significant differences between groups in the responses or reaction times towards target images. However, button responses were used to verify wakefulness rather than to assess recognition performance. In general, decreasing stimuli presentation times of emotional faces impairs recognition (Calvo and Lundqvist, 2008), and it would be interesting to investigate how this paradigm is linked to behavioral recognition responses.

Also of interest would be the effects of different levels of sleep restriction and sleep deprivation on performance. In particular, there has been found to be a dose-response effect of sleep loss on measures of neurobehavioral functioning (Van Dongen et al., 2003). These authors report that cumulative sleep restriction results in performance impairments which are comparable to that of total sleep deprivation. Meerlo et al. (2008) have also discussed the effects of sleep restriction with regards to markers of stress and arousal. They report that the adverse effects of chronically restricted and disrupted sleep can occur in two ways. Firstly, the effects of sleep deprivation on sympathetic and hypothalamic–pituitary–adrenal axis (HPA axis) activity could accumulate. Secondly, the chronic effects of this could be to dysregulate the stress and arousal systems, resulting in altered sensitivity and responses to stress. These changes become persistent via gradual changes on the stress systems and its regulation.

Physiological studies

Evidence of hyper-reactivity following sleep deprivation has also been found with physiological measures, specifically, there are greater pupillary responses to negative images when sleep deprived (Franzen et al., 2009). In this task, high arousal positive, high arousal negative and neutral images were selected, and presented in emotion blocks of 5 images. Each image was preceded by a 2 second warning cue, and presented for 6 seconds. Subjects were awake from 31-33 hours. There was greater anticipatory pupillary reactivity to negative stimuli following a night of sleep deprivation. Larger pupillary reactivity in the inter-stimulus interval following neutral trials was found with sleep deprivation too, along with effects while viewing negative images (Franzen et al., 2009). However, there were no significant effects on emotion ratings or reaction times with sleep deprivation (Franzen et al., 2009). This study supports the evidence for anticipatory reactivity following sleep deprivation reported by Goldstein et al. (2013) on fMRI parameters. Moreover, behavioral results appear to be less sensitive to the effects of sleep deprivation, both in this study and Chuah et al. (2010).

Measures of facial movements have been linked to a somewhat different pattern of results, and sleep loss slows intentional movements (Dimberg and Thunberg, 1998). Schwarz et al. (2013) asked subjects whose sleep opportunity had been partially restricted (sleeping between 2am and 6am) to “smile” or “frown” in response to emotional faces and scenes. Using electromyography (EMG) responses, they found that sleep deprived subjects were slower to respond. Dimberg suggests that the facial muscles involved in smiling and frowning respond congruently to emotional stimuli (Dimberg and Thunberg, 1992; Dimberg, 1990; see also Achaibou et al., 2008), although

importantly these movements may not be visible by eye (Dimberg, 1990). Modulating affective displays may, in turn, affect emotional experiences. In the facial feedback hypothesis, Izard (1990) suggests that modulating expressed emotion affects the emotional experience itself. Facial muscle movements could therefore contribute towards both emotional experience and emotional regulation (Izard, 1990).

Furthermore, the relationships between different types of measures could be affected by sleep deprivation. Franzen et al. (2008) examined how sleep deprivation affects the relationships between different measures, using the same sample as Franzen et al. (2009). In exploratory factor analyzes, they found that sleep deprivation influenced the relationships between subjective measures (such as self-reported mood and sleepiness) and objective measures (such as pupillography, and the multiple sleep latency test, or MSLT), making this distinction less clear (Franzen et al., 2008). This suggests that the relationships between the different components of emotions become dysregulated following sleep deprivation.

Behavioral studies

The effects of sleep deprivation on the ratings of emotional images have also been tested. Subjects were compared on their ratings of arousal and valence towards images depicting pleasant events, unpleasant events, and neutral. Images were presented in a random order for two seconds and rated after a one second pause. Behaviorally, a night of sleep deprivation was linked to more negatively valenced ratings of neutral images, with no effects on positive or negative stimuli when test and retest were compared (Tempesta et al., 2010). This group difference was significant when mood was included as a covariate. Some evidence of effects with arousal ratings were also reported, with sleep deprived subjects rating unpleasant images more arousing than pleasant ones, an effect not found in control subjects (Tempesta et al., 2010). This task appears comparable to that of Franzen et al. (2009), although with discrepant results. In particular, Franzen et al. reported no effects with behavioral ratings.

One explanation may relate to the precise stimuli which were selected, and their associated normative ratings of valence and arousal. However, these ratings appear comparable (Tempesta et al. (2010), pleasant valence: 8.0, arousal: 5.1; neutral stimuli arousal: 5.0, valence: 3.0; unpleasant valence: 2.0, arousal: 6.0; Franzen et al. (2009) positive valence: 7.6, arousal: 5.3; neutral valence: 5.0, arousal: 3.3; negative valence: 2.3, arousal: 5.7; all ratings out of 10). However, Franzen et al. collected behavioral ratings several hours after the eye-tracking task, and Tempesta et al. displayed stimuli

on screen while response judgments were made. These differences may be relevant to the discrepant behavioral results between these two tasks. Also relevant could be that the images of Tempesta et al. were also presented in color whereas Franzen et al. used grayscale images. Additionally, the sleep deprivation period of Franzen et al. appears to be longer. Future studies should investigate the reasons for these discrepant behavioral results in greater depth. However, in general, behavioral studies appear to be less sensitive to the effects of sleep deprivation than neuroimaging or psychological studies.

Sleep deprived participants also show evidence of diminished inhibition and greater impulsivity to negative stimuli, in an emotional Go/NoGo task (Anderson and Platten, 2011). This task involves the presentation of neutral and emotional (positive and negative) words, which subjects are asked to respond to, or inhibit responses towards. Following 36 hours of sleep deprivation, subjects made fewer correct responses overall, with more incorrect responses to negative emotional stimuli. Sleep deprived subjects also responded more quickly (and incorrectly), to negative stimuli, suggesting a failure to inhibit responses to such stimuli (Anderson and Platten, 2011). When emotion words are compared to faces, emotion faces are processed faster, although with similar patterns of brain activity (Schacht and Sommer, 2009), suggesting that word stimuli are comparable to results with facial stimuli. However, it would be interesting to repeat this task using alternative stimuli, and using alternative measures of impulsivity.

Facial stimuli have also been used to investigate the effects of sleep deprivation. Pallesen et al. (2004) used schematic faces within a delayed matching-to-sample task. In this study of cadets, sleep deprivation was found to impair performance on accuracy and reaction times in the face-matching task. Dushenko and Sterman (1984) have also investigated the effects of sleep loss, using a matching task with schematic faces. Following REM deprivation, the recognition of these faces was found to improve in the left hemisphere first presentation condition. Both of these studies were interested in hemispheric differences. In general task performance was impaired following sleep loss, and improved following REM sleep deprivation, although the emotion specificity of results was not reported. These studies indicate that the nature of sleep deprivation affects results, with different sleep stages exerting different effects on performance.

In an emotion categorization task, Huck et al. (2008) found no effects of sleep loss on simple emotion recognition, although complex emotion recognition was significantly affected. Stimulant medications were found to improve recognition of complex emotions following sleep deprivation. The emotion-specificity of these effects was not reported. In this experimental paradigm faces are presented until response, with the six

emotional labels (anger, surprise, fear, sadness, happiness and disgust) presented on screen below the image. A similar task was employed in a complex emotion recognition task. Complex emotional faces were defined as those which are blends of two emotional faces, resulting in a ratio of two emotions which are displayed on the face. However, the extent to which such "complex" images are representative of how expressions are formed in real life is unclear. Different facial action units (AUs) may be displayed in different ways to represent emotional expressions (Yu et al., 2013). The facial physiology is capable of displays of more than one emotional state at once, and emotion blends or sequences are commonplace (Ekman and Friesen, 1969). This flexibility contributes to the ability to modulate facial expressions of emotion, depending on the situation (Ekman, 1992). Such emotional expressions may therefore be said to increase perceptual difficulty (Cote et al., 2014).

Also using face stimuli, Van Der Helm et al. (2010) found reduced intensity ratings (in the mid-intensity range) following sleep deprivation, results which were driven by responses towards angry and happy faces. Subjective sleepiness was not found to correlate with results. In this task the stimuli were created by morphing expressions of anger, sadness, and happiness with the neutral image of a single male identity. The resultant ten blends, or morphs, were shown in emotional blocks in a randomized order. Participants were familiarized with the image sets prior to testing, and rated the perceived intensity of emotion. This was done on a four-point scale (definitely neutral, more neutral than emotional, more emotional than neutral, definitely neutral) following a two second image presentation.

Behavioral facial emotion recognition tasks typically involve the random presentation of the emotional expressions of several posers, with subjects asked to categorize the emotion shown using emotion labels. This is akin to real-life situations, where emotions are recognized from several options. Emotion recognition can also occur at high accuracy following a brief image presentation (Calvo and Lundqvist, 2008), and extended presentation durations (10 seconds) have been linked to results which fail to replicate under more naturalistic presentation times (Cooper, Rowe, and Penton-Voak, 2008). Although emotion categorization and intensity responses are linked (Utama et al., 2009), the presentation of stimuli within emotion blocks could also have affected results. For example, angry faces have been linked to sensitization responses within the hippocampus and other brain areas (Strauss et al., 2005). We suggest that sensitive tasks of emotion recognition performance could include categorization and intensity judgments, or the use of dynamic stimuli based on facial movements (Yu, Garrod, and Schyns, 2012). Alternatively, Adolphs (2002) has suggested that intensity ratings could

be made concurrently on several different emotional categorization options for each trial. Measures of accuracy could then be derived from these responses.

Emotional expressivity

While the previous studies have investigated the perception of emotion, other authors have been interested in how emotions are expressed. This is relevant to both social interactions and emotional regulation. Specifically, Minkel et al. (2011) induced amusement or sadness via movie clips, and subjects' facial movements were visually recorded. Sleep deprived subjects were significantly less facially expressive when shown both types of movies compared to rested controls, with a larger effect size towards amusing clips than sad movies. However, this study assessed overall expressivity of facial movements via the "FACES" scoring system. As such, the way in which sleep deprivation affects the specific components of emotional expressions is unknown. With regard to facial expressions as social signals, Ekman and Friesen (1976) and Ekman, Friesen, and Tomkins (1971), detail how different AUs combine to display the different emotional expressions. How facial expressivity was linked to expressed emotion in this study is therefore unclear, although the precise links of facial expressions to emotional experiences is subject to debate.

Ekman (1992) suggests that some emotions have no distinct expression, and that subjective affect may not correspond to facially expressed emotion. This indicates that the processes involved in emotional experience and emotion expressions are separable. Emotional expressions serve social motives (Fridlund, 1991), and the "readout" hypothesis suggests that motivational-emotional processes with social implications are displayed on the face (Ekman and Friesen, 1976). Furthermore, Blair (2003) stresses the role of expressions in communication, with an audience important to emotion displays. Ekman and Friesen (1969) have described four display rules, which can serve to de-intensify, over-intensify, appear neutral, and mask experienced emotion, and these are subject to social norms and context. Such modulations of affective displays may, in turn, affect emotional experiences (Izard, 1990). Expressing fear alters parameters (e.g. faster eye movements, increased nasal volume) which are linked to behavioral action tendencies (Susskind et al., 2008). Future studies in this area should consider reporting the social context of testing (e.g. solitary, group testing, or experimenter present), and this would be interesting to investigate further. Furthermore, the environment has been identified as a fourth component of emotional instability (Marwaha et al., 2013) (see Table 1).

Individual differences in emotional expressivity could also be relevant. People vary in their emotional expressivity, and this relates to differences in the specificity of expressions, the threshold for expressions, and aspects of the timing of expressions (Ekman, 1992). Furthermore, there are differences between people in the structure and differentiation of facial muscles, and in the neural control of them (Schmidt and Cohn, 2011). The ability and tendency to produce universal facial expressions also varies between people (Schmidt and Cohn, 2011). Such differences have been observed experimentally. In 1924, Landis conducted a series of experiments whereby the facial expressions produced by emotion-inducing stimuli were recorded, and noted two factors of emotional disruption. One was related to the tendency to display emotion, and the other was the emotional stability, or duration of emotional impact, following an emotional upset. As such, individuals have been found to respond in idiosyncratic ways to emotion-inducing procedures, with different patterns of facial expressions observed between individuals (Landis, 1924). Future studies could consider how facial reactivity relates to measures of emotion, and how sleep affects this.

Sleep deprivation also affects vocal expressivity (McGlinchey et al., 2011). Following a night of sleep deprivation, subjects use fewer words, and are judged to express less positive affect and more negative affect via speech. The acoustic properties of speech are also affected by sleep deprivation, with effects on several parameters (McGlinchey et al., 2011). Of 30 acoustic properties studied, there was significant disruption to several properties (e.g. increased jitter, decreased psycho-acoustical bark at specific high frequency energy band, increased shimmer, decreased pauses and decreased high frequency energy). In an earlier study, Harrison and Horne (1997) have reported effects of sleep deprivation on speech. These authors found that sleep deprivation decreased word count on trial 3, with decreased nouns and adjectives on this trial. Sleep deprived subjects also generated a larger proportion of semantically related words at trials 2 and 3. In addition, intonation was found to be affected by sleep deprivation, and was more inappropriate, with voices reported to be more monotonic or flat. There was a corresponding increase in ratings of fatigue.

Taken together these results suggest that speech is affected by sleep deprivation, and that perceivers detect these differences on several measures. Specific parameter differences can also be detected by computerized analysis. Given the importance of vocal expressivity to social interactions, it might be worth investigating how perceivers interact with sleep deprived individuals. The emotional reactions of observers to such expressions might also be worth investigating. This would seem to be relevant to both facial and vocal cues. Axelsson et al. (2010) have found that facial cues of sleep

deprivation are detected by observers, with such images rated as being less healthy and attractive. The social implications of sleep loss are therefore worth investigating in terms of the effects on the perceiver. Also relevant is the concordance between measures of timing and maximum expressivity in a social exchange. This might be relevant to individuals with insomnia disorder (Kyle, Espie, and Morgan, 2010), who are thought to have difficulties with emotion regulation (Harvey, 2002), and there is evidence that voices can be "fatigue-proofed" (Bagnall et al., 2011).

Furthermore, individuals who have been sleep deprived are perceived to be more sad and fatigued, with significant differences in facial cues (Sundelin et al., 2013). The specific facial cues included were redder eyes, hanging eyelids, swollen eyes, darker circles under the eyes, pale skin, more wrinkles/fine lines around the eyes, and more droopy corners of the mouth. This is from images that were taken of unexpressive, or "neutral" faces following 31 hours of sleep deprivation and 5 hours of sleep. Sadness ratings and ratings of fatigue were found to be correlated. Such studies suggest that sleep loss affects appearances and that this can be detected by observers. Future studies might wish to consider the role of familiarity in how faces are assessed. This could also be relevant to insomnia disorder subjects, in whom social interactions are an area of concern (Kyle, Espie, and Morgan, 2010), and sensitivity to these changes may contribute towards the maintenance of insomnia disorder (Harvey, 2002).

Emotional intelligence

Emotional intelligence could also modulate reactions to sleep loss. Killgore, (2013) assessed emotional intelligence, measured subjectively (via the emotional quotient inventory, or EQ-i) and objectively (via the Mayer-Salovey-Caruso emotional intelligence test, or MSCEIT). Subjects reporting < 6.5 hours of sleep the previous night scored significantly lower on subjective emotional intelligence than those subjects who reported > 8 hours of sleep the previous night, with no significant group differences on objectively measured performance. Insomnia complaints were not found to account for the correlation of total sleep time with subjective emotional intelligence. When resting-state fMRI results were analyzed, greater negative functional connectivity was found between the right ventro-medial prefronal cortex and right amygdala, and this was negatively correlated with greater reported total sleep time. The strength of this connectivity correlated with overall subjective emotional intelligence, but effects with objective emotional intelligence were not significant. Furthermore, insomnia complaints appear to account for the correlations between emotional intelligence and functional connectivity (Killgore, 2013). Of particular interest is the failure to find

deficits in objective performance, although there were significant effects on subjective and neuroimaging measures.

In a new approach from the same group, Weber et al. (2013) calculated sleep credit as the difference between habitual sleep time, and the reported minimum amount of sleep necessary before impairments became apparent in ability to work. In the right middle orbitofrontal gyrus and the left gyrus rectus/superior and medial orbitofrontal gyrus, greater grey matter volume was associated with habitual "sleep credit". Total EQ-i scores were found to correlate with the left gyrus rectus and the superior and medial orbitofrontal gyrus. Volume of this area correlated with interpersonal subscale of the EQ-i. Impairments in subjective emotional intelligence following sleep deprivation have also been previously reported by this group (Killgore et al., 2008). Taken together these results suggest significant effects of sleep on subjective emotional intelligence and brain responses, but not on objective measures of emotional intelligence.

ROLE OF (REM) SLEEP IN EMOTIONAL REACTIVITY

There is much evidence that sleep benefits the memory of emotional content, and it has been suggested that REM-sleep plays a role in maintaining an emotional memory, while removing its associated emotionality, or "affective tone" (Walker and Van Der Helm, 2009). We now briefly review the evidence for how sleep affects emotional reactivity.

Baran et al. (2012) report evidence of attenuated arousal and valence ratings of negative emotional images following wakefulness, in contrast to sleep. This effect was associated with greater time in REM sleep. Evidence of more rapid extinction of fear responses have also been reported by Kuriyama, Soshi, and Kim (2010). These authors used movie clips of vehicle accidents or safe driving as stimuli. They report that there were significant differences between sleep and wake groups on the fear ratings of "safe" movies on day three, with lower ratings following sleep deprivation. Sleep deprivation was also associated with a diminished skin conductance response (SCR) to incorrectly identified stimuli in all contexts. However, Van Der Helm et al. (2011) report decreased intense emotional ratings, and increased non-emotional ratings with sleep, with no such effects found in wakefulness. This result was associated with REM sleep. As such the evidence as to how sleep affects emotional reactivity is currently unclear, and further work is needed to clarify this issue, and the role of specific sleep stages and especially REM sleep.

Another approach has been to assess emotional ratings comparing old-new images, with early or late sleep periods. Wagner, Fischer, and Born (2002) found late, REM-rich sleep to enhance old-new valence ratings of images towards more negative ratings, compared to early sleep and wakefulness. The early sleep condition took place for three hours from around 23:00 to 02:00, and had significantly greater slow-wave sleep than late sleep. Late sleep took place from 03:00 to 06:00, with significantly greater REM sleep. An additional experiment found similar effects with a full night of sleep. Significant differences were found between these sleep conditions on the emotion ratings of images before and after sleep (i.e. emotional habituation/sensitization). Images were rated more positively after early sleep and more negatively after late sleep, with no such effects on arousal ratings (Wagner, Fischer and Born, 2002).

Emotional ratings of old-new images have also been studied in conjunction with partial sleep deprivation. Emotional adaptation has been investigated via REM sleep deprivation (Lara-Carrasco et al., 2009). Lara-Carrasco et al. found REM sleep deprivation to be linked to greater emotional adaptation towards images, on subjective ratings of arousal. These results suggest that emotional adaptation/habituation is not benefited by REM sleep (Wagner, Fischer and Born, 2002; Lara-Carrasco et al., 2009). Partial sleep deprivation has also been investigated with other tasks. Rosales-Lagarde et al. (2012) report that REM sleep deprivation enhances brain reactivity to emotion, in a task whereby participants were asked to imagine themselves within an emotional scene shown on screen. These results indicate the need for further research to clarify the role of REM sleep in emotional reactivity. However, REM-sleep deprivation paradigms may produce results which are difficult to translate to everyday life. In this regard we prefer the early/late sleep deprivation paradigm of Wagner, Fischer and Born (2002).

Nap paradigms have also been used to investigate emotional responding. Gujar et al. (2011) report a role of REM sleep in diminishing emotional reactivity following a nap, when subjects are asked to rate the emotional intensity of a face displaying varied emotional intensities, shown in emotion blocks (anger, fear, happy, sad). In this task, participants who did not nap showed increased reactivity to these emotional expressions, which was reversed by napping and in particular by REM sleep. Napping could also aid habituation towards emotional images when physiological reactivity is assessed (Pace-Schott et al., 2011). Pace-Schott et al. used negative and neutral images, and assessed responses towards repeated and novel image sets. These authors report evidence of habituation (skin conductance response) in the nap group to repeated stimuli, a result which was not found in the wake group. This was found for negative stimuli. There was also significant inter-session habituation in the wake group,

but not the nap group, on heart-rate deceleration and corrugator electromyogram (EMG) responses. EMG responses in the wake group were found to sensitize, and these results were found for both negative and neutral stimuli. In general, results suggest a role of REM sleep in emotional responding. However the precise role of REM sleep in emotional reactivity clearly warrants future attention.

EFFECTS OF POOR SLEEP QUALITY & INSOMNIA

In the following section, we describe those studies which have investigated the links of poor sleep quality and insomnia with socio-emotional functioning. All insomnia subjects were tested outside of the context of any treatment study.

Neuroimaging studies

Prather, Bogdan and Hariri (2013) used the Pittsburgh sleep quality index (PSQI; Buysse et al., 1989) to obtain a subjective evaluation of sleep quality, and investigated its links with brain activity. This was done via a matching task with emotion faces; a task previously found to engage the amygdala. They found that poor sleepers, defined on the basis of PSQI scores, showed significant relationships of amygdala activity with measures of psychopathology (depression, stress, and anxiety). These results were not found in normal sleepers. Such results add support to the relationships of sleep and mental health (Benca et al., 1992; Baglioni et al., 2011), and suggest that poor sleep quality affects the relationships of brain activity with indicators of psychological distress. Franzen, Siegle and Buysse (2008) have previously found sleep deprivation to alter the relationships between various subjective and objective measures of sleep and emotion, and the process by which emotional dysregulation occurs could be different in normal sleepers and poor sleepers. Interestingly, the precise task demands can affect how stimuli are processed. In particular, selecting emotion labels has been described as a cognitive task, with evidence of top-down effects on the emotional brain, whereas matching faces has been described as perceptual (Hariri, Bookheimer, and Mazziotta, 2000; Lieberman et al., 2007). Such tasks could be useful in order to assess emotional regulation and sleep.

Another type of paradigm is to investigate emotion regulation strategies by asking participants to regulate their reactions to emotional information. Minkel et al. (2012) asked subjects to view negative or neutral emotional images, and to either maintain attention, or to use cognitive reappraisal to reduce their emotional reaction. In a sample drawn from the general population, subjective sleep quality, as measured by the

PSQI, was not found to be associated with amygdala activation during emotion regulation. However, the use of sleep medication was associated with less activation in a medial prefrontal cortex area, with no effects of other PSQI subcomponents. Future studies might wish to employ this paradigm with normal sleepers and varying levels of sleep deprivation, in order to assess changes in the processes by which emotional regulation occurs.

Baglioni et al. (2014) have also recently studied the effects of sleep-related and emotional stimuli on amygdala activity in insomnia disorder. These authors report that at the first presentation of stimuli, healthy controls show increased amygdala activation to negative stimuli compared to neutral stimuli with similar arousal levels. There were no such differences in insomnia disorder. When insomnia-relevant stimuli were shown, insomnia subjects showed increased amygdala activation to disorder-related stimuli compared to the non-insomnia stimuli. The opposite pattern was found in healthy controls. When neutral moderate, negative moderate and sleep-related negative stimuli were presented to healthy good sleepers, the amygdala activation levels were comparable between the first and second presentations. However, insomnia participants showed increased amygdala activation levels at the second presentation of neutral moderate images. These subjects also showed less amygdala activation at the second presentation of negative moderate and sleep-related stimuli.

Physiological studies

Emotional reactions have also been investigated in insomnia participants with multiple measures of emotion. Baglioni et al. (2010) presented subjects with positive sleep, negative sleep, positive, negative, and neutral images, and measured their responses using physiological measures (EMG, cardiac vagal tone and heart-rate) and behavioral ratings. These authors report evidence for emotional reactivity in a group of primary insomnia subjects. People with insomnia showed decreased tonic activity of the corrugator muscles in response to sleep-positive stimuli, unlike controls. There was also evidence of increased tonic activity of the zygomatic muscle to all stimuli in those with insomnia. Cardiac vagal tone was also increased to all stimuli in insomnia. The valence ratings of negative and negative sleep-related images in insomnia were not significantly different, unlike controls. Similar results were found with arousal ratings, and negative sleep-related stimuli were rated as more arousing than positive sleep-related stimuli, which was also unlike controls.

These results suggest that in insomnia there was less activation of the "frowning" muscles to positive sleep-related stimuli, and increased activation of the "smiling" muscles to all stimuli. Insomnia participants rated sleep-related negative stimuli and negative stimuli similarly on valence, and rated positive sleep-related stimuli as less arousing than negative sleep-related stimuli. All stimuli were linked to increased cardiac vagal tone in insomnia subjects. These results may be interpreted in terms of an increased negative emotional reaction to positive sleep-related emotional stimuli in normal sleepers, and similar arousal ratings of sleep positive and sleep negative stimuli. Normal sleepers also evidenced less positive emotional responses to all five emotional image categories. The valence ratings of negative and sleep-negative stimuli were significantly different in normal sleepers. Normal sleepers also responded with decreased cardiac vagal tone to all stimuli, unlike insomnia subjects. These results suggest systematic differences between normal sleepers and insomnia subjects across multiple levels of responsivity: subjective, cardiac, and EMG.

A second study has also investigated emotional responses in insomnia, via physiological measures. Evidence of lowered subjective ratings of valence was found in response to emotional images in insomnia participants, who also showed evidence of impaired ability to regulate emotion (DelVentura et al., 2014). Specifically, participants were shown images of mutilation, erotica, or neutral images, and their emotional reactions were assessed via behavioral ratings and physiological measures. The relationships between pain ratings and a physiological measure of the pain response (nociceptive pain reflex, or NPR) were diminished in insomnia subjects. Insomnia appears to disrupt the relationships between physiological measures, subjective measures, and responses to stimuli. Future studies might wish to consider the point at which these relationships become dysregulated as insomnia disorder develops.

Behavioral studies

There is also recent evidence that insomnia affects the subjective ratings of emotional stimuli. Kyle et al. (2014), using emotional face stimuli, found evidence that the subjective intensity ratings of fear and sadness were blunted in insomnia subjects. The categorization judgments of angry, sad, happy, and fearful faces were unaffected. In this study emotional faces were presented in a random order, and displayed until response. Emotional intensity judgments were not found to correlate with sleep diary responses, measures of sleepiness, or daytime functioning. However, there were significant negative associations of intensity judgments with anxiety and depression scores in insomnia subjects. In these subjects anxiety correlated negatively with anger,

happiness and overall judgments. Overall intensity judgments and sadness ratings were negatively correlated in this group. In healthy controls, sadness ratings were linked to anxiety. As such the perception of emotion from faces appears to become linked to psychological distress, whereas normal sleepers fail to show such a pattern. This could be relevant to the results of Kyle, Espie and Morgan (2010), who found that insomnia subjects reported concerns regarding social interactions.

SUMMARY

Results from neuroimaging studies suggest that sleep loss affects the processing of emotion, with similar effects found with positive (Gujar et al., 2011) and negative (Yoo et al., 2007) stimuli. These results are suggestive of both increased reactivity, and altered connectivity. Sleep loss also seems to amplify anticipatory activity, towards all cues (Goldstein et al., 2013) or when expecting negative emotional stimuli (Franzen et al., 2009), and this discrepancy could depend on the measure of emotion (fMRI/pupillography) and/or task. Similar neural effects of sleep deprivation are reported by Chuah et al. (2010). These authors report that increased emotional distraction with sleep deprivation is linked to impaired prefrontal-amygdala connectivity. These results could be in keeping with behavioral evidence of impaired inhibition following sleep deprivation (Anderson and Platten, 2011). Furthermore, emotional images could be associated with greater subjective emotion and more sustained neural processing, suggesting sustained patterns of activity (Britton et al., 2006). However, an important issue with these neuroimaging studies relates to the relative nature of the reported results. In particular, results are contrasted relative to another condition or resting state activity, and as such, the responses to the initial condition could result in the appearance of positive or negative results. However, results appear consistent with regard to effects of sleep deprivation on neural activity, across different types and durations of sleep loss.

The effects of sleep loss on behavioral ratings are less consistent. With emotional images, Chuah et al. (2010) and Franzen et al. (2009) found no effects of sleep deprivation on emotional ratings, although effects have been reported by Tempesta et al. (2010) on neutral images. Yoo et al. (2007) and Gujar et al. (2011) also report evidence of effects on emotional ratings with sleep deprivation. The reasons for these discrepant results appear unclear, although possible explanations may be related to the images presented, features of their presentation, or the rating scale which was employed. Also relevant could be characteristics of the participant group. Working memory (Chuah et al., 2010) and trait anxiety (Goldstein et al., 2013) have both been

identified as factors which modify the effects of sleep loss on neural responses. Future studies should investigate the reasons for these different reactions to emotional images in greater depth.

With emotional faces, Huck et al. (2008) reported no effects on basic emotion recognition, and Motomura et al. (2013) found no effects with button responses to target stimuli. However, Huck et al. (2008) reported effects of sleep loss on complex emotion recognition, and Van Der Helm, Gujar, and Walker (2010) found impairments in intensity ratings of subtle, mid-intensity ambiguous, emotional images. Pallesen et al. (2004) also reported lower accuracy and increased reaction times, in a matching task with schematic images. Using schematic images, Dushenko and Sterman (1984) also found impaired facial recognition performance. However, neither of these two studies or Huck et al. (2008) reported effects on specific emotions. Sleep loss seems to impair the processing of emotion from faces, and this effect seems most apparent with more complex tasks. Different cognitive processes could also be involved in these different tasks. Specifically, perceptual processes could be more involved in face matching, whereas cognitive processes could be more involved in emotional labeling, a task which has also been linked to emotional regulation processes (Hariri, Bookheimer, and Mazziotta, 2000; Lieberman et al., 2007). Emotional intensity ratings have also been studied less, and may follow on from categorization decisions (Utama et al., 2009). We suggest that future studies should focus on identifying sensitive and ecologically valid tasks of facial emotion recognition, taking account of the social implications of such tasks.

Evidence of impaired performance on emotional face tasks appears consistent with studies of expressed emotion, and with self-reported decreases in emotional intelligence and interpersonal functioning following sleep deprivation (Franzen, Siegle and Buysse, 2008). Minkel et al. (2011) found that sleep deprived subjects were less facially expressive in response to emotional stimuli, and Schwarz et al. (2013) found evidence of slower facial responses with sleep loss. Similarly, McGlinchey et al. (2011) report that sleep deprived subjects were less vocally expressive, with similar results reported by Harrison and Horne, (1997). These results seem consistent with reports of impaired subjective emotional intelligence following sleep loss (Killgore et al., 2008). However, objective measures of emotional intelligence have been found not to be affected by sleep loss, despite significant effects on subjective emotional intelligence and neural reactivity (Killgore, 2013). As such, sleep loss seems to impair performance on tasks related to social functioning, on subjective measures, performance measures, and expressivity measures. Importantly, these tasks could also be tapping in to

emotional processes. The relationships of emotion generation with emotional regulation are disputed (Gross, Sheppes and Urry, 2011; Gross and Barrett, 2011), and expressivity could both be a consequence of emotion (Buck, 1994; Dimberg, 1990), and contribute towards emotional regulation (Dimberg, 1990; Izard, 1990). Separating these processes out appears particularly relevant for insomnia disorder, due to its links with emotion regulation (Harvey, 2002).

With regard to insomnia, there is evidence of impaired emotional regulation (DelVentura et al., 2014), and increased emotional responsiveness (Baglioni et al., 2010), when multiple physiological measures are assessed. With socially-relevant stimuli, however, insomnia subjects make lower ratings of intensity in faces (Kyle et al., 2014), with comparable results with emotional images (Delventura et al., 2014). Evidence of altered brain activity has also been reported in conjunction with sleeping medication use and emotional regulation (Minkel et al., 2012). In poor sleepers, the relationships of amygdala activity with measures of psychopathology are also disrupted (Prather, Bogdan and Hariri, 2013), with similar results recently reported in insomnia disorder (Baglioni et al., 2014). Future studies should consider the trajectory of how emotional reactivity is altered across different phases of insomnia disorder (e.g. predisposing factors, acute, and chronic insomnia) and with different comorbidities.

LIMITATIONS

Task characteristics & demands

The studies of Yoo et al. (2007) and Gujar et al. (2011) have displayed stimuli in an increasingly emotional gradient, based on normative data, and this may exaggerate the effects of sleep deprivation. In particular, such a presentation would be expected to prohibit any return to an emotional baseline (Landis, 1924), which could itself be negatively affected by sleep loss. This could lead to more extreme emotional states which are increasingly difficult to regulate (Green and Malhi, 2006), and makes it difficult to specify whether the processes involved in emotion generation or emotion regulation are contributing towards results.

While the relationships between these processes are complex and debated (Gross and Barrett, 2011), it seems important to attempt to identify the stage(s) at which sleep deprivation affects emotions. Different task instructions could be used to assess this. Emotional labeling could engage cognitive processes, whereas a matching task may involve perceptual processes (Lieberman et al., 2007), and subjects have been asked to

modulate their emotional reactions via specific regulatory strategies (Minkel et al., 2012).

A further limitation may relate to the way in which facial stimuli are created. Ekman and Friesen (1976) have identified facial action units which are responsible for expressing an emotion, and physiological constraints are important to how expressions are formed and displayed in real life (Adolphs, 2002; Ekman and Friesen, 1969). Importantly, such faces could increase perceptual difficulty (Cote et al., 2014). Landis (1924) suggests the importance of distinguishing between emotional expressions and social expressions, and the social context in which testing took place could also be relevant to emotional expressivity (Schmidt, Cohn and Tian, 2003).

Social effects

The socio-communicative role of emotion is important, and this modulates the expression of emotional displays (Buck, 1994; Ekman and Friesen, 1969). Emotion perception involves stimulus appraisal, emotion regulation, and affective states (Phillips et al., 2003), and these brain systems overlap with those involved in face processing (Adolphs, 2002). The social relevance of tasks is therefore important to consider, and this seems especially important when expressivity is assessed, or when socially-relevant stimuli are used. Furthermore, the correspondence between self-reported mood and emotional task performance, and their associated neurological and physiological correlates, are likely complex. Indeed, the relationships between objective and subjective measures of emotion may be affected by sleep deprivation (Franzen, Siegle and Buysse, 2008), suggesting benefits of assessing subjects using multiple measurements, and the importance of the social context.

In support of this, altered perception of facial emotions is reported in insomnia and with sleep deprivation (Van Der Helm, 2010; Kyle et al., 2014). Sleep loss is known to alter the stress system (Meerlo et al., 2008), and insomnia has been associated with hyperarousal (Bonnet and Arand, 2010; Riemann et al., 2014). As such, evidence of hypervigilance or improved performance may be anticipated, although expressed emotion is also reportedly “blunted” with sleep loss (Minkel et al., 2011; McGlinchey et al., 2011) and with evidence of slower facial movements (Schwartz et al., 2013). The relationships between emotion perception, emotional expressivity, and emotional experiences also seem to be important to investigate further. For example, Kyle et al. (2014) reported differences between normal sleepers and insomnia participants on the associations between emotional intensity ratings and psychological distress. This could

occur as a result of the chronic effects of sleep loss (Meerlo et al., 2008) or pre-existing differences in emotions between groups.

FUTURE DIRECTIONS

Four avenues of future research seem most pertinent, related to 1) diverse measures of emotional functioning, 2) multi-faceted tests of social functioning, 3) role of sleep stages and circadian effects, and 4) inter-individual vulnerabilities of sleep and emotion.

Diverse measures of emotional functioning

Measured emotion

Emotion is complex, and single measures may provide an incomplete picture of emotional functioning. Self-reported measures can be used to indicate subjective emotion; behavioral tasks can be used to assess objective performance; and physiological and neuro-imaging measures can provide information as to the neural, physiological and temporal mechanisms of emotional processing and experience (see Table 4.). The inter-relationships of these measures could also be complex, and this is affected by sleep loss (Franzen, Siegle and Buysse, 2008). Furthermore, emotional processing could itself comprise several components, and differences may emerge at different stages. Emotion seems to comprise several elements, such as reactivity to emotion, the duration of emotional impact, and tendency to express emotion (Landis, 1924). To investigate these, experimental tasks could include manipulating the stimulus presentation time and inter-stimulus interval, as well as investigating the effects of feedback on performance and anticipatory effects, and subjective performance. The relationships between different types of measures also appear to be important to consider.

Table 3. Summary of relevant methods.

Measures	Processes
Questionnaires	Subjective emotional experiences, emotional regulation and intelligence, meta-emotion and meta-cognition, subjective social functioning.
Observed behavior	Facial expressivity, generation of social cues, reactions to social cues, social interactions.
Behavioral tasks	Objective measures of task performance, with accuracy and reaction times indicators of underlying cognitive processes.
Physiological responses	EMG, ECG measures derived from heart-rate, skin-conductance response, and measures of ANS/PNS activity.
Neuro-imaging techniques	EEG and ERPs, fMRI, PET can be used to measure recruitment of brain areas and activity, and connectivity between them.

Abbreviations: autonomic nervous system (ANS), electroencephalogram (EEG), electrocardiogram (ECG), electromyogram (EMG), event related potentials (ERPs), functional magnetic resonance imaging (fMRI), peripheral nervous system (PNS), positron emission tomography (PET).

Emotional context

The emotional context of facial expressions also affects their processing (Righart and de Gelder, 2008; Aviezer et al., 2008) and there is evidence that state anxiety is associated with a greater use of contextual cues (Blanchette, Richards, and Cross, 2006). As such, the situational context could exert additional effects on emotional processing following sleep loss. Furthermore, mood is thought to become more independent of the situation with increasing illness severity (Beck, 1971), and sleep could contribute to this (Bryant et al., 2010). The contribution of context to emotion has been investigated in depression, and insomnia and depression are linked (Baglioni et al., 2011). Blysma, Morris and Rottenberg (2008) have performed a meta-analysis of emotional reactivity (emotional context insensitivity; ECI) in major depressive disorder, finding that depressed individuals show diminished responses to both positive and negatively valenced stimuli. This was discussed as insensitivity towards the emotional context within this disorder. Beck (1971) has previously discussed the correspondence between a situation and mood states relevant to psychological disorders, suggesting a greater role of internal cognitive processes with psychological disorders. The precise context of an experiment is also important to consider when investigating socio-emotional functioning, although this may become less relevant with specific patient groups such as major depressive disorder (Blysma, Morris and Rottenberg, 2008).

Multi-faceted tests of social functioning

Social "building blocks"

Social functioning is complex, and several processes contribute towards socially-competent behavior (Iarocci, Yager and Elfers, 2007). Specifically, social interactions involve the comprehension of social cues, such as expressed behavior and speech, in conjunction with knowledge about a person and situation. Gaze (Ewbank, Fox and Calder, 2010; Bindemann, Burton and Langton, 2008), body cues (Aviezer, Trope, and Todorov, 2012), and voices (Yovel and Belin, 2013) are all relevant social signals. These different signals influence each other, and emotional faces showing direct gaze are processed less efficiently (Bindemann, Burton and Langton, 2008). The way in which this integration occurs can be affected by individual factors, such as anxiety (Ewbank, Fox and Calder, 2010). Social competence includes face recognition as well as emotion recognition (Iarocci, Yager and Elfers, 2007), and the changeable aspects of a face (such as movements) and its invariant aspects (e.g. identity) are both important to face perception (Haxby, Hoffman and Gobbini, 2000), and contribute towards the social context.

Related to this, information about an individual's identity is thought to be somewhat independent of emotion (Calder and Young, 2005; Vuilleumier and Pourtois, 2007). Importantly, "neutral" faces are arguably not unemotional. In particular, unexpressive faces are judged on two dimensions, namely dominance, and trustworthiness or valence, and these are linked to trait judgments (Oosterhof and Todorov, 2008). Familiarity of faces also affects the perception of emotion (Dobel et al., 2008), and familiar and unfamiliar faces seem to be recognized qualitatively differently (Johnston and Edmonds, 2009). Such basic sensory/perceptual, cognitive, and emotional processes are thought to lead to the development of the higher-order capabilities which are involved in social functioning (Iarocci, Yager and Elfers, 2007) – for example, emotional contagion is a pre-requisite of empathy (Frith and Frith, 2012). As such, future research aimed at these "building blocks" could help to identify the point at which emotional impairments appear.

Empathy & social interactions

Importantly, emotional behavior can serve social motives (Buck, 1994), and reflect emotional states (Gerber et al., 2008). Such social motives may contribute towards the

additional processes that are involved in social competence, such as theory of mind (Iarocci, Yager and Elfers, 2007), which has also been called cognitive empathy (Davis, 1980). Theory of mind, or "mentalizing", is a form of meta-cognition (Frith and Frith, 2008), and is thought to be an explicit process (Amodio and Frith, 2006; Zaki and Ochsner, 2012) which engages the neural systems involved in self-projection (Singer, 2012). Implicit processes which are involved in social cognition include prejudice and perspective taking (i.e. tracking the mental states of others). Such processes are relevant to social emotions, like compassion and embarrassment, and social decision making, which includes fairness, trust, and Schadenfreude (Singer, 2012). Indeed, performance on social decision-making tasks has been found to be affected by sleep deprivation (Anderson and Dickinson, 2012; Libedinsky et al., 2011; Killgore et al., 2007).

Context also contributes towards social behavior, and adaptive socially competent behavior is the result of the match between genotype and the environment (Iaroci, Yager and Elfers, 2007). The social environment might mitigate the effects of sleep loss. Trait factors such as extraversion-introversion, in interaction with the social context, have been linked to altered performance following sleep deprivation (Rupp, Killgore and Balkin, 2010). It is also important to consider the effect of sleep loss on dynamic social exchanges. For example, people who have been sleep deprived are judged to look less healthy and attractive (Axelsson et al., 2010), and such trait judgments correlate with perceived trustworthiness (Todorov, 2008). As such, sleep loss could affect the sleep-deprived person's ability to interpret social information. It could also affect the perceptions made of the sleep deprived person, and behavior towards them. Such factors may be important in the development of insomnia disorder.

Sleep stages & circadian effects

Sleep stages

The role of specific sleep stages, especially REM sleep, in emotional reactivity is of particular interest, and this sleep stage has been implicated in psychiatric disorders (Vanderkerckhove and Cluydts, 2010; Walker, 2009), such as depression (Vanderkerckhove and Cluydts, 2010; Walker and Van Der Helm, 2009). The "sleep to remember, sleep to forget" hypothesis posits that with sleep an emotional memory is strengthened while its associated emotional effects are weakened, and REM sleep is crucial to this (Walker and Van Der Helm, 2009). This process is thought to ameliorate any hyperarousal, and its disruption may contribute towards depression and post-

traumatic stress disorder (Walker and Van Der Helm, 2009). However, studies on how sleep relates to emotionality appear contradictory. Discrepancies in methods relating to sleep (early/late sleep restriction, REM sleep deprivation and restriction, nap paradigms), emotion (e.g. adaptation, old/new contrasts), and measurements (valence and arousal ratings, experimental tasks, neuroimaging, physiological) likely contribute towards these effects. Studies with combined methods and measures would help to clarify the role of REM sleep in emotional reactivity, as would studies investigating changes in emotionality over time, as well as the role of the different sleep phases.

Circadian effects

Few studies have investigated the role of circadian processes on emotional tasks. While sleep loss and sleep disruption have been found to affect emotion perception, the circadian system could also exert effects. Five studies, to our knowledge, have looked at how the circadian system affects emotionality in healthy subjects using objective measures of emotion. Circadian effects have been investigated via measures of diurnal preference, with Paradee et al. (2008) reporting a significant effect of chronotype-congruent test times in overall emotion recognition performance among a group of rehabilitation patients. Other researchers have investigated the role of light in emotion perception, and blue light, which plays an important role in the circadian system, has been found to affect how emotional information is processed (Vandewalle et al., 2010).

In another approach, emotionality has been assessed at different times of day. Hot, Leconte and Sequeira (2005), presented subjects with images of neutral scenes and high arousal negatively-valenced scenes, and recorded the skin conductance response. In a within-subjects design with seven different test times, at 2-hour intervals between 09:00 and 21:30, they found a time of day effect on the skin conductance response to aversive images. Specifically, they found a significant linear trend to unpleasant images, and a significant quadratic trend to neutral images, across the day. Hasler et al. (2008) have also investigated how emotional behavior varies across the day via the sampling of ambient noise. These authors report a significant diurnal effect on behaviors associated with positive affect, namely socializing, laughing, and singing. No such effects were found with arguing or sighing, suggesting that behaviors linked to negative emotion show no diurnal patterns. Similarly, Golder and Macy (2011) found effects of time of day, day of the week, and season in the affective content of twitter messages. These studies suggest the importance of controlling for time of day when investigating the role of sleep in socio-emotional interactions.

Inter-individual vulnerability in sleep & emotion

Sleep loss & emotion

Individuals vary in their response to sleep loss, and in how different types of measures are affected (Leproult et al., 2003). These factors could be somewhat dissociable (Leproult et al., 2003), suggesting that perceived impairments may not correlate with objective performance deficits, and the rate of sleep loss could also affect this (Drake et al., 2001). The dose-response effects of sleep loss are also important to consider, and the effects of different kinds and durations of sleep loss. Individual differences in vulnerability to sleep loss seem important to investigate in conjunction with socio-emotional tasks, as this could contribute towards the effects of sleep loss on functioning. Baseline cognitive abilities, such as working memory, could also mediate the effects of sleep loss (Chuah et al., 2010). In addition, individuals also vary in aspects of emotion (Ekmna, 1992; Landis, 1924), and social behavior is similarly complex (Iarocci, Yager and Elfers, 2007). In particular, trait anxiety has been reported to magnify the emotion effects of sleep disruption (Goldstein et al., 2013). As individual differences related to the effects of sleep loss interact with tasks to affect performance, a detailed understanding of these relationships would be useful in order to predict the effects of sleep loss.

CONCLUSIONS

Sleep loss affects socio-emotional functioning in several ways. Emotional reactivity to emotional stimuli appears to be enhanced with sleep deprivation, with results most obvious in neuro-imaging studies. Tasks of behavioural inhibition are also sensitive to sleep deprivation. Sleep loss impairs social functioning, with results most apparent in measures of subjective emotional intelligence, and with some tasks of emotional face recognition. Emotional expressivity also seems to be impaired with sleep loss, and this has clear implications for social functioning. In general, the relationships between sleep, emotion, and social functioning are complex, and the specific outcome measures (e.g. subjective/objective, behavioral, physiological, brain) could also affect reported results. Future studies should include multiple measures of emotion, and consider the inter-relationships between tasks, stimuli and measures. Consideration of the social context, and individual differences in emotion, social functioning, and sleep are also important. In addition, the role of (recovery) sleep, sleep stages, and circadian processes require clarification. As sleep affects socio-emotional functioning, and is associated with psychopathology, understanding the links of socio-emotional functioning and task performance with sleep is an important avenue of future research.

Figure 2. Key practice points from the systematic review

Practice points

- Sleep deprivation increases emotional reactivity, which is most apparent with fMRI results.
- Sleep deprivation impairs social functioning, measured by emotional expressivity parameters, and behavioral performance with emotional faces.
- Sleep-related daytime functioning, emotion, and social interactions are interlinked.
- Emotional tasks comprise different component processes, and multiple measures of performance are optimal.
- The social context of emotion tasks is as important as the emotional context.

Figure 3. Research agenda from the systematic review

Research agenda

- Include multiple measures of emotional responding, across domains.
- Consider the effects of the social context of experimental studies.
- Compare results of tasks measuring different components of socio-emotional functioning.
- Clarify the role of sleep stages, especially REM sleep, in emotional phenomenology.
- Investigate the effects of the circadian system in socio-emotional functioning.
- Assess the role of individual differences in sleep, emotion, and social functioning, in task performance.

The following chapters in this thesis address the role of sleep in socio-emotional task performance. This was done by looking at the sleep of normal sleepers, poor sleepers, and people with insomnia. Emotional faces were chosen as stimuli and task performance was assessed via behavioural measures of accuracy and reaction times. As a result of this review it was hypothesised that socio-emotional task performance would be impaired in poor sleepers and people with insomnia disorder. We also assessed how emotion recognition performance changes across the day in normal sleepers, in order to investigate variability within a healthy sample. The final experimental chapter in this thesis extended these results to theory of mind tasks.

Chapter Three

Sleep disruption, emotion perception, and empathy: An exploratory study

Abstract

Sleep disruption is common in the general population, and especially in students. In general, sleep disruption and insomnia are associated with adverse daytime effects on social interactions and mood. The purpose of the current study was to assess the effects of PSQI-derived sleep groups on sleep and mood, emotion perception, and empathy in a student population. These results were then used to inform subsequent studies within this thesis. In total 56 healthy subjects completed measures of sleep, mood, and empathy, as well as an emotion recognition task. Sleep data were gathered on a continuum basis, and sleep groups were formed on the basis of questionnaire cut-offs. There were significant effects of sleep group, and significant differences were found with regards to mood and sleep diary measures of sleep disruption. Results suggest that the previous night's sleep was significant predictor of happiness recognition, in both accuracy and reaction time measures. Depression scores also contributed towards happiness recognition. There were no significant effects of sleep group or predictor variables in measures of empathy. As a consequence of this study, future studies employed more sensitive measures of emotion face recognition, and targeted recruitment towards normal sleepers and people with insomnia.

Sleep disruption, emotion perception, and empathy: An exploratory study

Introduction

Sleep disruption is common in the general population, with around 30% of people displaying insomnia symptoms in a year (LeBlanc et al., 2009). When insomnia is defined more stringently, around 7% meet criteria for insomnia (LeBlanc et al., 2009). Sleep disruption is also high among students, 32% of whom are unable to fall asleep within 30 minutes at least once a week, and this is most commonly attributed to stress (35%) or ambient noise (33%; Lund et al., 2010). In practice, the Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989) and the Insomnia Severity Index (ISI; Bastien, Vallieres, and Morin, 2001) are commonly used measures to assess sleep quality, and define sleep groups (e.g. Jones et al., 2005; Ree, Pollitt, and Harvey, 2006). When the PSQI is used among a student population, 34% score within the normal range, with a majority of students experiencing poor sleep (Lund et al., 2010). Sleep diaries provide information about the daily timing of sleep, as well as measures of sleep continuity (e.g. wake after sleep onset) and its qualitative experience, and are regarded as the "gold-standard" in measuring subjective sleep experience (Carney et al., 2012). Sleep diary measures of sleep onset latency and sleep duration correlate highly with PSQI derived estimates (Backhaus et al., 2002). Sleep quality, assessed by the PSQI, and chronotype, assessed by the moringness-eveningness questionnaire (MEQ), have been found to be linked (Barclay et al., 2010).

Sleep disruption is also linked to negative mood states (Baglioni et al., 2010; Lund et al., 2010), and people with insomnia have identified social interactions as an area of impaired functioning (Kyle, Espie, and Morgan, 2010). Emotion face perception involves emotional brain areas (Phillips et al., 2003), and the recognition of emotional faces has been studied with regard to psychiatric disorders (Phillips et al., 2003; Gilboa-Schechtman, Erhard-Weiss, and Jeczmien, 2002; Joormann and Gotlib, 2006; LeMoult et al., 2009; Langenecker et al., 2005; Beevers et al., 2009; Surguladze et al., 2004). Furthermore, emotional faces are recognized with a high degree of accuracy, and curtailing stimuli presentation times may be a way of increasing task sensitivity (c.f. Calvo and Lundqvist, 2008).

Due to the novelty of research investigating the links between sleep and emotion, this exploratory study was conducted in order to investigate the sensitivity of measures and tasks within the student population. In the current study, participants completed measures of sleep, mood, and empathy, and a task of emotion perception. The aims of

the current study were to assess the links between sleep assessments and their effects on emotion recognition performance, in order to inform subsequent studies in this thesis. We also aimed to assess the effects of sleep groups on emotion perception and empathy. Participants in the current study were predominately recruited from the student population, where experiences of sleep disruption are common (Lund et al., 2010). These subjects completed the questionnaire measures and emotion task and where assigned to sleep groups retrospectively, with measures gathered on a continuum basis. This allowed for the testing of exploratory regression models with regard to emotion recognition performance and empathy. Furthermore, these results could be used to inform future studies.

Methods

Participants

In total 56 participants were recruited from around campus at the University of Glasgow, and took part in exchange for course credit or £6. These participants were recruited via convenience sampling of student forums and snowball techniques. This study was in accordance with the University of Glasgow, School of Psychology Ethics Committee, and all subjects provided written consent. Subjects ranged in age from 16 to 61, with a mean age of 22.12 (*SD* = 8.07). There were 35 females and 21 males, and no subjects were currently receiving treatment for a psychiatric disorder or mental condition.

Measures

- Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989).
- Insomnia Severity Index (ISI; Bastien et al., 2001).
- Morningness-Eveningness Questionnaire (MEQ; Horne and Ostberg, 1976).
- Sleep diaries for the previous nights' sleep (Morin and Espie, 2004).
- The Hospital Anxiety and Depression Scale (HADS; Zigmond and Snaith, 1983).
- Interpersonal Reactivity Index (IRI; Davis, 1980).

Further information as to these measures can be found in Appendix 1.

Stimuli

Four male and four female identities were selected from the Ekman and Friesen (1976) stimuli set, and their corresponding emotion displays (anger, fear, happy, surprise, disgust, and sad) were selected, creating 144 trials. These greyscale faces were presented at average dimensions of 6.43 x 9.04 cm.

Figure 4. Example facial stimuli

Procedure

To familiarize participants with the task response keys, participants initially completed practice trials. In these trials 6 emotion words (sad, happy, fear, disgust, surprise, and anger) were presented for 50 ms and followed by a blank black screen. Words were presented following a fixation cross lasting 750 ms, and each was repeated 5 times in total. Participants were asked to respond as quickly and accurately as possible. If an incorrect response was made, participants were asked to respond with the correct key to continue. The following keys were used: ‘s’ (sad), ‘d’ (happy), ‘f’ (fear), ‘j’ (disgust), ‘k’ (surprise), and ‘l’ (anger), and were labelled with ‘Sa’, ‘H’, ‘F’, ‘D’, ‘Su’ and ‘A’. Words were presented as black text within a white box, at a max height of 0.5 cm.

Following these practice trials, participants completed the emotion task. Emotion faces were presented for 50 ms on a black background followed by a blank screen, and

preceded by a fixation cross of 750 ms. Participants were told to identify the emotion shown as sad, happy, fear, disgust, surprise, and anger, by using the labelled keys, and were instructed to respond as quickly and accurately as possible. The presentation of the face set was randomized, and repeated three times, with two breaks in total. Faces were presented at a viewing distance of approximately 60 cm on a Dell laptop, and the experiment was created within the SuperLab 4.0 program. Data in this pilot study were analyzed using Analysis of Variance (ANOVA), as in practice this is a robust test, and violations have relatively minor effects (Howell, 2002). Effect sizes may be found in Appendix 2.

Results

Identification of insomnia

In a first step, PSQI scores were used to create two sleep groups, with 35 normal sleepers *vs.* 21 poor sleepers. Sleep groups were found to differ significantly in ISI scores (T (54) = - 6.52, $p < 0.001$), with normal sleepers scoring significantly lower. Groups also differed significantly in general distress (HADS-A, t = - 3.50, p = 0.001; HADS-D, t = - 3.52, p = 0.001). However, there were no significant group differences in diurnal preference (t (54) = 0.80, p = 0.43).

The Pittsburgh Sleep Quality Index and the Insomnia Severity Index were then correlated, and these measures were significantly highly correlated (*rho* = 0.71, $p <$ 0.001). Both measures also correlated significant with anxiety (ISI, *rho* = 0.44, p = 0.001; PSQI, *rho* = 0.41, p = 0.002) and depression (ISI, *rho* = 0.44, p = 0.001; PSQI, *rho* = 0.34, $p < 0.01$). Neither sleep measure correlated with diurnal preference (ISI, *rho* = - 0.07, p = 0.62; PSQI, *rho* = - 0.17, p = 0.20).

Table 4. Sleep diary results

	Percentile 05	Percentile 25	Median	Percentile 75	Percentile 95	Mean	Std Dev
SOL (mins)	.00	5.00	10.00	20.00	75.00	20.66	31.34
WASO (mins)	.00	.00	.00	9.50	45.00	6.73	13.76
WAKE (#)	.00	.00	.00	1.00	4.00	1.44	5.36
TST (hours)	4.00	5.54	6.63	7.63	9.00	6.63	1.40
TIB (hours)	5.25	6.79	7.50	8.35	9.75	7.57	1.28
SE (%)	52.63	85.27	94.65	97.74	100.30	89.08	14.42
REST (0-4)	1.00	2.00	2.00	3.00	4.00	2.23	.87

QUAL (0-4)	1.00	2.00	2.00	3.00	4.00	2.30	.91

Abbreviations: sleep onset latency (SOL), wake time after sleep onset (WASO), number of night-time awakenings (WAKE), total sleep time (TST), time in bed (TIB), sleep efficiency (SE), restful/restorativeness (REST), quality (QUAL).

Sleep before testing was assessed via a sleep diary, and this information was then used to corroborate the results above. In general, subjects slept well prior to testing, with a majority experiencing few indicators of sleep disruption (SOL, WASO, WAKE, SE). Subjects also tended to report normal sleep duration (TST), with a normal time in bed (TiB). The subjective sleep experience of subjects also tended to be good (QUAL, REST).

Sleep diary measures were then compared on PSQI scores; there were significant group differences in SOL (t (54) = - 2.01, p = 0.05), TST (t (54) = 2.40, p = 0.02), SE (t (54) = 3.54, p = 0.001), REST (t (54) = 3.84, p < 0.001) and QUALITY (t (54) = 2.67, p = 0.01). For all measures results were in the expected directions.

Table 5. Sleep diary results by sleep group

	PSQI			
	< 7		> 6	
	Mean	Std Dev	Mean	Std Dev
SOL	14.33	16.42	31.21	45.34
WASO	4.77	12.08	10.00	15.94
WAKE	.51	.85	2.98	8.59
TST	6.97	1.28	6.08	1.44
TIB	7.43	1.19	7.80	1.40
SE	93.88	8.18	81.08	18.72
REST	2.54	.78	1.71	.78
QUAL	2.54	.89	1.90	.83

Abbreviations: sleep onset latency (SOL), wake time after sleep onset (WASO), number of night-time awakenings (WAKE), total sleep time (TST), time in bed (TIB), sleep efficiency (SE), restful/restorativeness (REST), quality (QUAL).

We then compared the inter-relationships between these different test-day sleep diary measures. Of the measures of sleep continuity, WASO and WAKE were significantly correlated (*rho* = 0.94, p < 0.001), and these measures correlated with subjective sleep experience. There were significant correlations between SOL and QUALITY (*rho* = - 0.27, p = 0.05), WASO and REST (*rho* = - 0.38, p = 0.004), WAKE and REST (*rho* = - 0.34, p = 0.01), and SE and REST (*rho* = 0.41, p = 0.002) and SE and QUALITY (*rho* = 0.41, p =

0.002). TST also correlated significantly with TiB (*rho* = 0.55, $p < 0.001$), SE (*rho* = 0.57, $p < 0.001$), and QUALITY (*rho* = 0.39, $p = 0.003$).

Emotion Perception

We then assessed emotion perception (accuracy and reaction times) for each of the sleep groups, in four independent repeated measure ANOVAs. When PSQI groups were compared on accuracy, there was a main effect of emotion (F (3.95, 213.42) = 37.08, $p < 0.001$), but no significant effects with emotion and group (F (3.95, 213.42) = 0.15, $p = 0.96$), or group (F (1, 54) = 0.51, $p = 0.48$). For reaction times, there was a main effect of emotion (F (3.38, 178.85) = 25.29, $p < 0.001$), but no effects of emotion by group (F (3.38, 178.85) = 0.68, $p = 0.64$), or group (F (1, 53) = 2.12, $p = 0.15$). As results were not significant the issue of multiple comparisons was not relevant.

In order to assess whether sleep and mood would predict emotion recognition, the predictor variables of HADS-D, HADS-A, MEQ, ISI and PSQI scores were entered alongside whether subjects were currently experiencing sleep disruption, and measures of the previous night's sleep – total sleep time, time in bed, sleep efficiency, sleep onset latency, wake time after sleep onset, number of night-time awakenings, and the subjective measures of sleep quality and restorativeness. These variables were used against measures of accuracy and reaction times for each of the six emotions, i.e. anger, fear, happiness, surprise, disgust and sadness in exploratory regression models. Significant models ($p \leq 0.05$) were followed up via individual predictor variables.

The resultant models were not significant for the accuracy of recognition towards angry faces (F (14, 41) = 0.59, $p = 0.86$), fearful faces (F (14, 41) = 0.93, $p = 0.54$), sad faces (F (14, 41) = 1.73, $p = 0.09$), disgusted faces (F (14, 41) = 0.70, $p = 0.76$), or surprised faces (F (14, 41) = 1.04, $p = 0.43$), although the model was significant for happy faces (F (14, 41) = 3.02, $p < 0.005$). Significant individual predictors were the number of awakenings the previous night (B = - 0.24, $S.E.$ = 0.06, $p = 0.001$), depression (B = - 0.38, $S.E.$ = 0.13, $p = 0.007$), and the previous night's sleep quality (B = 1.26, $S.E.$ = 0.52, $p = 0.02$).

Similar results were found for reaction times. The models were not significant for anger (F (14, 41) = 1.01, $p = 0.46$), fear (F (14, 41) = 0.72, $p = 0.75$), sadness (F (14, 41) = 1.09, $p = 0.39$), disgust (F (14, 41) = 1.12, $p = 0.38$), or surprise (F (14, 41) = 0.61, $p = 0.84$), although the model was significant for happiness (F (14, 41) = 1.95, $p = 0.05$). Significant individual predictors were the previous night's total sleep time (B = 174.91, $S.E.$ = 69.21, $p = 0.02$), quality (B = - 97.80, $S.E.$ = 41.51, $p = 0.02$), wake time after

sleep onset (*B* = 5.26, *S.E.* = 2.40, *p* = 0.03), time in bed (*B* = - 139.74, *S.E.* = 64.24, *p* = 0.04), and sleep efficiency (*B* = - 12.21, *S.E.* = 5.70, *p* = 0.04).

Empathy

We next compared the four empathy subscales for sleep groups in repeated-measures ANOVAs. For PSQI groups, there was a main effect of empathy (*F* (2.53, 136.57) = 28.06, *p* < 0.001), but no interaction (*F* (2.53, 136.57) = 1.11, *p* = 0.34) or main effect of group (*F* (1, 54) = 0.07, *p* = 0.80). Exploratory regression models were also created for the four subscales of the Interpersonal Reactivity Index using the same predictor variables as above. The overall models were not significant for perspective taking (*F* (14, 41) = 1.11, *p* = 0.38), fantasy scale (*F* (14, 41) = 1.14, *p* = 0.35), empathy component (*F* (14, 41) = 0.46, *p* = 0.94), or personal distress (*F* (14, 41) = 1.22, *p* = 0.30).

Discussion

Results suggest that sleep groups created on the basis of PSQI scores show the expected differences in ISI and HADS scores. Poor sleepers have higher ISI, HADS-A and HADS-D scores, but there were no significant differences in morningness-eveningness preference. PSQI scores also correlate with ISI scores, and both measures correlate with anxiety and depression scores, but not with a measure of diurnal preference. Sleep groups also showed the expected differences in SOL, TST, SE, REST and QUALITY scores. Furthermore, there were significant correlations with TST and TiB, SE and QUALITY. SOL and QUALITY were correlated, as were WASO and REST, WAKE and REST, and SE and REST. There were no significant effects of sleep group on emotion recognition performance, on either accuracy or reaction times. When exploratory regression models were created using these predictor variables, the models were significant for the accuracy and reaction times towards happy faces. For accuracy, significant predictors were depression, and the previous night's QUALITY and WAKE. For reaction times, significant predictors were the previous night's TST, QUALITY, WASO, TiB and SE. There were no significant effects with the components of empathy.

Three main conclusions were drawn from the current study. Firstly, there were few significant effects of sleep group on the face task, even though this task was intentionally made more difficult by a restricted viewing time. As a consequence, future studies will make use of dynamic face stimuli in order to increase task difficulty. Moving stimuli which are based on physiological Action Units provide an ecologically valid means of assessing emotion perception while simultaneously varying the displayed

intensity (Adolphs, 2002; Ekman, Friesen, and Tomkins, 1971; Ekman and Friesen, 1976). Such an approach is also in keeping with the psychopathology literature.

It also appears noteworthy that although sleep groups were not associated with significant effects on emotion perception, regression models identified effects of sleep on happiness. With regards to the accuracy of happiness recognition, depression, sleep quality, and the number of night-time awakenings were significant predictors. This is perhaps surprising as previous results have shown that happy faces are recognized with high accuracy (Calvo and Lundqvist, 2008). With regards to reaction times, significant results were found with happiness for total sleep time, sleep quality, wake-time after sleep onset, time in bed, and sleep efficiency. These results may be interpreted within the context of anhedonia, and overlap of depression and insomnia (Staner, 2010). There is evidence that depression is linked to a response bias towards sadness, with some evidence that sad and happy faces are recognized more poorly (Bourke, Douglas, and Porter, 2010). However, the recognition of positive emotion has not been found to be affected by psychological distress within a healthy population (Csukly et al., 2008). Furthermore, sleep deprivation has been associated with impaired intensity ratings of angry and happy faces in the mid-intensity range (Van Der Helm et al., 2010).

Secondly, the correspondence of different sleep measures was considered. In particular, the PSQI and ISI are highly correlated, although some participants (29%) classification would be altered if a solitary measure was used. Indeed, this is the method employed by LeBlanc et al. (2009). In this study, 'insomnia syndrome' was a combination of ICD-10 and DSM-IV-TR criteria. Participants with insomnia syndrome were identified as those who were dissatisfied with their sleep and reported distress on the ISI. The PSQI was used to assess the presence of sleep disruption, and participants were also included within the insomnia syndrome group if they reported the use of sleep medication. Furthermore, Edinger et al. (2004) define insomnia disorder as reported sleep disruption with associated daytime effects, which occur despite adequate sleep opportunity.

Specifically, results suggest that in the current study, sleep groups evidenced the expected effects in measures of distress (i.e. anxiety and depression), and on test-day sleep diary measures of sleep disruption. Furthermore, sleep group mean scores suggest that restricted time in bed was not a cause of significant sleep disruption. In the current sample there were a low number of participants who reported clinically significant levels of insomnia severity as defined by the ISI. This could reflect flexibility within the student lifestyle, which may result in sleep disruption being accommodated by flexibility of sleep timing, thus mitigating the effects of poor sleep. Alternatively, these

insomnia participants may be experiencing transient insomnia, and have a preserved sleep system which is able to accommodate stress (Espie, 2002). Indeed, normal sleepers are known to experience transient sleep disruption (Espie, 2002; Espie et al., 2012). Furthermore, the sleep of people with insomnia has been found to show night-to-night variability (Sanchez-Ortuno et al., 2011; Vallieres et al., 2011; Vallieres et al., 2005), and future studies may wish to consider this in order to understand the development of chronic insomnia.

Thirdly, results suggest that assessing subjective empathy is of limited value within the current population, as no significant effects of sleep or mood were found on either of the two measures of cognitive empathy or affective empathy. Using another measure of subjective empathy, Killgore et al. (2007) report significant effects of sleep deprivation on three subscales, i.e. intrapersonal functioning, interpersonal functioning, and stress management. The failure to find significant associations of sleep and mood in the current study may indicate that the sleep disruption of the current subjects was either not chronic enough, or that they had not experienced sufficient levels of sleep loss. However, concern with social interactions has been reported in patients with insomnia (Kyle, Espie, and Morgan, 2010). In particular, insomnia is perceived to be isolating, with a lack of understanding by others, and this has been attributed to fatigue. The failure to find effects on empathy may also relate to the relatively low levels of insomnia severity within this sample.

A limitation of the current study was that the subjects were assigned to sleep groups retrospectively on the basis of questionnaire responses. The higher PSQI score was chosen due to increased sensitivity for insomnia disorder (Buysse et al., 1989; Backhaus et al., 2002). However, it should be noted that levels of sleep disruption among the normal sleepers group was relatively high. Furthermore, a majority of participants were students, and sleep disruption is common in this population (Lund et al., 2010). As such, results may be somewhat different with a sample of subjects with insomnia disorder. However, results from the current study suggests that indicators of sleep disruption the previous night, and depression scores, affect the recognition of happiness from faces. These results would be expected to be more pronounced in samples with stricter inclusion criteria. An additional limitation may relate to sample size, and the possibility that the srudy was under-powered. However, the sample size was consistent with existing experimental work in this area, and it appears more plausible that the anticipated effects are subtle.

In summary, results suggest that the formation of sleep groups would be improved by the intentional recruitment of insomnia and normal sleeper subjects. In particular, this is anticipated to increase the levels of insomnia severity which are reported by poor sleeper groups. However, the identification of sleep measures affecting the recognition of happiness, on both reaction time and accuracy measures suggests that sleep affects emotion perception. Emotion is known to play a key role in insomnia (e.g. Riemann et al., 2010; Bonnet and Arand, 2010; Espie, 2002; Harvey, 2002), and this result is supported by patient experiences of daytime impairments, and the overlap of emotion perception with emotional brain areas. The ability of future studies to identify effects of sleep on emotion perception will also be improved by the use of 4D stimuli, which are expected to be an ecologically valid means of increasing task difficulty and therefore increasing the sensitivity towards emotional dysregulation and sleep disruption. As a result of this study we decided to investigate the effects of sleep on emotion recognition in greater depth, and the current study informed the subsequent one in several ways. The subsequent study recruited people with insomnia disorder, rather than investigating the effects of poor sleep calculated from PSQI scores. We also employed a more sensitive task of emotion recognition, and this task displayed emotions dynamically, over three levels of emotional intensity. Dependent variables were therefore emotion recognition accuracy (labels and intensity), as well as reaction times. In this study we anticipated effects on happiness recognition, based on the current results.

Chapter Four

Does insomnia disorder affect the recognition of emotion?

Abstract

Daytime impairments in functioning are a feature of insomnia disorder, and this is reflected in both diagnostic criteria and subjective reports. Social functioning is an important part of daytime functioning, and people with insomnia report that this is a pertinent area of concern for them. One way in which social functioning can be assessed is via the perception of emotion in faces. However how insomnia affects emotion recognition has only recently been investigated, and the current study extends this literature by the use of dynamic stimuli, at three levels of intensity. Normal sleepers and participants reporting insomnia completed the emotion recognition task with angry, fearful, sad, happy, surprised and disgusted faces, and were required to categorize the emotion shown and rate its intensity. Participants were found to show impairments in emotion categorization towards high-intensity expressions of sadness and low intensity expressions of surprise. There were no significant effects of sleep group on emotional intensity ratings or reaction times. Regression analyses also found significant effects of sleep and mood on happiness recognition. Results indicate that insomnia affects emotion recognition, suggesting that the social functioning complaints in insomnia patients are supported by objectively measured performance impairments.

Does insomnia disorder affect the recognition of emotion?

Introduction

Insomnia is a disorder which significantly affects an individual's daily life, including functioning and performance at work (Kucharczyk, Morgan, and Hall, 2012), and ultimately impacts upon society as a whole (Kucharczyk, Morgan, and Hall, 2012; Léger and Bayon, 2010). For example, 14.5% of all costly workplace accidents are associated with insomnia (Shahly et al., 2012), and annual losses to the US labour force equal to 367 million days and 91.7 billion dollars (Kessler et al., 2011). Such daytime functioning impairments are a criterion for the diagnosis of insomnia, and can include impairments in social interactions (Edinger et al., 2004), which have been identified by insomnia patients as an area of concern (Kyle, Espie, and Morgan, 2010). Furthermore, people with insomnia are reported to be more self-focused, and less engaged, than good sleepers (Marchini et al., 1983). However, to date the socio-emotional effects of insomnia disorder have been largely based on self-reports.

Recently, objective measures of emotion performance have begun to be used in patients with insomnia. Baglioni et al. (2010) report effects of emotional sleep-related stimuli on physiological measures of emotional reactivity. In particular, people with insomnia were less responsive to sleep-related positive images, measured via tonic responses of the "frowning" muscles, and rated all stimuli as more arousing, with enhanced cardiac vagal tone. In support of this, evidence of impaired emotional regulation has recently been reported by DelVentura et al. (2013), using physiological measures and self-reports in response to emotional images. Among poor sleepers, amygdala activity has been found to correlate with measures of psychopathology, in an emotion face-matching task (Prather, Bogdan, and Hariri, 2013). In a task of emotional enhancement and regulation in response to emotional images, the use of sleeping medication was linked to less activity within the medial prefrontal cortex (Minkel et al, 2012). Such results are consistent with the importance of emotion in the development and maintenance of insomnia (c.f. Baglioni et al., 2011).

While these studies report effects of insomnia and poor sleep on measures of emotion, a recent study has assessed how insomnia affects the perception of emotion from faces, and these faces are particularly salient for social interactions (e.g. Bruce and Young, 1998; Calder and Young, 2005). Kyle et al. (2014) report impairments in the recognition of emotional *intensity* in sad and fearful expressions among people with psychophysiological insomnia. This result is thought to reflect either the effects of sleep

loss, the effects of psychological distress, and/or the effects of trait-level emotional coping strategies, although future research is necessary to clarify this. Regardless of the underlying reason for impaired recognition of emotional intensity, such results are in keeping with the reported adverse effects of insomnia on functioning (Kyle, Espie, and Morgan, 2010). No effects were found on the ability to categorize emotion, a result which likely represents high levels of accuracy in all subjects.

Indeed, variations of emotion intensity are often employed among patient groups by morphing expressions, a procedure which usually involves placing markers on two images (of the same individual) and combining them (e.g. Adolphs, 2002). Such expressions are commonly used with patient groups, in order to assess the effects of psychopathology on emotion recognition of varied intensity levels within static images (Mendlewicz et al., 2005; Csukly et al., 2009; Beevers et al., 2009; Gilboa-Schechtman et al., 2008; Yoon, Joorman and Gotlib, 2009; Surguladze et al., 2004), or movies depicting emotional displays (Kan et al., 2004; Joorman and Gotlib, 2006; Joorman, Gilbert and Gotlib, 2009; LeMoult et al., 2009). In everyday social interactions, emotional displays are changeable and dynamic, and dynamic images may be linked to greater effects on arousal (Sato and Yoshikawa, 2007). In research conditions, dynamic faces have previously been created by morphing facial expressions. Given that insomnia has been described as a disorder of hyperarousal (e.g. Riemann et al., 2010), such images may therefore involve the arousal processes which are thought to be particularly relevant to this disorder.

The current study aimed to extend research on the daytime effects of insomnia (Kyle, Espie, and Morgan, 2010; Kyle et al., 2014), via the use of a socially-relevant emotion recognition task. The 4D faces used as stimuli in the current study confer several additional advantages over previously used morphed images. Importantly, the facial movements of the 4D stimuli are derived from real-life emotion expressions which are based on physiological movements (see, e.g. Adolphs, 2002). In particular, these stimuli have been developed from the work of Ekman and Friesen (1976), who have identified facial Action Units which are responsible for expressing an emotion. Stimuli were created from individual trained in these facial movements, which were demonstrated and captured electronically in order to be parameterized, and modeled on new facial identities. As such, the 4D platform created by Yu, Garrod and Schyns (2012) may provide a sensitive, and ecologically valid, measure of emotion recognition performance in poor sleepers. Based on the theory of insomnia as a disorder of hyperarousal (Riemann et al., 2010), poor sleepers may be expected to show performance enhancement, relative to normal sleepers. Alternatively, and perhaps more likely, poor

sleepers may be expected to show impaired performance, consistent with the face task of Kyle et al. (2014), and in general (e.g. Kyle, Espie and Morgan, 2010). Performance was assessed via accuracy, reaction times, and intensity ratings, and based on the previous results sleep was expected to contribute towards happiness recognition. In conjunction with this, subjective measures were included to characterize self-reported mood and emotional functioning, via the personality trait of alexithymia.

Methods

Participants

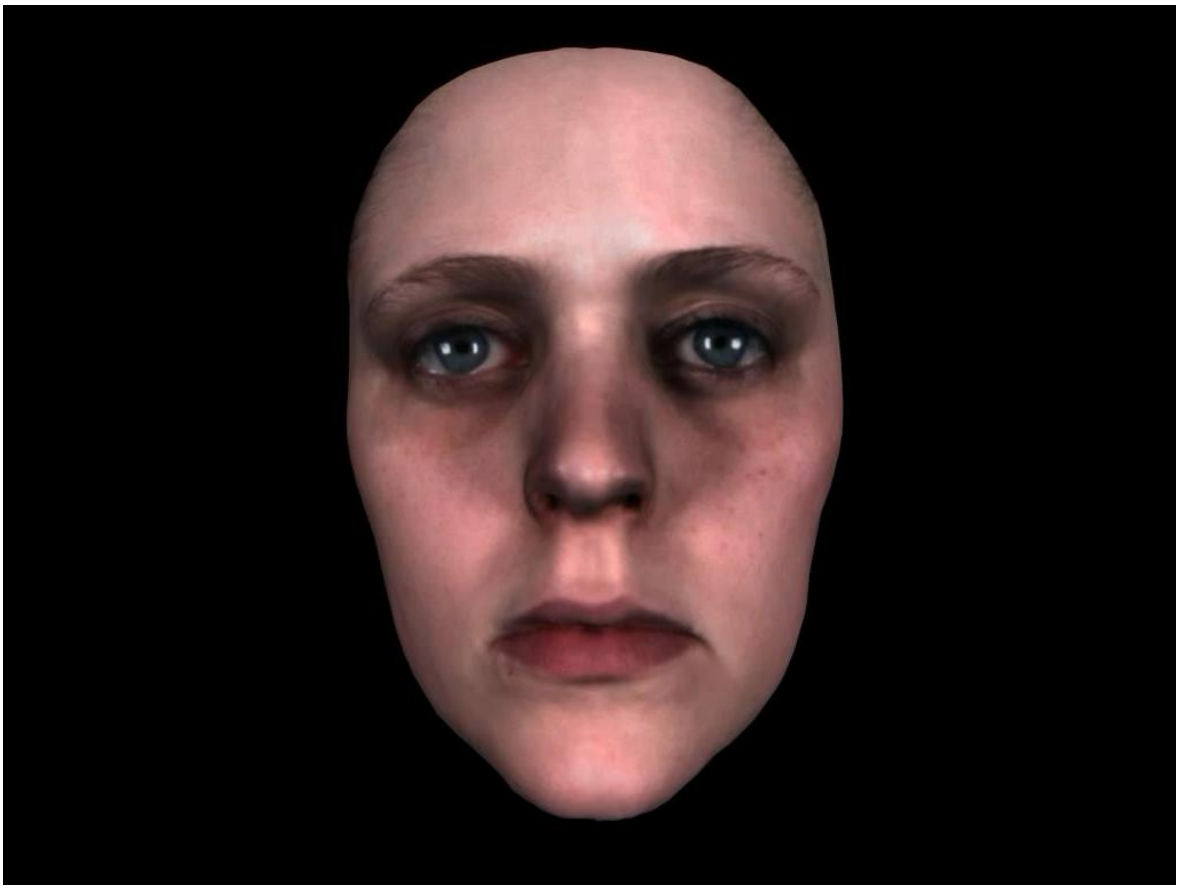
In total, data was available from 27 subjects, with three additional subjects excluded due to incomplete task data. Ages ranged from 19 to 63, with a mean of 29.07 (*SD* = 13.15). All subjects reported good physical health, with no sleep disorders (apart from insomnia), or recent psychiatric disorders. All subjects were Western Caucasian. This study had the approval of the local Ethics Committee and participants provided informed consent. Subjects were identified via emailing the departmental Subject Pool and word of mouth. Participants completed the PSQI and ISI prior to testing to indicate likely normal sleepers and insomnia participants, and these measures were repeated at testing and reported below. Sleep groups were created on the basis of PSQI scores.

Stimuli

Stimuli were created using the 4D stimuli platform, which is described in detail by Yu, Garrod and Schyns (2012), and described in Appendix 2.

Emotions were shown with four male and four female Caucasian identities, each showing the six emotions (anger, fear, sad, disgust, surprise, happy) at three levels of intensity (low, medium, and high). Expressions began with a neutral expression before developing to display the full expressions, and back to neutral. Each expression was shown twice, giving 288 trials. Emotions were displayed over 1.25 seconds, at average dimensions of 8 cm by 13 cm, at a viewing distance of 72 cm.

Figure 5. Example dynamic face stimuli



Measures

- PSQI (Buysse et al., 1989).
- ISI (Bastien, Vallieres, and Morin, 2001).
- MEQ (Horne and Ostberg, 1976).
- Sleep diary completed on test day (Carney et al., 2012).
- HADS (Zigmond and Snaith, 1983).
- Toronto Alexithymia Scale, 20 item version (TAS-20; Bagby, Parker and Taylor, 1994).
- Fear of Negative Evaluation (FNE; Watson and Friend, 1969).

Further information as to these measures can be found in Appendix 1.

Procedure

Potential subjects were invited to complete the PSQI and ISI as screening measures, which were used to indicate likely normal sleeper and insomnia participants. Suitable participants were then invited to take part in the task, and testing took place between

12 noon and 6 pm. Participants completed two sets of practice trials first, which involved identifying emotion words with the congruent word label; and categorizing and rating the intensity of static emotion face stimuli (one individual displaying six emotions) was selected from the Ekman and Friesen (1976) stimuli set.

Dynamic face stimuli were then presented in a random order, and following their presentation participants categorized the emotion shown and rated its perceived intensity of expression. The keys 'Z', 'X', 'C', 'V', 'B', and 'N' were used to label the emotions of happiness, surprise, fear, disgust, anger, sadness, with an additional option of the space-bar if subjects were unsure. Participants then rated the intensity on a 7-point scale, anchored at 'not very intense' and 'extremely intense', and were asked to respond as quickly and accurately as possible. Following the task, participants completed the questionnaire battery, including the two sleep measures of the PSQI and ISI, from which final group allocation was confirmed.

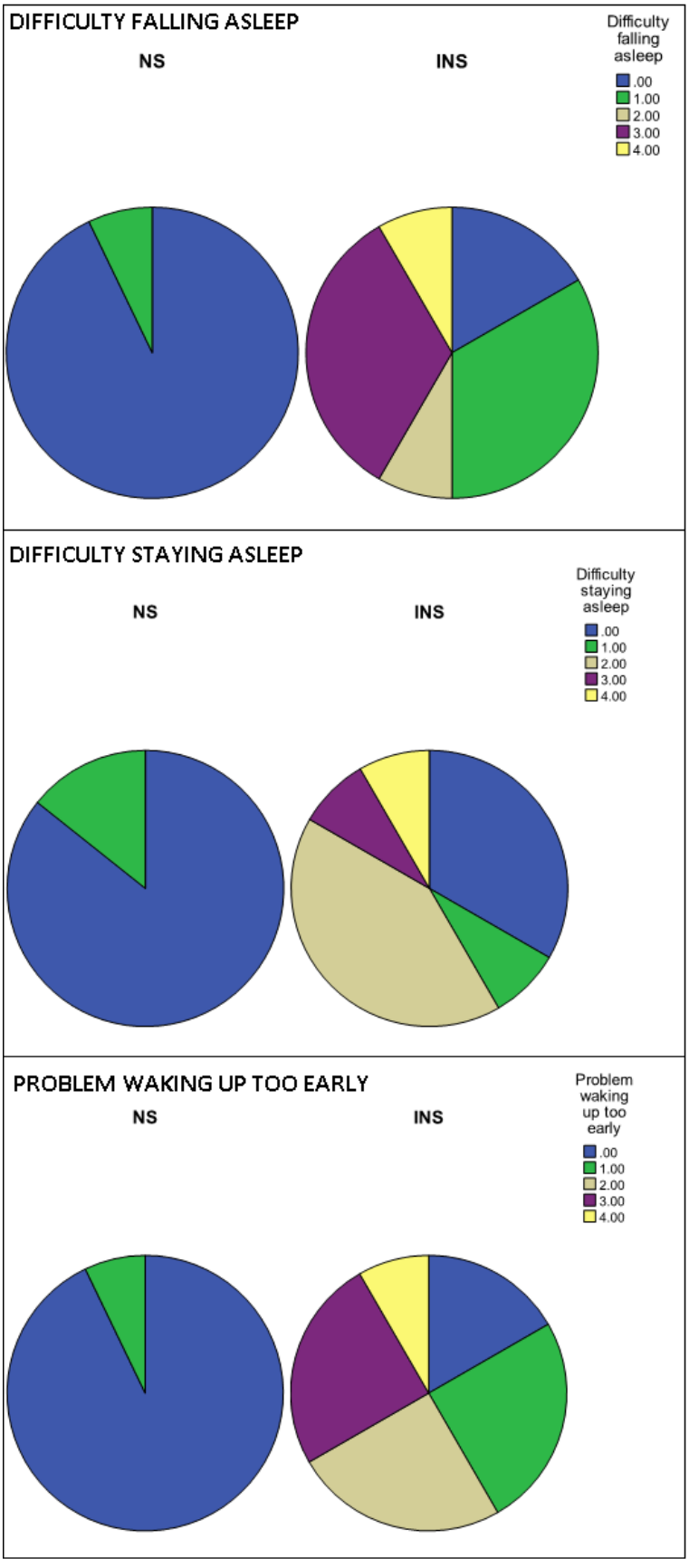
Results

Group characterization

Sleep groups were created using the PSQI cut-off of 6, which resulted in 15 Normal Sleepers (NS) and 12 Insomnia (INS) subjects. NS scores ranged from 1 – 5, whereas INS scores ranged from 7 – 15. We then assessed the effects on ISI scores, with ISI data unavailable for one INS subject. NS scores ranged from 0 – 3, whereas INS scores ranged from 7 – 18. No NS subjects endorsed an insomnia complaint, although 58% of INS did.

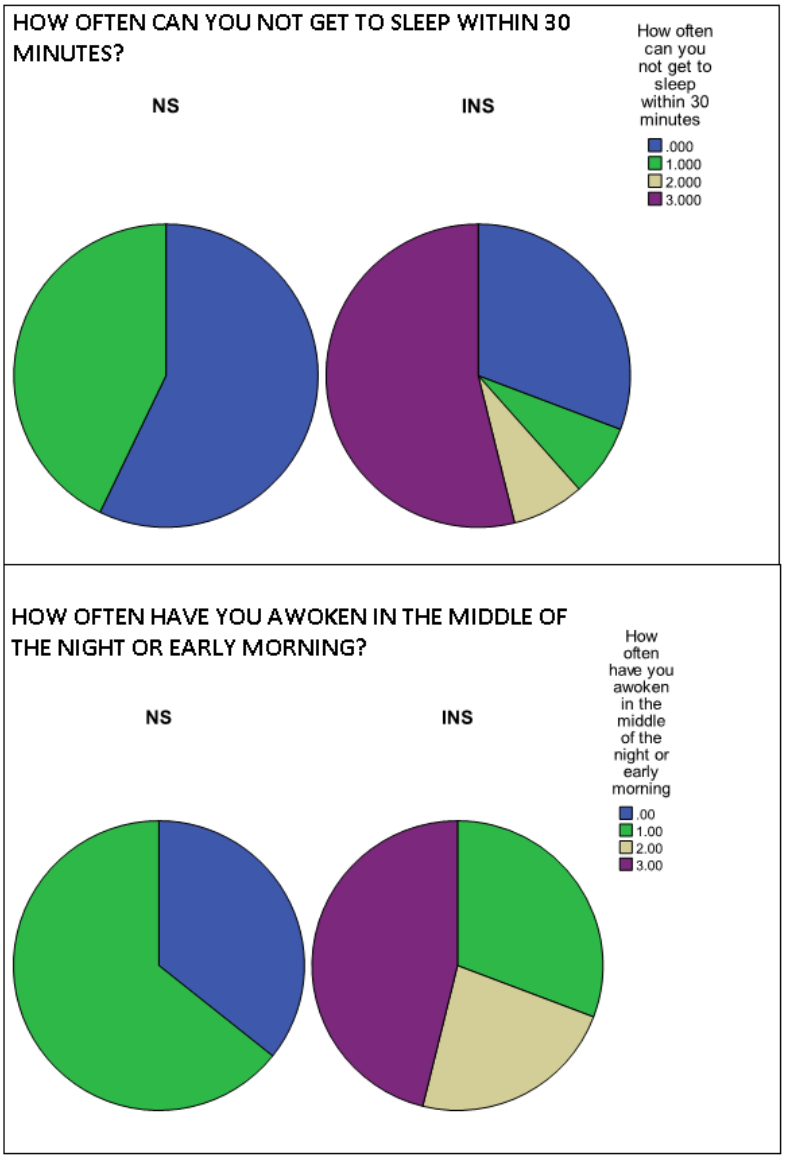
With regards to the severity of sleep disruption taken from ISI scores, all normal sleepers reported none or mild sleep disruption over the previous week. In contrast, INS participants reported greater severity of sleep disruption. For all three measures of sleep disruption, a majority of insomnia subjects reported moderate or greater levels of insomnia severity.

Figure 6. Proportions of insomnia symptom severity between groups. 0 = none, 1 = mild, 2 = moderate, 3 = severe, 4 = very severe.



With regards to the frequency of sleep disruption taken from PSQI scores, all normal sleepers reported an inability to get to sleep one night a week or less, and the same results were found with night-time awakenings. A majority of people with insomnia reported difficulties getting to sleep or staying asleep once or twice a week, or three or more times a week.

Figure 7. Frequency of insomnia symptoms (problems falling asleep, staying asleep and early morning awakenings) between groups. 0 = not during the past month, 1 = less than once a week, 2 = once or twice a week, 3 = three or more times a week.



We then assessed sleep satisfaction and the daytime effects of sleep disruption taken from ISI scores. All normal sleepers were either very satisfied or satisfied with their sleep, with minimal effects on the three additional measures. In contrast, a majority of insomnia subjects reported sleep dissatisfaction, with a majority experiencing daytime distress at least somewhat.

Figure 8. Proportions of the daytime effects of insomnia between groups. 0 = very satisfied, not at all interfering, not at all noticeable, not at all worried/distressed. 3 = very dissatisfied, very much interfering, very much noticeable, very much worried.

In a next step, the effects of sleep groups on the questionnaire measures of the PSQI, ISI, MEQ and HADS, and FNE and TAS-20 were assessed. These results were confirmed by questionnaire responses.

Table 6. Questionnaire responses by sleep group

	Group							
	NS				INS			
	Count	Mean	Median	Standard Deviation	Count	Mean	Median	Standard Deviation
PSQI	15	2.33	3.00	1.54	12	10.92	11.00	2.99
ISI	15	1.00	1.00	.85	12	12.92	13.50	3.53
MEQ	14	49.50	49.50	7.18	12	45.83	41.00	12.45
HADS-A	14	4.21	4.50	2.75	12	10.58	9.00	4.01
HADS-D	14	3.93	3.50	1.59	11	6.91	7.00	2.21
TAS-20	14	42.36	42.00	7.93	12	48.42	47.50	10.61
FNE	14	14.50	17.00	7.99	12	20.33	21.50	8.45

Abbreviations: Pittsburgh Sleep Quality Index (PSQI); Insomnia Severity Index (ISI); Morningness-Eveningness Questionnaire; Hospital Anxiety and Depression Scale – Anxiety (HADS A); Hospital Anxiety and Depression Scale – Depression (HADS D); Toronto Alexithymia Scale 20 item version (TAS 20); Fear of Negative Evaluation (FNE).

As expected, there were significant group differences in PSQI (t (26) = - 9.76, p < 0.001) and ISI (t (25) = - 12.69, p < 0.001) scores. There were no significant group differences in MEQ scores (t (24) = 0.94, p = 0.36). However, the group differences in anxiety (t (24) = - 4.78, p < 0.001) and depression (t (23) = - 3.92, p < 0.001) were significant. There were no significant group differences in alexithymia scores, assessed by the TAS-20 (t (24) = - 1.15, p = 0.11), but not in the fear of negative evaluation (t (24) = - 1.80, p = 0.08). Sleep groups were then compared on their sleep reports the night before testing.

Table 7. Sleep diary results by sleep group

	Group							
	NS				INS			
	Count	Mean	Median	Std Dev	Count	Mean	Median	Std Dev
SOL (mins)	15	14.33	6.00	15.76	12	43.13	45.00	32.88
WAKE (#)	15	.60	1.00	.63	12	2.71	2.50	2.37
WASO (mins)	15	4.73	2.00	8.66	12	53.13	45.00	50.56
TST (hours)	15	8.02	8.00	.98	12	6.23	5.88	1.32
TiB (hours)	15	9.15	9.33	1.37	12	8.33	8.50	1.09
SE (%)	15	88.89	90.23	9.94	12	75.29	79.37	15.01
QUALITY (0-5)	15	3.40	4.00	.74	12	2.00	2.00	1.04
REST (0-5)	15	2.93	3.00	.80	12	1.25	1.00	.97

Abbreviations: sleep onset latency (SOL), wake time after sleep onset (WASO), number of night-time awakenings (WAKE), total sleep time (TST), time in bed (TIB), sleep efficiency (SE), restful/restorativeness (REST), quality (QUAL).

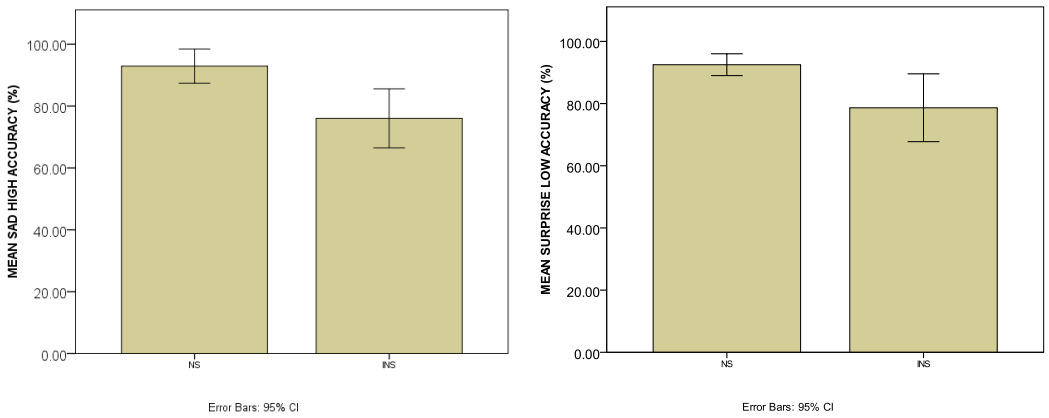
These were significant differences between groups in the measures of sleep onset latency (t (26) = - 2.61, p = 0.02), wake-time after sleep onset (t (26) = - 3.35, p = 0.003), and the number of awakenings (t (26) = - 2.87, p = 0.008). The group differences in total sleep time (t (25) = 1.85, p = 0.08), and sleep efficiency (t (26) = 0.63, p = 0.54) were not significant, although time in bed was (t (26) = 2.35, p = 0.03). However, there were significant group differences in subjective sleep quality (t (26) = 4.12, p < 0.001) and restorativeness (t (26) = 4.44, p < 0.001).

Task Results

Performance on the emotion task was compared between groups using three measures of performance: categorization accuracy, categorization reaction times, and intensity judgements. Categorization reaction times and intensity judgements were calculated as median responses of correct categorizations. Data was unavailable for one subject. Data were analyzed using Analysis of Variance (ANOVA), as in practice this is a robust test, and violations have relatively minor effects (Howell, 2002).When follow-up tests were conducted, the threshold for significance was set as 0.01, in order to provide an appropriate compromise between type I and type II errors (Perneger, 1998). Indeed, 0.05/6 = 0.01. Effect sizes can be found in Appendix 2.

For the accuracy of responses, results were compared in an ANOVA with emotion and intensity as repeated measures. There was a significant main effect of emotion (F (5, 125) = 20.02, p < 0.001), but no significant interaction of emotion and group (F (5, 125) = 0.62, p = 0.68). There was a significant main effect of intensity (F (2, 50) = 11.85, p < 0.001) but no significant interaction of intensity and group (F (2, 50) = 0.37, p = 0.69). The interaction of emotion and intensity was significant (F (5.93, 148.25) = 2.89, p = 0.002), but the three way interaction was not significant (F (5.93, 148.25) = 1.29, p = 0.26). The main effect of group was significant (F (1, 25) = 4.61, p = 0.04). Follow-up t-tests suggest that this effect was driven by responses to sad faces of high intensity (t (25) = 3.50, p =0.002), and surprise faces of low intensity (t (25) = 2.90, p = 0.008). These results were confirmed using non-parametric tests (sad high, U = 28.00, p = 0.002; surprise low, U = 40.50, p = 0.014).

Figure 9. Accuracy of recognition of sad faces of high intensity and surprise faces of low intensity between groups



We then assessed reaction times of categorization judgements. There was a significant main effect of emotion (F (2.50, 59.88) = 17.93, p < 0.001), but no significant interaction of emotion and group (F (2.50, 59.88) = 0.27, p = 0.93. There was a significant main effect of intensity (F (2, 48) = 7.82, p = 0.001), but no significant interaction of intensity and group (F (2, 48) = 0.51, p = 0.60. The interaction of emotion and intensity was significant (F (3.44, 82.63) = 4.21, p = 0.006), but there was no significant three-way interaction (F (3.44, 82.63) = 0.74, p = 0.55. There was no significant main effect of group (F (1, 24) = 0.18, p = 0.68).

This analysis was then repeated with the intensity ratings. There was a significant main effect of emotion (F (3.62, 86.88) = 19.90, p < 0.001), but no significant interaction of emotion and group (F (3.62, 86.88) = 0.23, p = 0.91). There was a significant main effect of intensity (F (1.36, 32.62) = 280.43, p < 0.001), but no significant interaction of intensity and group (F (1.36, 32.62) = 1.02, p = 0.34). There was a significant interaction of emotion and intensity (F (10, 240) = 8.26, p < 0.001), but no significant three-way interaction (F (10, 240) = 0.85, p = 058). The main effect of group was not significant (F (1, 24) = 1.27, p = 0.27).

Exploratory Regression Models: Happiness

Similarly to the previous chapter, the predictor variables of HADS-D, HADS-A, FNE, TAS-20, MEQ, ISI and PSQI scores were entered alongside measures of the previous night's sleep – total sleep time, time in bed, sleep efficiency, sleep onset latency, wake time after sleep onset, number of night-time awakenings, and the subjective measures of sleep quality and restorativeness were used as predictor variables for happiness recognition labels. This analysis was repeated for each of the three levels of happiness

intensity. Significant models ($p \leq 0.05$) were followed up via individual predictor variables.

The resultant models were significant for the accuracy of recognition towards happy faces of low intensity (F (15, 23) = 6.28, p = 0.007), medium intensity (F (15, 23) = 5.18, p = 0.01), and high intensity (F (15, 23) = 3.14, p = 0.05).

Significant individual predictors of happiness low intensity were PSQI (B = 17.22, $S.E.$ = 2.60, p < 0.001), ISI (B = - 2.97, $S.E.$ = 1.28, p = 0.05), HADS-A (B = - 2.88, $S.E.$ = 1.05, p = 0.03), TAS-20 (B = - 3.27, $S.E.$ = 0.61, p = 0.001), SOL (B = - 1.33, $S.E.$ = 0.28, p = 0.001), WAKE (B = 15.56, $S.E.$ = 2.96, p = 0.001), WASO (B = - 1.01, $S.E.$ = 0.20, p = 0.001), TST (B = - 73.84, $S.E.$ = 18.81, p = 0.004), TiB (B = 74.04, $S.E.$ = 16.99, p = 0.002), SE (B = 6.98, $S.E.$ = 1.76, p = 0.004), and MEQ scores (B = 1.47, $S.E.$ = 0.47, p = 0.01).

Significant individual predictors of happiness medium intensity were PSQI (B = 7.42, $S.E.$ = 1.72, p = 0.003), ISI (B = - 2.14, $S.E.$ = 0.85, p = 0.04), HADS-A (B = - 1.71, $S.E.$ = 0.70, p = 0.04), TAS-20 (B = - 1.06, $S.E.$ = 0.41, p = 0.03), WAKE (B = 5.62, $S.E.$ = 1.95, p = 0.02), WASO (B = - 0.30, $S.E.$ = 0.13, p = 0.05), TST (B = - 42.09, $S.E.$ = 12.43, p = 0.01), TiB (B = 38.49, $S.E.$ = 11.22, p = 0.009), and SE (B = 3.24, $S.E.$ = 1.16, p = 0.01).

Significant individual predictors of happiness high intensity were HADS-A (B = - 2.94, $S.E.$ = 1.04, p = 0.02), and WASO (B = - 0.48, $S.E.$ = 0.20, p = 0.04). This analysis was then repeated for reaction times. However, the model was not significant for happy faces of low intensity (F (15, 23) = 2.27, p = 0.12), medium intensity (F (15, 23) = 1.69, p = 0.23), or high intensity (F (15, 23) = 2.21, p = 0.13). Similarly, the model was not significant for intensity ratings of happy faces of low intensity (F (15, 23) = 1.24, p = 0.39), medium intensity (F (15, 23) = 0.90, p = 0.59), of high intensity (F (15, 23) = 0.48, p = 0.90).

Discussion

In summary, people with insomnia were found to be impaired at recognizing emotion from sad faces of high intensity and surprise faces of low intensity, with significant differences found in the emotional categorization labels used. There were no significant differences in reaction times or intensity labels. Sleep groups showed the expected differences in measures of sleep and mood disruption. There were also significant effects of sleep and mood measures on happiness recognition. In particular, HADS-A and

WASO scores contributed towards the recognition of all three intensity levels, with additional effects of sleep continuity measures and total sleep time, as well as alexithymia, on the recognition of medium and low intensity levels.

Results from this task suggest that insomnia affects emotion categorization performance when dynamic stimuli are used. In particular, results suggest that sad faces of high intensity, and surprised faces of low intensity, are recognized less accurately in insomnia. Such results are consistent with reports of daytime performance impairments in insomnia (Kyle, Espie and Morgan, 2010), and with results reported by Kyle et al. (2014) with static faces. Both the current study and Kyle et al. (2014), found effects with sad faces, although Kyle et al. (2014) reported additional effects with fearful expressions. Another potential difference relates to the precise task where effects were found. While both Kyle et al. (2014) and the current study required subjects to first categorize the emotion then rate its intensity, the current study found effects on emotional categorization, whereas Kyle et al. (2014) report effects on intensity ratings. However, the dynamic images of the current study were manipulated to show varying levels of intensity (based on normative data), thus providing a more difficult task, and it is possible that similar processes are contributing to both effects. Furthermore, the current study extends the perceived emotional 'blunting' effect reported by Kyle et al. (2014), to impaired categorization performance, when using dynamic stimuli with varying levels of expressed intensity. Measures of sleep and mood were also found to contribute towards happiness recognition in keeping with the previous chapter, although there were no significant group differences.

Kyle et al. (2014) have identified three possible reasons as to why emotion perception is altered in insomnia, which relate to the effects of sleep loss, the effects of psychological distress, and/or the effects of trait-level emotional coping strategies. These three predictions will now be discussed with reference to the current data. Firstly, the insomnia participants in the current study experienced poorer sleep. On the night before testing, these subjects reported lower sleep duration, increased sleep disruption, and experienced their sleep as of poor quality and not restorative. Results from the ISI and PSQI also suggest that subjects with insomnia had experienced poorer sleep over the previous two weeks and month (ISI and PSQI). However, these group differences are based on self-reports, and this data would be supported by use of actigraphy data and sleep diaries over a longer period. In addition, while total sleep deprivation has been associated with impaired emotional intensity judgements (e.g. van der Helm et al., 2010), total sleep deprivation and sleep disruption are different phenomena, and induced sleep disruption in otherwise healthy sleepers could itself

differ from the experience of insomnia patients (Bonnet and Arand, 1996). The extent to which sleep disruption and sleep loss affect emotion perception would require additional research, including objective measures of the sleep of normal sleepers and insomnia participants.

Psychological distress could also affect the perception of emotion in faces, and a number of previous studies have reported effects of psychopathology on emotion perception tasks (e.g. Mendlewicz et al., 2005; Csukly et al., 2009; Beevers et al., 2009; Gilboa-Schechtman et al., 2008; Yoon, Joorman and Gotlib, 2009; Surguladze et al., 2004; Kan, et al., 2004; Joorman and Gotlib, 2006; Joorman, Gilbert and Gotlib, 2009; LeMoult et al., 2009). The insomnia subjects in the current sample reported greater psychological distress, evidenced by anxiety and depression scores, and this could contribute towards results. Indeed, sleep and mood are inter-linked, and the relationships between them are complex (Kahn, Sheepes and Sadeh, 2013). This also appears in keeping with the results of the regression models showing effects of sleep and mood on happiness recognition. Furthermore, it may not be possible to fully dissociate the effects of insomnia and mood disruption. For example, insomnia and depression are often comorbid, and this vulnerability towards insomnia and depression could be related (Staner, 2010). Alternatively, the vulnerability for insomnia could contribute towards both depression and insomnia, and the vulnerability for depression could contribute towards both depression and insomnia (Staner, 2010). Another possibility is that the comorbidity of insomnia and depression could represent an additional vulnerability factor and separate disorder (Staner, 2010). As such, emotional distress would be expected to relate to sleep disruption and mood disorder vulnerability, and to contribute towards alteration in emotion perception due to overlapping brain regions (Phillips et al., 2003). A direct test of this would require longitudinal studies among subject groups with different mood disorder vulnerabilities. However, the report by van der Helm et al. (2010) of impaired perception of anger and happiness in healthy sleep-deprived subjects indicates that sleep disruption contributes towards mood disruption. These results were not found to be affected by self-rated sleepiness. Sleep disruption could therefore represent an additional factor which can contribute towards psychiatric disorders (Bryant et al., 2010; Yoo et al., 2007).

With regards to a role of trait-level differences in emotional coping strategies, the relationships between emotion perception, emotional regulation, and psychiatric illness have been reviewed by Phillips et al. (2003). These authors suggest that emotional regulation processes could directly affect the initial appraisal of stimuli. Emotional regulation is also thought to affect emotion perception indirectly, by modulating mood

state which also affects the appraisal of emotional stimuli. Dysregulation of these processes are thought to contribute towards the impairments found in psychiatric illnesses (Phillips et al., 2003). In support of this, symptoms of depression have been linked to emotion recognition performance, and modulated by emotion recognition strategies (Aldinger et al., 2013). With regards to insomnia, emotional regulation is thought to contribute towards the sleep problems reported by these patients, and insomnia has been characterized as a disorder of regulating arousal (c.f. Espie, 2002; Perlis et al., 1997; Riemann et al., 2010). For example, people with insomnia experience less control over stressful life events, and perceive daily events as more stressful (Morin, Rodrigue, and Ivers, 2003). This may be due to their greater tendency to employ emotion-focused coping strategies (Morin, Rodrigue, and Ivers, 2003). When the relationships between variables were modeled, emotion-focused coping was found to contribute towards the impact of stress and pre-sleep cognitive arousal, thus affecting sleep efficiency (Morin, Rodrigue, and Ivers, 2003).

In the current study, alexithymia, a personality trait which is linked to the ability to identify and describe one's own emotions, was measured. This trait has been linked to lower scores of cognitive empathy (perspective taking) and greater empathic distress (Moriguchi et al., 2006). The alexithymia subscales of 'difficulty identifying feelings' and 'difficulty describing feelings' load on a factor alongside empathic personal distress, whereas cognitive empathy and empathic concern loaded with the alexithymia subscale of externally oriented thinking (Grynberg et al., 2010). In response to a theory of mind task, those with high alexithymia tendencies report a lower appreciation of mental state, and appropriateness of animations, in terms of representing the intended narrative (Moriguchi et al., 2006). These response differences were reflected in lower activity of the right medial prefrontal cortex and superior temporal area (Moriguchi et al., 2006). As such, this trait would be expected to be linked to the ability to perceive emotion, and has previously been linked to a number of psychological disorders (c.f. Bagby, Parker and Taylor, 1994). Although there no were significant group differences in this scale, it is possible that alexithymia was a contributing factor in the emotion recognition impairment found in the current sample. As such, a contribution of this trait, or other measures of emotional coping, to emotion perception performance in insomnia disorder cannot be ruled out. Future studies could also investigate whether this trait represents a cause or effect of sleep disruption. In particular, alexithymia was found to contribute towards happiness recognition in exploratory regression models. Also pertinent could be the emotionality (i.e. arousal and valence) of these emotions, with sadness a low-arousal unpleasant emotion and surprise a high-arousal pleasant one (Adolphs, 2001). Happiness is also a pleasant emotion of mid-arousal, with disgust mid-

arousal unpleasant and anger and fearful both high-arousal unpleasant emotions (Adolphs, 2001). Visual attention towards the eye region could also contribute towards effects (Murphy and Isaacowitz, 2010).

Regardless of the underlying reason for these results, the current results suggest that even among a young adult population of insomnia subjects, the experience of insomnia is linked to impairments in a socially-relevant task. Such impairments may contribute towards the development of insomnia disorder, which includes the experience of daytime impairments in functioning (e.g. Edinger et al., 2004). For example, selective attention and monitoring of the daytime effects of poor sleep are thought to contribute towards the maintenance of this disorder (Harvey, 2002).

Social functioning has been suggested as an endpoint of treatment (Bech 2005), and a possible 'biomarker' for disorders (Derntl and Habel, 2011; Isaac, 2012). Current results perhaps suggest the utility of this task as an indicator of impairments in insomnia patients, and improvements following treatment, with results suggesting an effect of insomnia on the impaired recognition of high-intensity sadness faces. Future studies could investigate the neural correlates of this effect, and how the treatment of insomnia improves performance. This result adds to the existing literature (Kyle et al., 2014) and patient reports (Kyle, Espie and Morgan, 2010), in showing that insomnia is linked to impairments in social functioning. However, the current emotion recognition task measures one aspect of social functioning, and the ability to assess another person's perspective also contributes towards social competence (c.f. Iarocci, Yager, and Elfers, 2007). As such, future studies should investigate the effects of insomnia on additional measures of social functioning more broadly.

Limitations of this study include the young age of insomnia participants sampled and the sample size; however, results are consistent with previous reports of impaired emotion recognition in insomnia disorder (Kyle et al., 2014). This suggests that among people with insomnia there are differences in emotion recognition performance, and tha ability to detect these differences in the current study may relate to the use of a more sensitive task. Moreover, the sample size is consistent with that of Kyle et al., (2014). A further limitation of the current study relates to the nature of insomnia within the current sample. In particular, the severity of insomnia was lower than might be expected in a clinical setting, based on total ISI scores. However, a majority of insomnia participants reported moderate or greater levels of insomnia severity for each of difficulties falling asleep, staying asleep, and waking up too early. When these three measures were considered together, all normal sleepers reported mild insomnia

symptoms on a maximum of one measure of sleep disruption, in contrast to the insomnia participants. Furthermore, all insomnia participants scores above threshold on the PSQI, and as such, all insomnia participants met criteria for sleep disruption. Secondly, all insomnia participants reported that their sleep pattern was interfering with their daily life at least a little. Additionally, those subjects reporting no daytime effects on one of the remaining measures ('noticeable', and 'worried') reported effects on the other component. Thirdly, all insomnia participants reported adequate sleep opportunity, with a minimum time in bed of over six hours and no significant group differences with normal sleepers. As such all insomnia subjects met the criteria for insomnia disorder described by Edinger et al., (2004).

Results suggest that insomnia disorder affects emotion perception, with significant effects on emotion categorization with dynamic facial stimuli, and with significant effects on emotional intensity ratings with static stimuli. These results are most consistent with sad faces, and the current results with surprise and previous results with fear (Kyle et al., 2014) can be interpreted within the context of the confusabilities of these two emotions (Jack, Garrod, and Schyns, 2014). In particular, surprise and fear are frequently confused, as are anger and disgust (Jack, Garrod, and Schyns, 2014).

Future studies could also investigate the links of poor sleep and insomnia with the recognition of happy and sad faces, as there is evidence that results with these emotions may be affected by depression and show evidence of a negative bias (Bourke, Douglas, and Porter, 2010). In keeping with this suggestion, Kyle et al. (2014) reported significant moderate negative correlations of anxiety and depression with intensity rating in psychophysiological insomnia (PI), and similar results with anxiety in good sleeper controls. Furthermore, we report in Kyle et al. (2014) that anxiety was linked to sad ratings in good sleepers, and to anger and happiness ratings in PI. In patients, depression and sadness ratings were also found to be correlated. This may be consistent with reports by Van Der Helm et al. (2010) of impaired perception of happiness and anger in healthy participants who had been sleep-deprived. The role of happiness recognition in results would also be interesting to consider given the results of the previous chapter. However, exploratory regression analyses suggest that depression scores do not contribute to happiness recognition in the same way as the static stimuli used in the previous chapter. Low intensity happy faces, which are more ambiguous, also appear to be more sensitive to sleep and mood than high intensity stimuli, given the greater number of significant predictor variables associated with low intensity stimuli. As results were found with both INS and NS, we next assessed the effects of sleep on emotion perception in normal sleepers. In particular, we investigated the role

of time of testing on emotion perception, due to a lack of research in this area and its potential ability to inform our understanding of the etiology of insomnia (e.g. Espie, 2002). For example, insomnia has been conceptualized as a disorder of 24-hour hyperarousal, and normal sleepers ordinarily show fluctuations in mood and sleepiness.

Chapter Five

Is emotional sensitivity affected by sleep proximity? A study of normal sleepers

Abstract

Emotionality has been found to vary across the day (Hot, Leconte and Sequeira, 2005; Buysse et al., 2007); however, the extent to which this is apparent in sensitivity towards emotional images is unclear. As insomnia disorder is linked to hyperarousal (c.f. Espie, 2002; Riemann et al., 2010; Bonnet and Arand, 2010), it is important to understand how emotionality may vary in normal sleepers. Facial expressions of emotion are particularly salient for social interactions, and their recognition uses areas of the emotional brain with relevance for psychiatric disorders (c.f. Phillips et al., 2003). Of particular interest is how subjects recognize emotion early in the day and late in the day, as these periods may have particular relevance for insomnia disorder. The current study made use of dynamic facial stimuli, and subjects completed emotional recognition tasks, which comprised both categorization decisions and intensity ratings. In a first experiment, subjects completed the emotional recognition task in the lab, and testing took place at around three hours since waking or twelve hours since waking in a between-subjects design. Results suggest significant effects of sleep group with regards to sensitivity towards different temporal parameters of emotional faces. In a second experiment, subjects completed an online emotional recognition task upon awakening and close to bed-time, in a within-subjects design. Results suggest a significant effect of time since waking on emotional sensitivity in this experiment, in particular, with intensity ratings of mid-intensity expressions of sadness and anger. These effects could be linked to the testing environment and/or proximity to bed-time. Results are discussed in relation to the implications for insomnia disorder.

Is emotional sensitivity affected by sleep proximity? A study of normal sleepers

Introduction

Emotionality has been found to vary across the day in healthy subjects (e.g. Hot, Leconte and Sequeira, 2005; Gujar et al., 2011), and this could indicate that this variability may be disrupted in insomnia disorder. For example, poor sleep has been linked to the inhibition of de-arousal, which suggests that poor sleep results in diminished variability of arousal (Espie, 2002). An understanding of the processes involved in normal sleep is important in order to understand how insomnia develops (Espie, 2002; Buysse et al., 2011). With regards to waking, the ease of awakening from sleep is dependent upon circadian and homeostatic factors, as well as sleep propensity (Akerstedt et al., 2002). It has been suggested that, in healthy subjects, increasing wakefulness and the circadian low-point are linked to difficulties awakening and a lack of feeling refreshed in spite of good subjective sleep (Akerstedt et al., 2002). Furthermore, upon awakening, people with insomnia may excessively monitor their bodily sensations for signs of poor sleep (Harvey, 2002). Similarly, people with insomnia may excessively monitor their bodily signals for signs inconsistent with falling asleep around bed-time (Harvey, 2002). It has been suggested that "…there is not a single human physiological, subjective, or behavioural system which is unaffected by the process of falling asleep. And many of these profound changes occur before sleep actually begins." (Ogilvie, 2001).

Insomnia has been conceptualized as a 24-hour disorder of "hyperarousal" (Riemann et al., 2010; Bonnet and Arand, 2010), in which insomnia is thought to be maintained by daytime cognitive processes (Harvey, 2002). As such, it seems important to understand how daytime changes in emotionality may manifest in normal sleepers. In particular, emotion recognition is important for social functioning (Iarocci, Yager, and Elfers, 2007), and emotional experiences have been found to affect emotion labeling (Niedenthal, Halberstadt, and Innes-Ker, 1999). Such results appear to be in keeping with the model of Phillips et al. (2003) which suggests that emotion perception involves interactions of perception, mood, and appraisal of a stimulus. Moreover, human observers have a tendency to infer socially-relevant information from facial cues. These cues are derived from the variant and invariant properties of faces, and form the basis of judgments as to the gender, identity, and emotional state of others (Haxby, Hoffman, and Gobbini, 2000).

The human facial physiology is capable of depicting a number of different facial movements, or "action units", which form the basis of facial expressions of emotion (e.g. Ekman and Friesen, 1976) which are then recognized by others. Furthermore, facial expressions are recognized categorically, rather than on a continuum, and emotional blends of two emotions tend to be recognized as expressing one emotion or other, with a clear point of demarcation (e.g. Young et al., 1997). The context of emotional expressions also affects their categorization (Righart and de Gelder, 2008; Aviezer et al., 2008), suggesting that cognition contributes towards an individuals's processing of emotional expressions.

In order to assess emotional sensitivity towards facial stimuli, ambiguous facial stimuli were presented to subjects, by varying the intensity of displayed emotion. These stimuli are based on facial Action Unit (AU) movements, which are responsible for expressing an emotion (Ekman and Friesen, 1976), thus providing a naturalistic model of emotion expression formation in real life (see, e.g. Adolphs, 2002). Moreover, dynamic images may be linked to increased arousal ratings (Sato and Yoshikawa, 2007). The emotions used are also consistent with prior research into psychopathology, in which the recognition of subtle expressions of emotion has often been assessed (e.g. Gilboa-Schechtman, Erhard-Weiss, and Jeczemien, 2002; Joorman and Gotlib, 2006; LeMoult et al., 2009; Langenecker et al., 2005; Beevers et al., 2009; Gilboa-Schectman et al., 2008; Surguladze et al., 2004). These tasks involved viewing ambiguous facial movements, then categorizing the emotion and rating its intensity. Subjects completed the emotion task early in the day, and later on in the day, at around twelve hours since waking in Experiment One and fifteen hours since waking in Experiment Two. Emotional sensitivity was hypothesized to be affected by time since waking.

Experiment One

Methods

Participants

In total, 8 male and 8 female subjects took part. Ages ranged from 18 – 28, with a mean of 21.06 (*SD* = 3.04), and all subjects were Western Caucasian with minimal experience with other-race cultures. All subjects reported keeping in good physical and mental health, with no current use of recreational drugs. No subjects showed any evidence of narcolepsy, sleep breathing disorder, periodic limb movements or restless legs syndrome, or circadian rhythm sleep disorder, and no subject reported an insomnia

problem. Participants took part in exchange for course credit or £15, and this study had the approval of the Faculty Ethics Committee.

Exclusion criteria

- The Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989) was used to exclude poor sleepers, identified as scores > 6.
- The Insomnia Severity Index (ISI; Bastien et al., 2001) was used to exclude any subjects with significant evidence of insomnia symptoms, with scores > 14. No subject reported an insomnia problem.
- The Morningness-Eveningness Questionnaire (MEQ; Horne and Ostberg, 1976) was used to exclude those subjects with an extreme diurnal preference were excluded, i.e. scores < 31 or > 69.
- The Hospital Anxiety and Depression Scale (HADS; Zigmond and Snaith, 1983) was used to exclude those with significant evidence of subjective distress, defined as scores on either of the subcomponents > 10.

Pre-test questionnaires

The University of Glasgow Sleep Centre (UGSC) screening interview (with supplemental algorithm, Wilson et al., 2006), was used to provide a preliminary assessment of the sleep of subjects, and to identify whether any sleep disorders were present. Experience of other cultures was assessed via the 5-item questionnaire developed by Jack et al. (2009). These questions formed the basis of a semi-structured interview which was used to exclude any subjects who might have significant experience of other cultures. In order to individualize testing times according to each subjects's sleep patterns, the Sleep Timing Questionnaire (STQ; Monk et al., 2003) was used, which was developed to assess sleep scheduling. Participants are asked about their typical, earliest, and latest sleep times. These were recorded for both weekdays and free-days, and for mornings and evenings. This measure was used to gauge sleep patterns, and to aid in the agreement of individualized testing times and rising times for each subject.

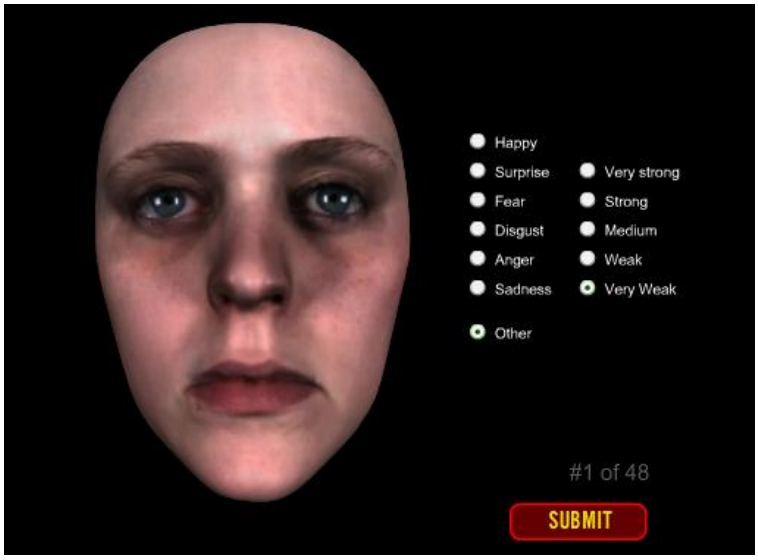
State questionnaires

- The Karolinska Sleepiness Scale (KSS; Akerstedt and Gillberg, 1990).
- The Daytime Impact of Sleep Scale (DISS; Buysse et al., 2007).
- The consensus sleep diary (Carney et al., 2012).

Facial Stimuli

The platform used to generate the facial stimuli is described in detail by Yu, Garrod and Schyns, (2012), and described previously in Chapter Three. Each facial animation was generated on the basis of six parameters, which corresponded to peak amplitude, peak latency, onset latency, offset latency, acceleration, and deceleration of facial action units (AUs). These values were selected for each AU pseudo-randomly, creating 1200 facial animations, which were shown on eight individual facial identities (four male and four female).

Figure 10. Example dynamic face stimuli



Procedure

Participants were recruited via the School of Psychology subject pool, and those subjects who expressed an interest completed the UGSC screening interview. Normal sleepers were recruited to take part in this study, and the STQ was used to guide the scheduling of test times at 3 hours or 12 hours since wake. This created early or late test groups, in a between-subjects design. As testing took place in the laboratory in order to control for test conditions, twelve hours since waking was selected as the late time period for ethical reasons relating to time of testing. Participants were asked to keep a regular sleep schedule during testing, based on usual rise times.

Due to the numbers of trials involved, testing took place over three or four consecutive weekdays, with testing scheduled for the same time each day. Dynamic expressive faces were shown to subjects over these days, with participants responding to each face by selecting emotion labels (anger, fear, happy, sad, disgust, surprise, and unsure), and

intensity judgements. Each animation was viewed by participants at 68 cm, before being categorized with the 6 emotion labels (and unsure). The perceived intensity of the emotion was judged on a scale of 1-5. Participants completed state measures after each test day, with the remaining questionnaires administered upon completion.

The data were analyzed as per Jack et al. (2012), and reverse correlation techniques were applied to create models of emotion for each observer. These were based on the six temporal parameters, of acceleration, peak amplitude, and deceleration, as well as onset latency, offset latency, and peak latency. In an initial step, emotion category responses labels were identified which were associated with the facial AUs. Parametric tests were used in keeping with Howell (2002). The local Ethics Committee approved this study.

Results

Firstly, the sleep diary parameters were averaged over test days for each subject.

Table 8. Sleep diary results in the sample

	Percentile 05	Percentile 25	Median	Percentile 75	Percentile 95	Mean	Std Dev
SOL (mins)	5.00	9.31	18.54	22.50	33.75	17.34	9.52
WASO (mins)	.00	.00	2.19	7.13	22.50	5.39	7.26
WAKE (#)	.00	.00	.50	1.25	4.33	.88	1.14
TST (hours)	6.44	6.81	7.67	8.28	8.63	7.55	.76
TiB (hours)	7.11	8.03	8.56	9.01	9.92	8.53	.83
SE (%)	63.53	78.00	86.71	95.25	100.00	85.90	10.74
QUAL (0-3)	1.00	1.75	2.25	2.83	3.00	2.17	.68
REST (0-4)	1.50	2.00	2.50	3.00	3.67	2.54	.57

Abbreviations: sleep onset latency (SOL), wake time after sleep onset (WASO), number of night-time awakenings (WAKE), total sleep time (TST), time in bed (TIB), sleep efficiency (SE), restful/restorativeness (REST), quality (QUAL).

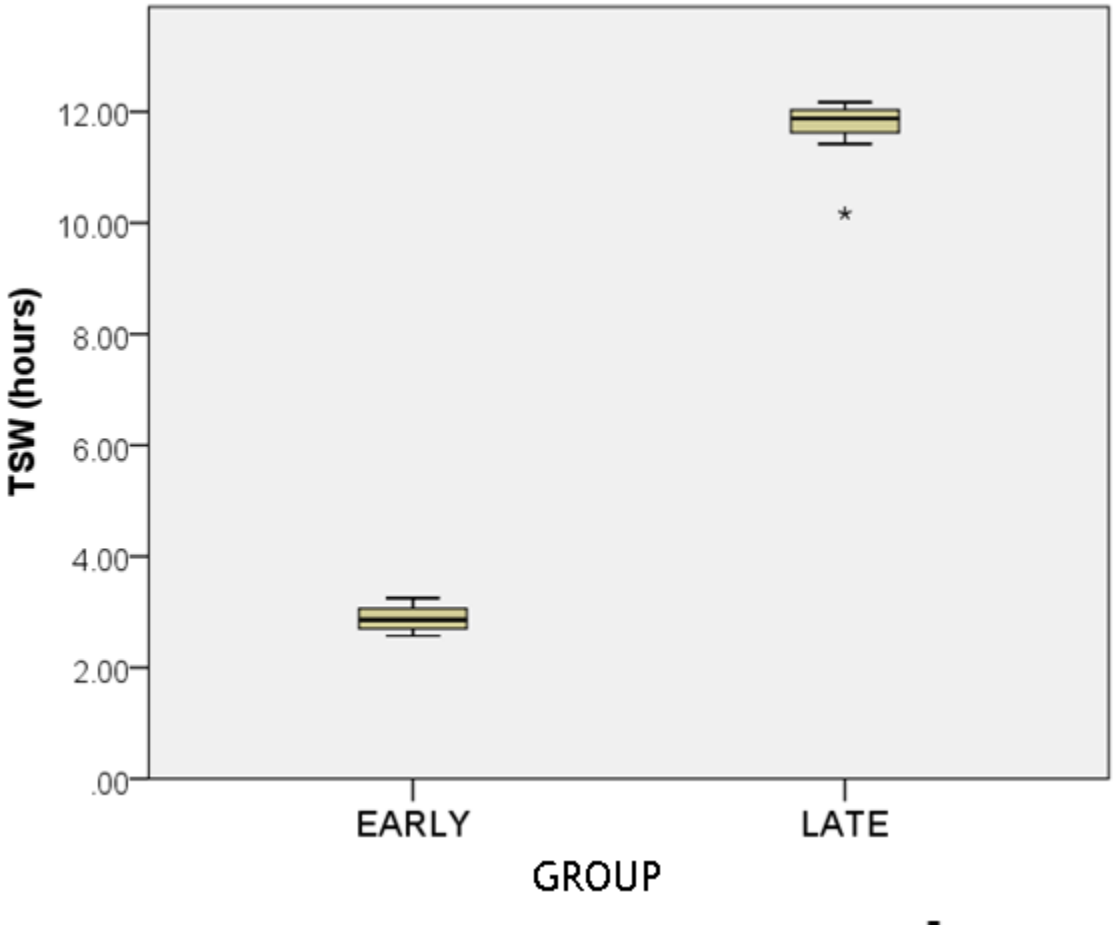
Results suggest that the sleep of the sample was good, with a short sleep onset latency and brief wake time after sleep onset, and few night-time awakenings. The total sleep time of the sample was also good, at over six hours, and subjects tended to report that their sleep was restful and of good quality.

Manipulation check

1) In an initial step, we confirmed the presence of the intended group differences on time since waking (TSW). Sleep diaries on test days were used to calculate the

difference between wake time and test time, and mean scores were then calculated for each individual. Results confirmed that testing took place at the intended TSW in the early group (M = 3.76, SD = 2.50), and in the late group (M = 11.68, SD = 0.65), with significant group differences (T (14) = - 8.67, p < 0.001).

Figure 11. Time since waking between groups



Groups tested early and late were tested at different times since waking. The '*' indicates a data outlier. Abbreviations: Time Since Waking (TSW).

2) We then compared subjects on subjective sleepiness, with scores averaged over test days. However, there were no significant group differences on average sleepiness measured via KSS scores.

Table 9. Karolinska Sleepiness Scale (KSS) scores between groups

	GROUP						
	EARLY			LATE			p-value
	Mean	Median	Std Dev	Mean	Median	Std Dev	
KSS	4.31	4.38	1.37	4.79	4.63	1.64	0.54

Abbreviations: Karolinska Sleepiness Scale (KSS).

3) Groups were then compared on subjective mood, assessed via the components of the DISS. However, there were no significant group differences on any of the four subcomponents, of alert cognition, negative affect, positive affect, and sleepiness-fatigue.

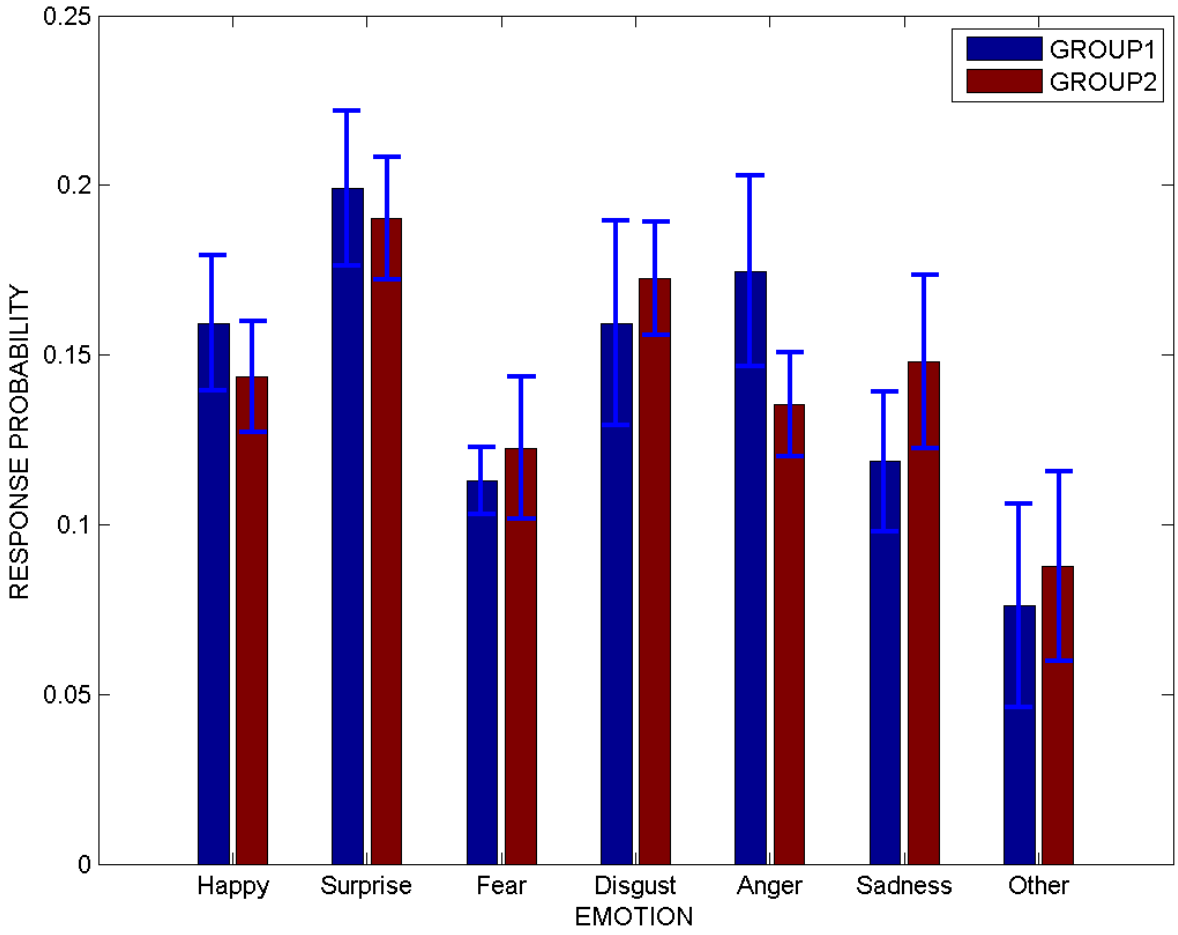
Table 10. Daytime Impact of Sleep Scale (DISS) scores between groups

	GROUP						
	EARLY			LATE			
	Mean	Median	Std Dev	Mean	Median	Std Dev	P-value
AC	62.13	59.89	11.45	66.24	65.38	19.77	0.62
NA	27.36	26.16	13.28	19.82	14.63	14.15	0.29
PA	58.85	56.70	7.39	68.62	72.22	17.09	0.16
SF	39.78	42.97	13.35	34.65	32.46	21.39	0.57

Abbreviations: Alert Cognition (AC); Negative Affect (NA); Positive Affect (PA); Sleepiness/Fatigue (SF).

These reports, taken together, indicate that the early and late groups were tested at significantly different times since waking, although this difference was not reflected in subjective sleepiness or mood.

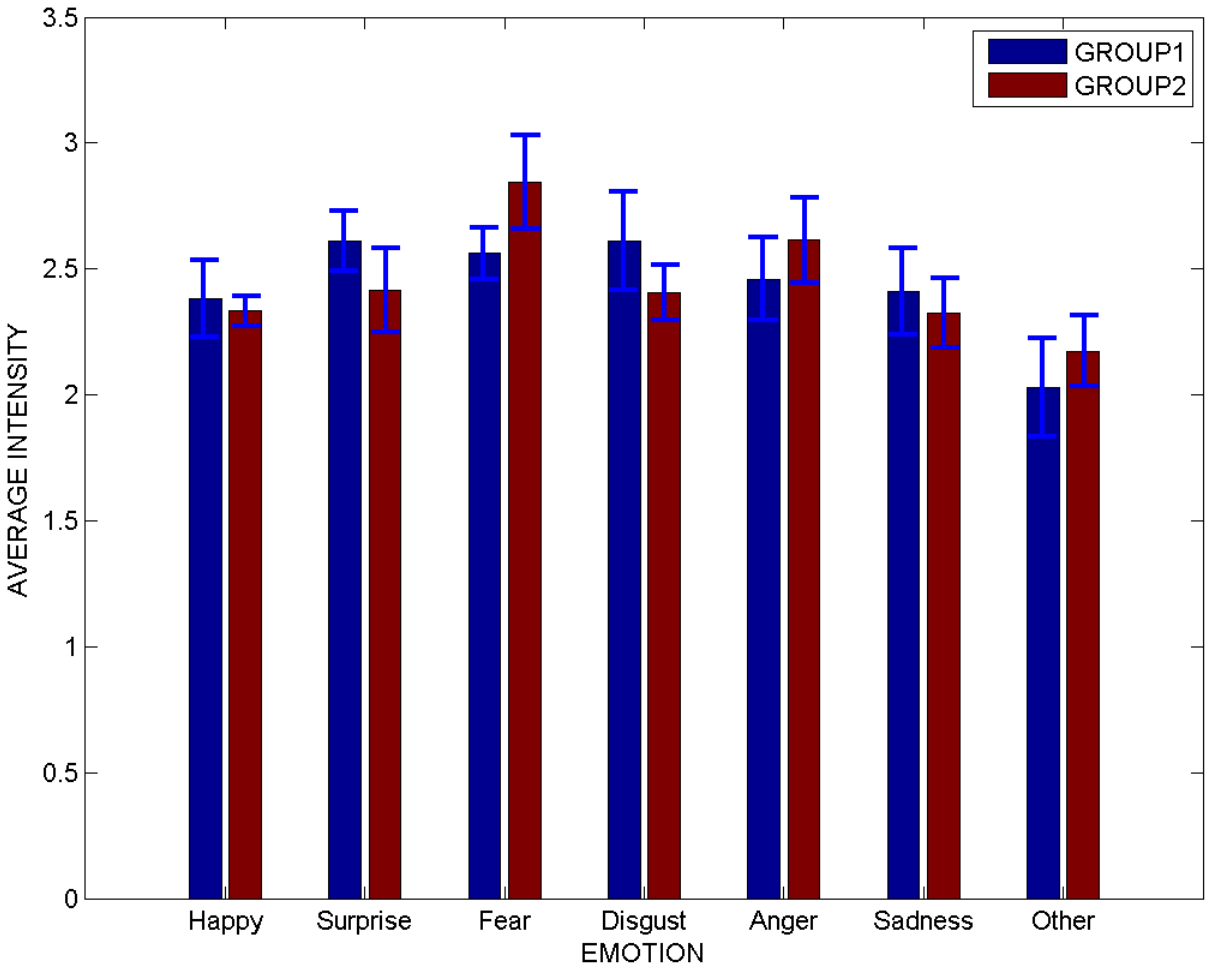
Figure 12. Response probabilities between groups. Group 1 corresponds to the early group and Group 2 corresponds to the late group.



Response probability refers to the likelihood of an emotional label being used.

The use of emotional labels and intensity judgements were compared between groups. However, there was no significant interaction of group with emotional expression (F (6, 98) = 0.52, p = 0.79), or main effect of group (F (1, 98) = 0.00, p = 1.00), although there was a main effect of emotional expression (F (6, 98) = 5.22, p < 0.001).

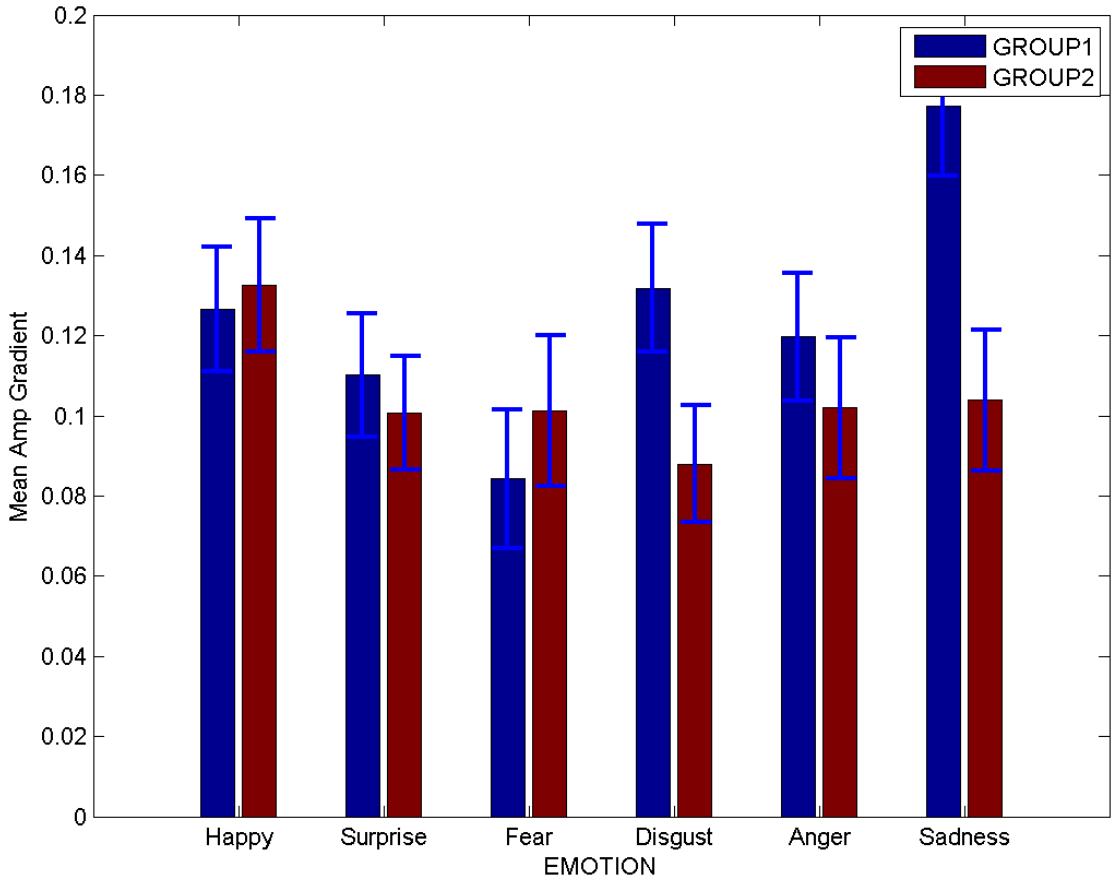
Figure 13. Average intensity judgements between groups. Group 1 corresponds to the early group and Group 2 corresponds to the late group.



Similar results were found with intensity judgements. There was no main effect of group (F (1, 98) = 0.01, p = 0.93), or interaction of group with emotional expression (F (6, 98) = 0.77, p = 0.59), although there was a main effect of emotional expression (F (6, 98) = 3.09, p < 0.01).

In a next step, reverse correlation techniques were used to generate cognitive models of emotion for each of the six temporal parameters and emotions.

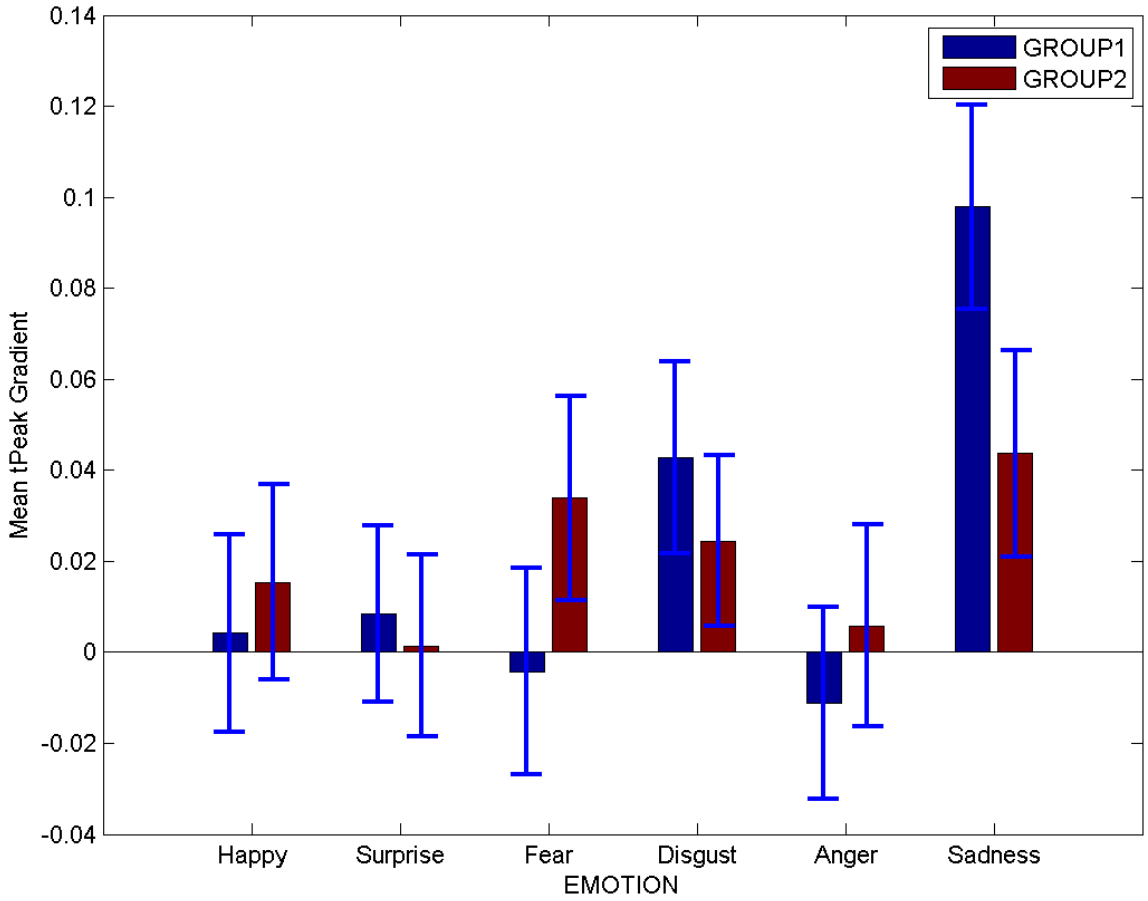
Figure 14. Average amplitude between groups. Group 1 corresponds to the early group and Group 2 corresponds to the late group.



Mean Amplitude Gradient (Amp Gradient) refers to the peak amplitude of the action unit.

For amplitude, there was a significant main effect for each of emotion (F (5, 3528) = 2.24, $p < 0.05$), action unit (F (41, 3528) = 7.43, $p < 0.001$) and group (F (1, 3528) = 4.76, $p < 0.05$). There was a significant two-way interaction of emotion and action unit (F (205, 3528) = 1.63, $p < 0.001$), but no significant interactions of emotion and group (F (5, 3528) = 2.01, $p = 0.07$) or action unit and group (F (41, 3528) = 0.66, $p = 0.95$). However, the three-way interaction was significant (F (205, 3528) = 1.29, $p < 0.005$).

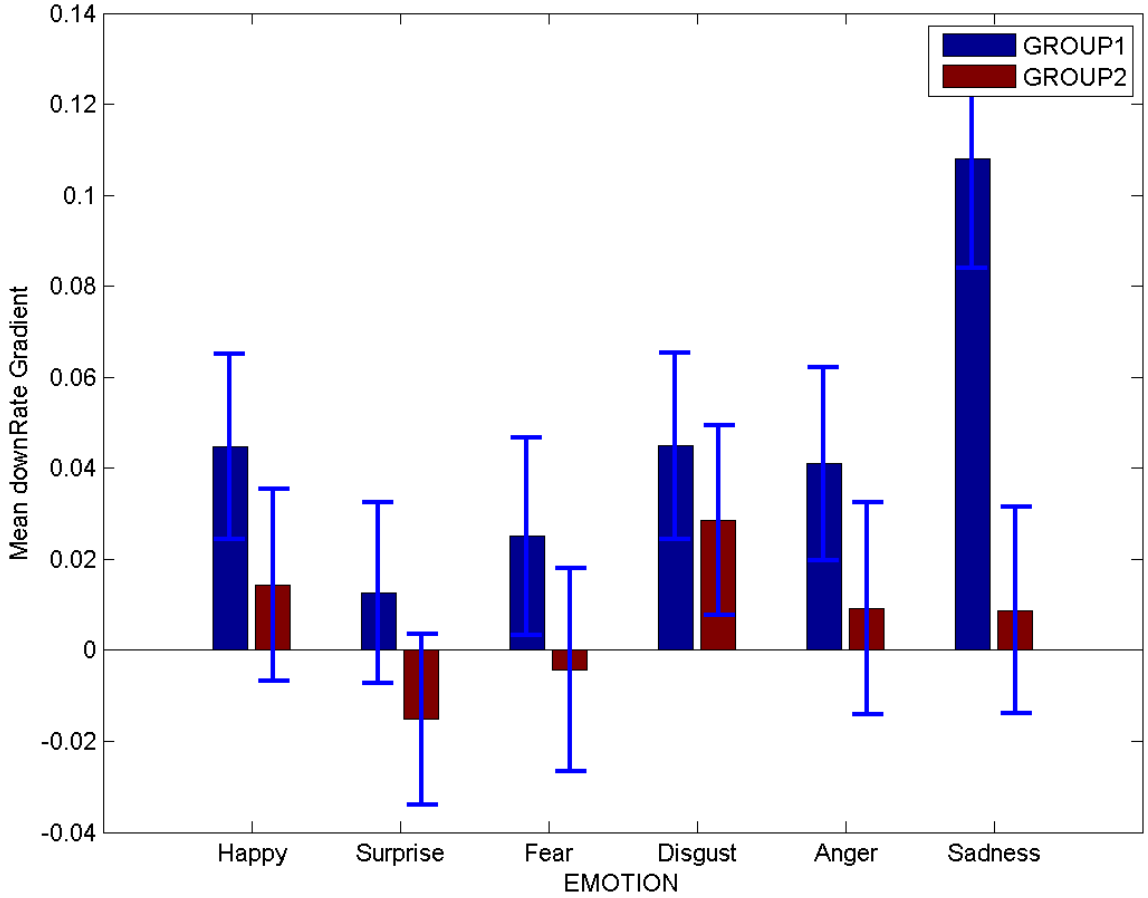
Figure 15. Average peak latency between groups. Group 1 corresponds to the early group and Group 2 corresponds to the late group.



Time to Peak Gradient (tPeak Gradient) refers to the time to the peak amplitude of the Action Unit.

For peak latency, there was a significant main effect of emotion (F (5, 3528) = 3.79, p = 0.002) and action unit (F (41, 3528) = 2.69, p < 0.001). There was no significant main effect of group (F (1, 3528) = 0.11, p = 0.74). The interaction of emotion and action unit was significant (F (205, 3528) = 2.19, p < 0.001), but there were no significant interactions of emotion and group (F (5, 3528) = 1.17, p = 0.32) or action unit and group (F (41, 3528) = 1.29, p = 0.10). The three-way interaction of emotion, action unit, and group was significant (F (205, 3528) = 1.18, p = 0.04).

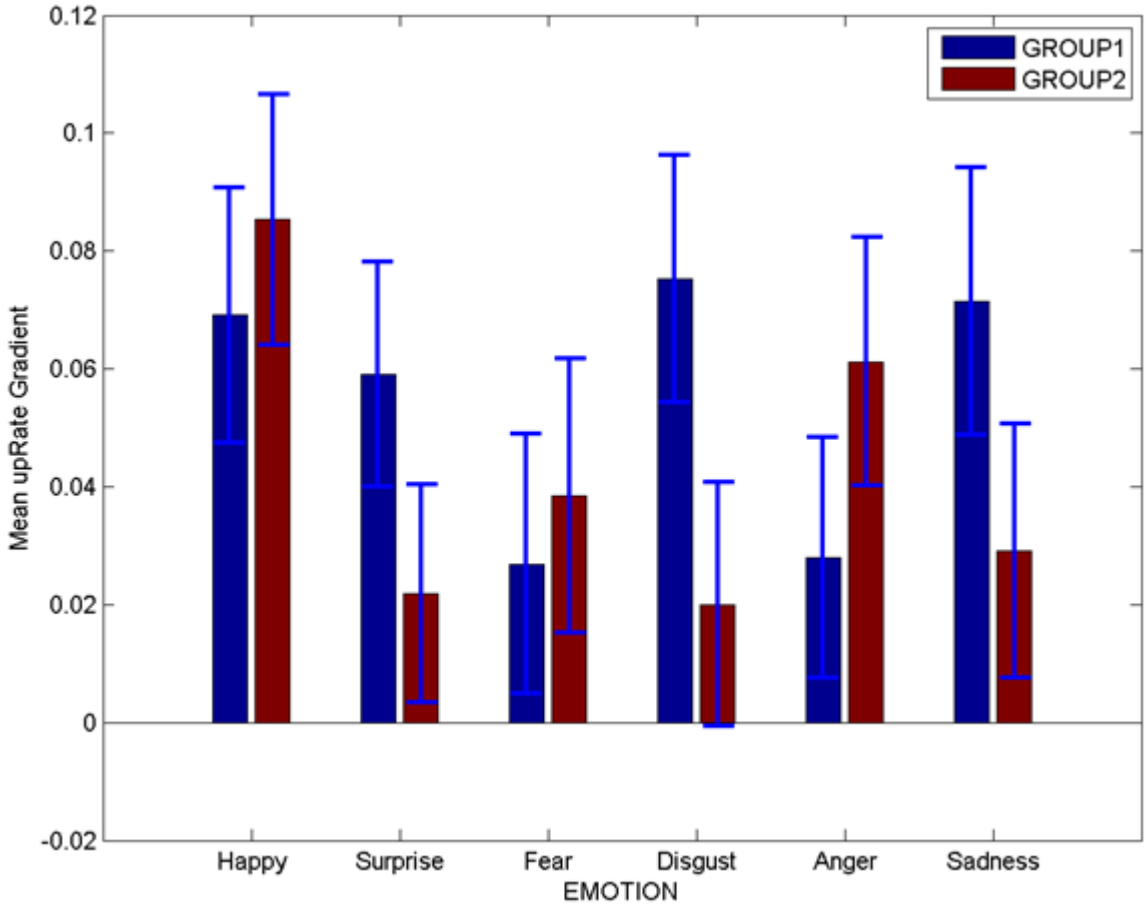
Figure 16. Average down rate between groups. Group 1 corresponds to the early group and Group 2 corresponds to the late group.



Down rate refers to the rate of deceleration of the Action Unit.

For down rate, or deceleration, there was a significant main effect for each of action unit (F (41, 3528) = 5.76, p < 0.001) and group (F (1, 3528) = 12.97, p < 0.005), but not emotion (F (5, 3528) = 2.00, p = 0.08). There was a significant two-way interaction of emotion and action unit (F (205, 3528) = 2.34, p < 0.001), but no significant interactions of emotion and group (F (5, 3528) = 0.89, p = 0.49) or action unit and group (F (41, 3528) = 0.81, p = 0.80). The three-way interaction was not significant (F (205, 3528) = 1.08, p = 0.22).

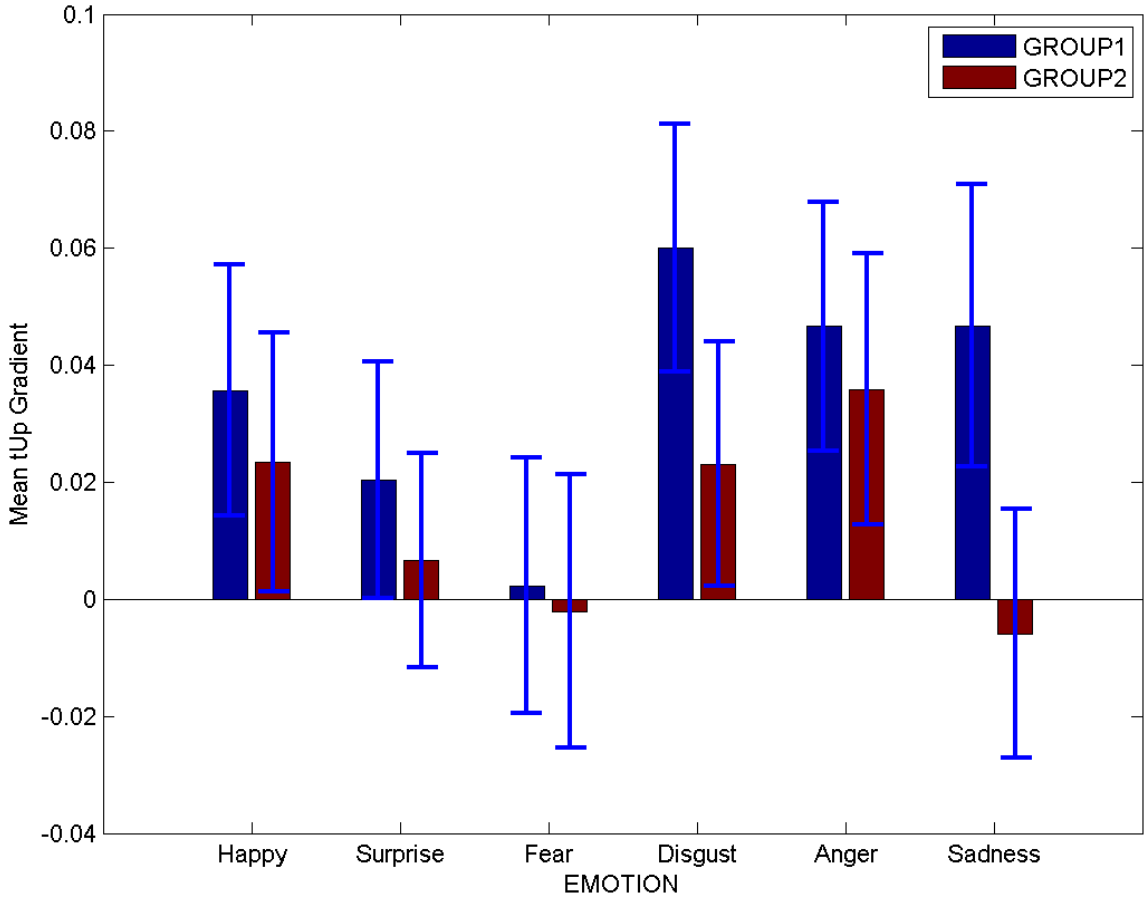
Figure 17. Average up rate between groups. Group 1 corresponds to the early group and Group 2 corresponds to the late group.



Up rate refers to the acceleration of the Action Unit.

For up rate, or acceleration, there was a significant main effect of action unit (F (41, 3528) = 2.09, p < 0.001). The main effects of emotion (F (5, 3528) = 0.89, p = 0.49) and group (F (1, 3528) = 1.66, p = 0.20) were not significant. The interaction of emotion and action unit was significant (F (205, 3528) = 1.80, p < 0.001), but there were no significant interactions of emotion and group (F (5, 3528) = 1.10, p = 0.36) or action unit and group (F (41, 3528) = 0.87, p = 0.71). The three-way interaction of emotion, action unit, and group was not significant (F (205, 3528) = 0.93, p = 0.74).

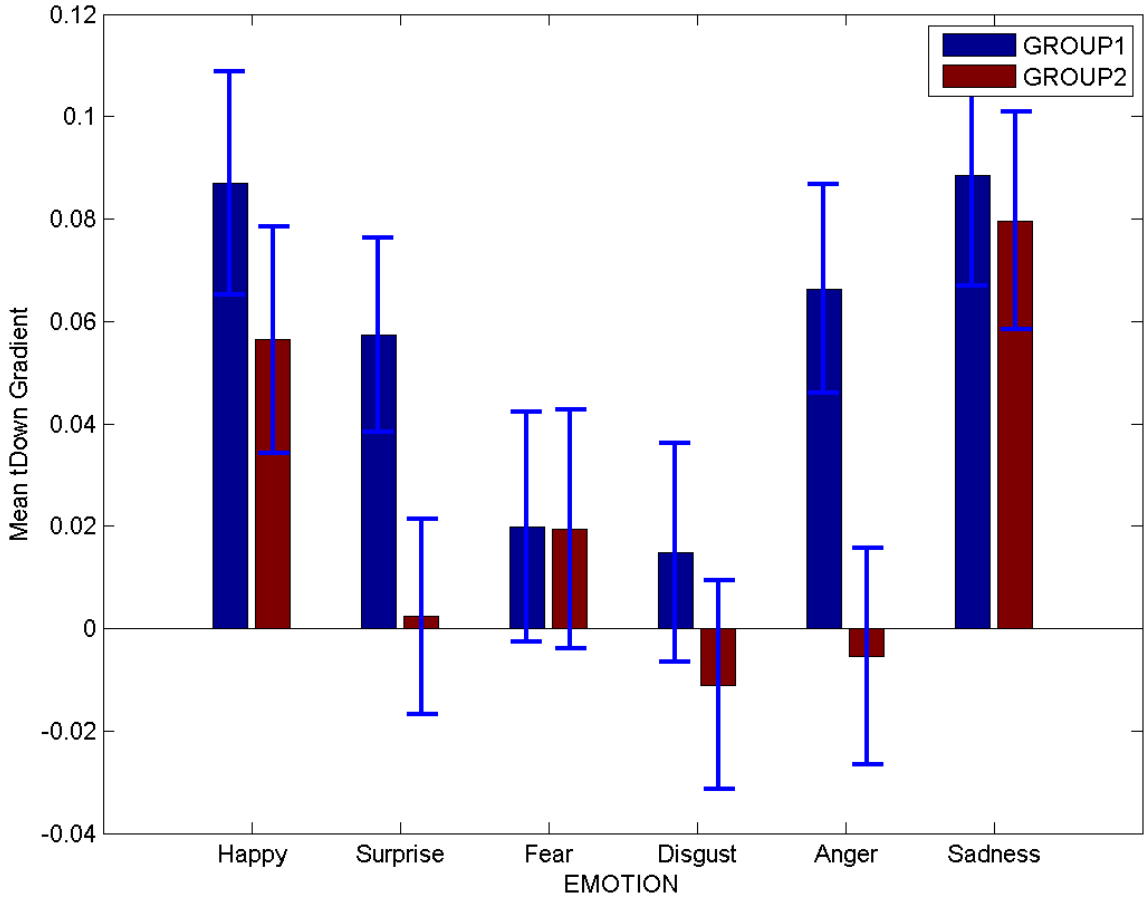
Figure 18a. Average onset latency between groups. Group 1 corresponds to the early group and Group 2 corresponds to the late group.



Mean time to up gradient (tUp Gradient) refers to the onset latency of the Action Unit.

For onset latency, there was no significant effect of emotion (F (5, 3528) = 1.41, p = 0.22) , but there were significant effects of group (F (1, 3528) = 3.81, p = 0.06), and action unit (F (41, 3528) = 4.77, p < 0.001). The interaction of emotion and action unit was significant (F (205, 3528) = 2.05, p < 0.001), but there were no significant interactions of emotion with group (F (5, 3528) = 0.85, p = 0.52) or action unit with group (F (41, 3528) = 0.61, p = 0.98). The three-way interaction of emotion, action unit, and group was significant (F (205, 3528) = 1.05, p = 0.31).

Figure 18b. Average offset latency between groups. Group 1 corresponds to the early group and Group 2 corresponds to the late group.



Mean tDown Gradient refers to the offset latency of the Action Unit.

For offset latency, there was a significant main effect of expression (F (5, 3528) = 4.60, $p < 0.001$), action unit (F (41, 3528) = 3.15, $p < 0.001$), and group (F (1, 3528) = 9.29, $p = 0.002$). The interaction of emotion and action unit was significant (F (205, 3528) = 2.19, $p < 0.001$), but there were no significant interactions of emotion with group (F (5, 3528) = 0.19, $p = 0.48$) or action unit with group (F (41, 3528) = 0.67, $p = 0.94$). The three-way interaction was significant (F (205, 3528) = 1.30, $p = 0.004$).

However, it should be noted that follow-up tests adjusting for multiple comparisons (Bonferroni) were not significant for any of these six temporal parameters.

Experiment One Summary

Results suggest that testing took place at significantly different times since waking, with the early group tested on average at three hours since waking and the late group tested on average at twelve hours since waking. However, this difference was not reflected in subjective sleepiness or mood. There were no significant group differences in sleepiness, alert cognition, positive affect, negative affect, or sleepiness/fatigue. With regards to the experimental task with dynamic emotional faces, there were no significant group differences in the emotional categorization or intensity labels used. However, there were significant group differences with regards to several of the temporal parameters which formed the dynamic stimuli. These results are shown in a summary table below.

Table 11. Summary of temporal parameter results by emotion, action unit and group

	Emotion	Action Unit	Group	Emotion x Action Unit	Emotion x Group	Action Unit x Group	Emotion x Action Unit x Group
Amplitude	Sig	Sig	Sig	Sig	N.S.	N.S.	Sig
Peak Latency	Sig	Sig	N.S.	Sig	N.S.	N.S.	Sig
Up Rate	N.S.	Sig	N.S.	Sig	N.S.	N.S.	N.S.
Down Rate	N.S.	Sig	Sig	Sig	N.S.	N.S.	N.S.
Onset Latency	N.S.	Sig	Sig	Sig	N.S.	N.S.	N.S.
Offset Latency	Sig	Sig	Sig	Sig	N.S.	N.S.	Sig

There were significant effects of group with regards to amplitude, down rate, onset latency, and offset latency. There were also significant three-way interactions with amplitude, peak latency, and offset latency. In general, the early group evidenced greater emotional sensitivity as shown by higher scores, and these results are summarized below. Descriptively, the early group were consistently more sensitive than the late group to surprise, disgust, and sadness across all six temporal parameters. The early group were also consistently more sensitive than the late group with regards to the temporal parameters of down rate, onset latency, and offset latency, for all six

emotional expressions.

Experiment Two

Methods

Participants

Participants had a mean age 23.22 (*SD* 2.9; range 20-29), and seven subjects were female with one male subject. Subjects reported good general health, with no evidence of any sleep disorder (i.e., narcolepsy, sleep-related breathing disorder, periodic limb movements or restless legs syndrome, circadian rhythm sleep disorder, or parasomnias). All subjects were Western Caucasian. In exchange for participation subjects were paid £20, and this study had the approval of the local Ethics Committee.

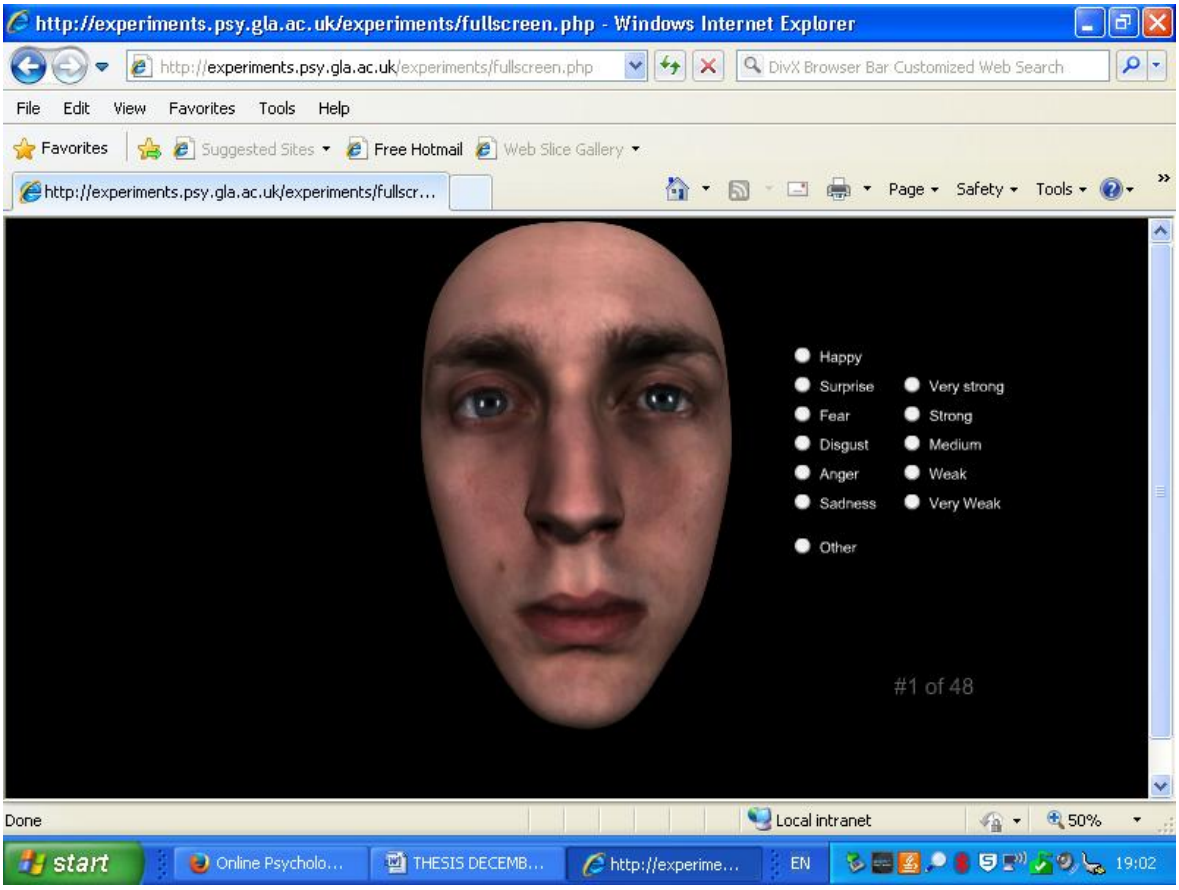
In addition, no subjects met the following exclusion criteria:

- Morningness-Eveningness Questionnaire (Horne and Ostberg, 1976) scores < 31, or > 69, indicating an extreme preference.
- Insomnia Severity Index (Bastien, Vallieres, and Morin, 2001) scores > 14.
- Hospital Anxiety and Depression Scale (Zigmond and Snaith, 1983) scores > 10 on either subcomponent, indicating clinically significant distress.
- Positive endorsement of "Do you have an insomnia complaint?"
- Pittsburgh Sleep Quality Index (Buysse et al., 1989) scores > 6.
- Toronto Alexithymia Scale (Bagby, Parker and Taylor, 1994) scores > 60.

Stimuli

The platform used to generate the facial stimuli is described in detail by Yu, Garrod and Schyns (2012). Dynamic expressions were shown on four male and four female identities, and displayed the six emotions (anger, fear, happy, sad, disgust, surprise, and happy), at 15 levels of intensity. Emotion expressions were presented in three sessions of 240 trials (720 in total), and each test session contained roughly the same distributions of intensities across all identities and emotions. This resulted in five blocks of 48 trials per session, with eight identities by six emotions. Example stimimii are shown below.

Figure 19. Example stimuli



Questionnaires

Participants completed the PSQI and ISI, as well as the MEQ, HADS, STQ, TAS-20, alongside demographic information. The state measures of the KSS and DISS were given after each task, as well as a sleep diary for the test day (Carney et al., 2012). Participants completed an additional 7-day sleep diary before testing, and in the week between the two test sessions.

Procedure

Potential subjects completed brief pre-test questions to ensure their status as normal sleepers. The study protocol is described in the figure below. After an initial meeting, where subjects were given actiwatches to wear on their non-dominant hand, subjects completed a 7-day sleep diary. This information was used to guide the scheduling of a 2-hour 'task window' at approximately three hours or fifteen hours since waking; times which were subsequently validated with task day sleep diaries. Actiwatches provided an additional measure of sleep scheduling, and were used record activity levels as an objective measure of sleep patterns. Half of the subjects completed the late session

first and the early session second in order to control for order effects, in a within subjects design. Participants completed the emotion task and state measures over three test sessions, followed by a 7-day sleep diary, and then the final emotion task. The emotions, identities, and intensity levels of expressions were counterbalanced across test days, and repeated at the second task. The emotion task consisted of viewing the facial display of emotion and labeling the emotion shown, from seven options, and rating its intensity. Intensity was judged via a five-point scale, anchored at 'very strong' and 'very weak'. Participants completed the study online from their own home, and were instructed to complete the task in a quiet room free from distractions, with the computer screen an arms's length away. Participants then returned the actiwatches to the researcher, and completed the final questionnaires before being debriefed. Parametric test were used for the analysis (Howell, 2002) and effect sizes are reported in Appendix 2.

Results

Actigraphy data

In a first step, actigraphy data were averaged over test days for each individual participant. Data was unavailable for one subject due to non-adherence, and 50% of the individual subject averages were calculated over the six test days.

Table 12. Summary of actigraphy scores in the sample

	Median	Mean	Std Dev
SOL (mins)	25.00	28.51	17.25
WASO (mins)	61.00	60.63	29.59
WAKE (#)	30.17	26.36	8.59
TST (hours)	5.95	6.40	1.01
TiB (hours)	8.28	8.02	1.23
SE (%)	77.19	78.24	8.45

Abbreviations: sleep onset latency (SOL), wake time after sleep onset (WASO), number of night-time awakenings (WAKE), total sleep time (TST), time in bed (TIB), sleep efficiency (SE).

Results from actigraphy suggest that participants fell asleep within thirty minutes and were awake for around thirty minutes during the night, with several night-time awakenings. The total sleep time of participants was around six hours or greater, with at least eight hours in bed. The sleep efficiency of participants was close to 80%.

Manipulation check

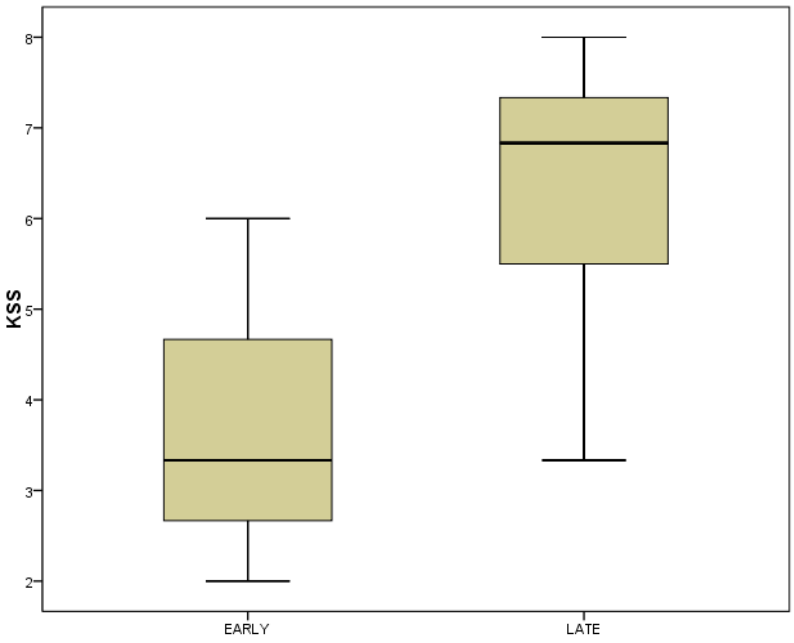
1) TSW

Results suggest significant differences between the early and late groups on time since waking (*T* (7) = - 16.28, *P* < 0.001). The testing of the early group took place on average at 3:04 (*SD* 01:33), and the testing of the late group took place on average at 14:55 (*SD* 00:56).

Figure 20. Time since waking (TSW) between groups.

2) Sleepiness

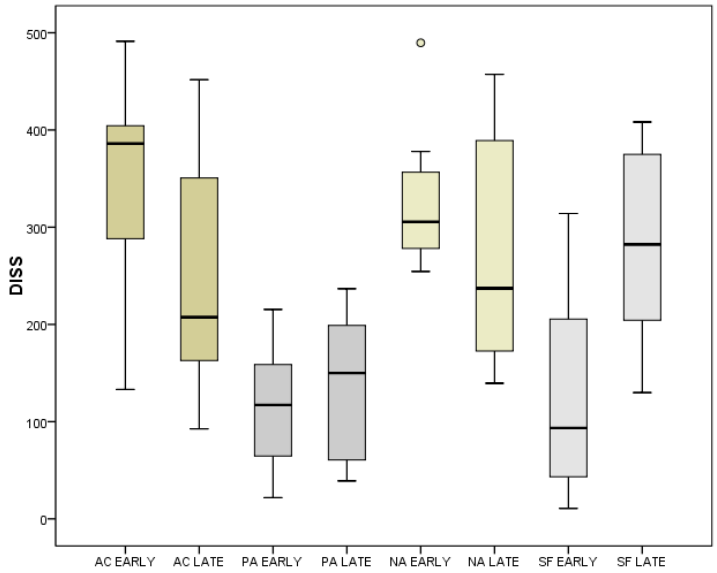
Figure 21. Karolinska Sleepiness Scale (KSS) scores between groups.



Results suggest significant differences between groups in subjective sleepiness (*T* (7) = - 2.89, *p* = 0.02).

3) Subjective mood

Figure 22. Daytime Impact of Sleep Scale (DISS) scores between groups



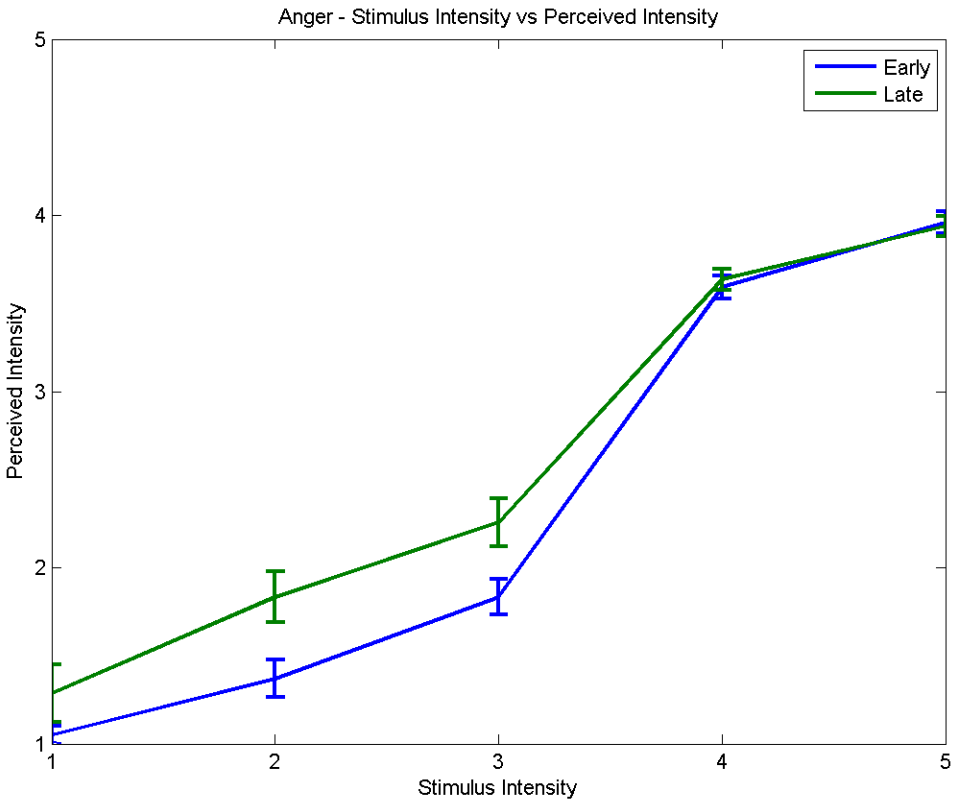
Abbreviations: Alert Cognition (AC); Positive Affect (PA); Negative Affect (NA), Sleepiness/Fatigue (SF). 'o' refers to a suspected data outlier.

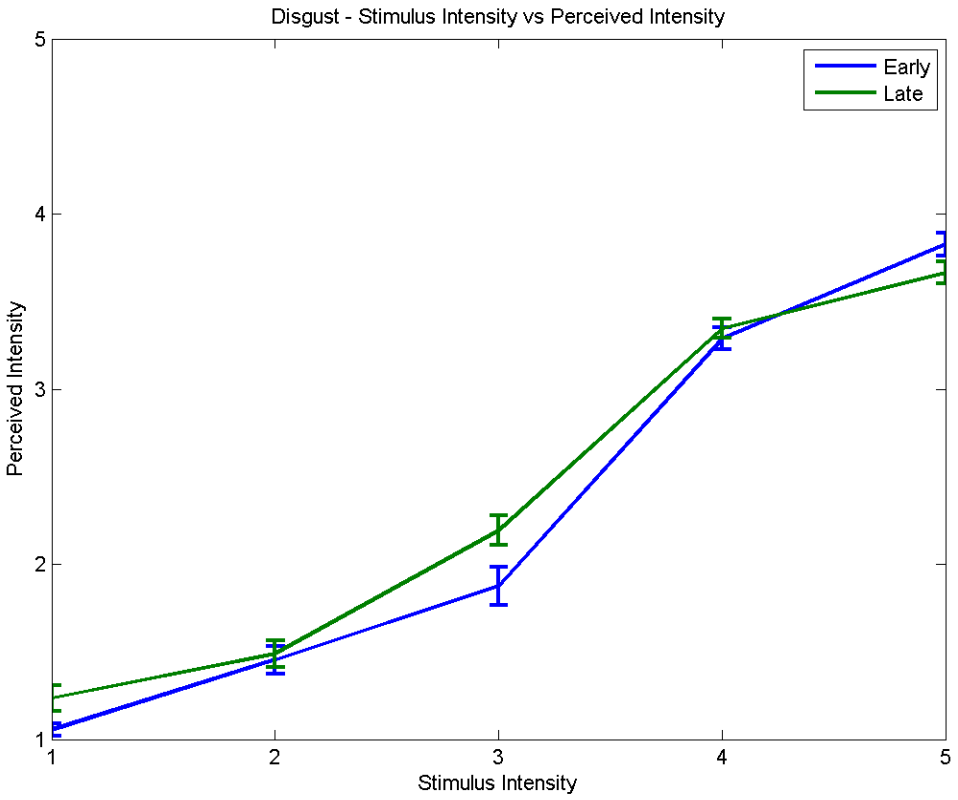
When sleep groups were compared on the DISS, there were significant group differences in AC (T (7) = 2.46, p, 0.05) and SF (T (7) = - 3.27, p < 0.05), with the late group reporting greater sleepiness/fatigue and less alertness. There were no significant group differences in PA (T (7) = - 0.81, p = 0.45) or NA (T (7) = 1.63, p = 0.15).

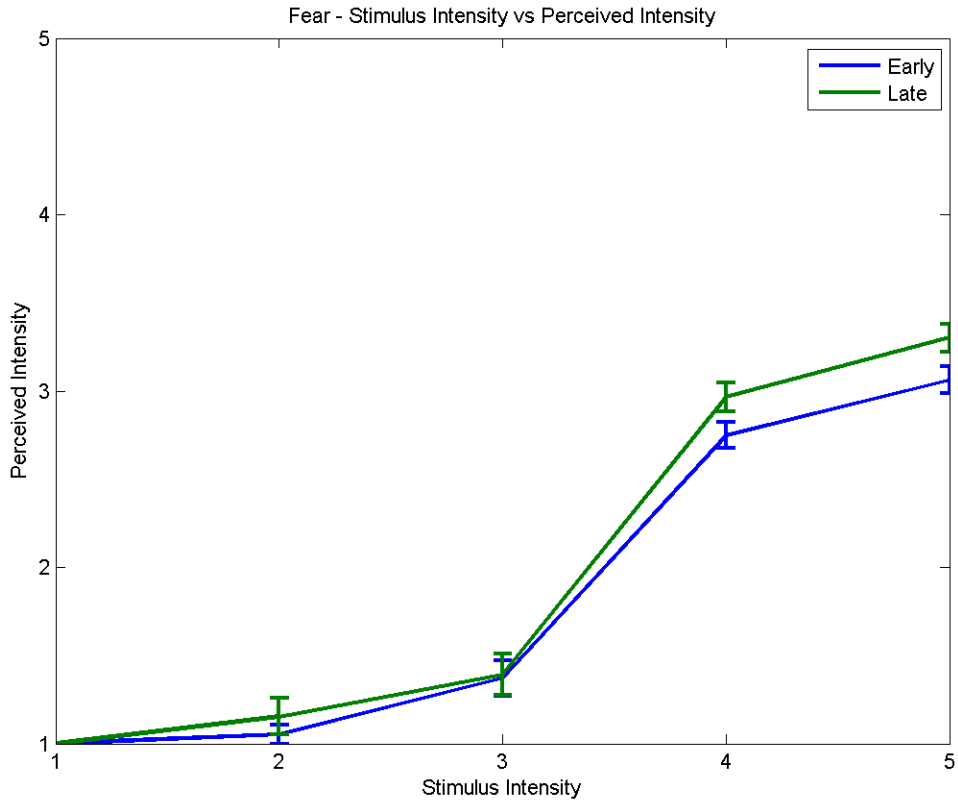
Task results

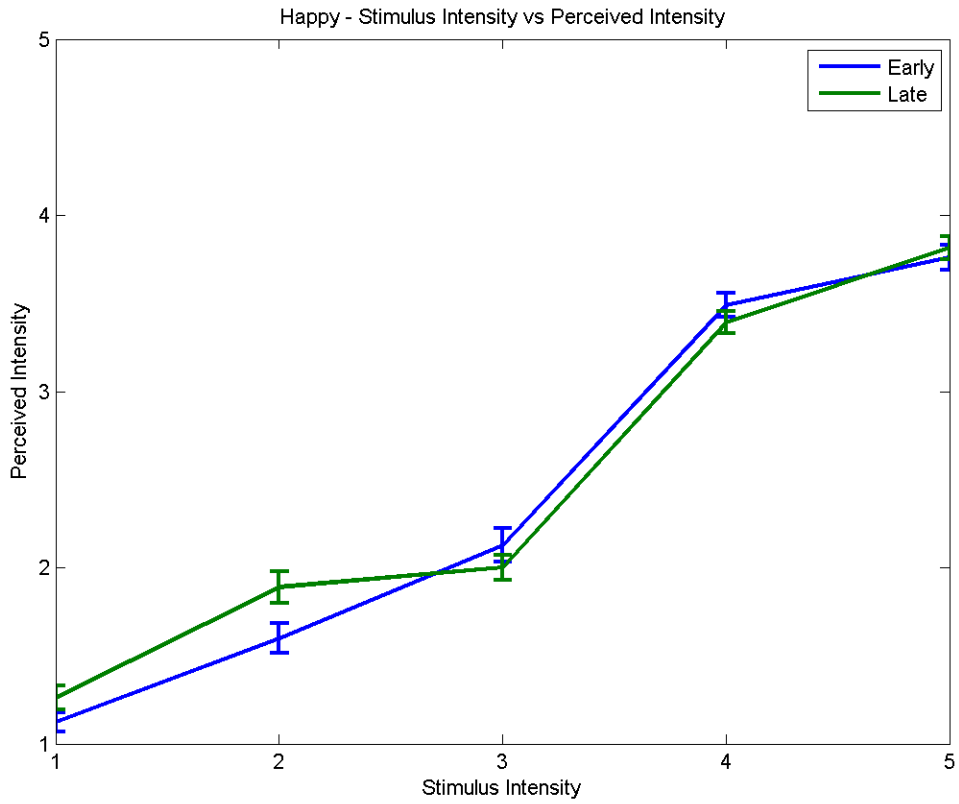
There were no significant group differences in categorization accuracy (z = -0.90, p = 0.37). We then compared groups on the intensity ratings of the emotional faces, with the fifteen levels of emotional intensity grouped into five stimuli intensity categories for the purpose of analysis. There were significant group effects with intensity ratings. In particular, there was a significant interaction of intensity and group (F (4, 6818) = 195.77, p < 0.001), emotion and group (F (5, 6818) = 6.63, p < 0.001), but no significant interaction of emotion and intensity (F (20, 6818) = 1.55, p = 0.06), or three-way interaction (F (20, 6818) = 0.87, p = 0.62). There were main effects of group (F (1, 6818) = 261.51, p < 0.001), intensity (F (4, 6818) = 189.56, p < 0.001), and emotion (F (5, 6818) = 3.21, p < 0.001).

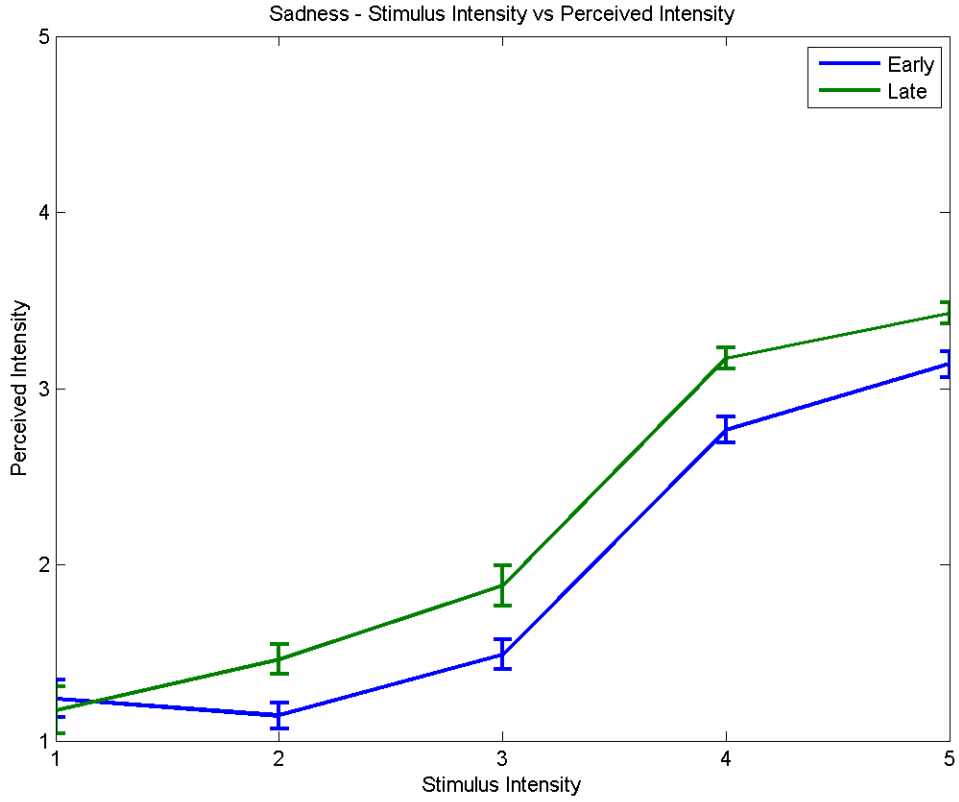
Figure 23. Perceived intensity of each of the six emotions between groups.

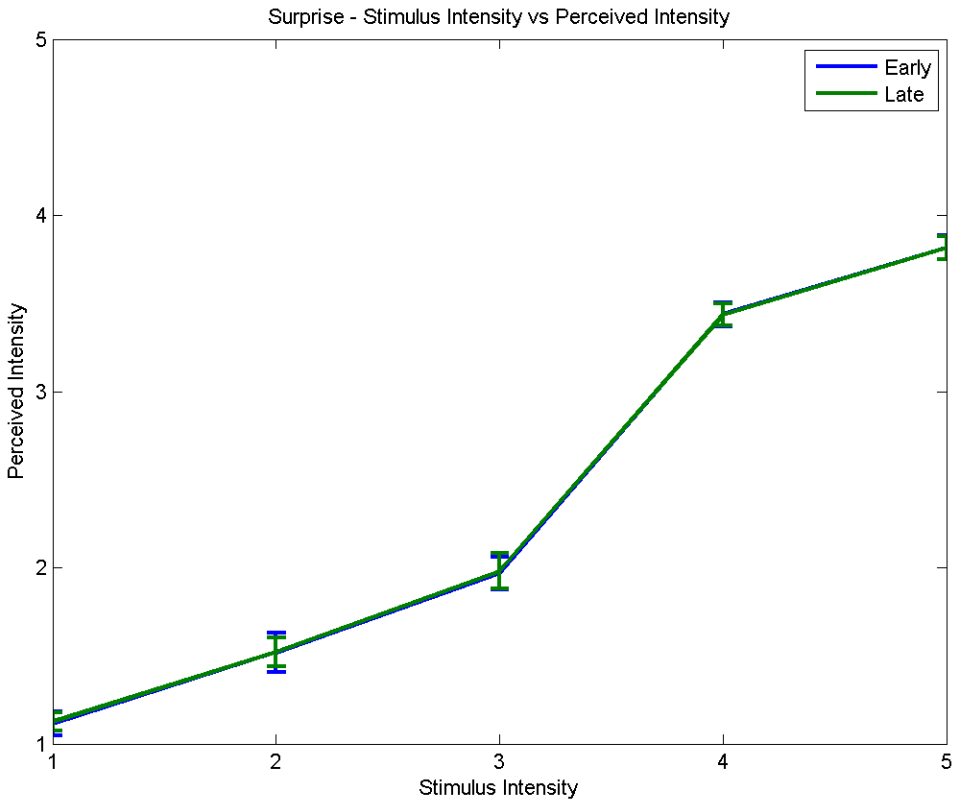



Disgust - Stimulus Intensity vs Perceived Intensity
Early
Late
Perceived Intensity
Stimulus Intensity
1
2
3
4
5


Fear - Stimulus Intensity vs Perceived Intensity
Early
Late
Perceived Intensity
Stimulus Intensity
1
2
3
4
5


Happy - Stimulus Intensity vs Perceived Intensity
Early
Late
Perceived Intensity
Stimulus Intensity
1
2
3
4
5


Sadness - Stimulus Intensity vs Perceived Intensity
Early
Late
Perceived Intensity
Stimulus Intensity
1
2
3
4
5



Follow-up t-test suggests that there are no significant differences with happy, surprise, fear or disgust (all $p > 0.01$), although there were significant group differences with anger, at intensity levels two and three (both $p = 0.01$), and with sad, at levels three ($p = 0.01$), four ($p < 0.001$) and five ($p < 0.005$). In particular, the late group made higher assessments of intensity. These comparions are the equivalent of significance at the level of 0.05/6 (= 0.01), and are consistent with the analysis in Chapter 4.

Experiment Two Summary

Results suggest that there were significant effects of group on time since waking. There were no significant effects on mood (positive affect or negative affect). However there were significant effects of group on sleepiness, alert cognition and sleepiness/fatigue. With regards to the emotion task, there were no significant effects of categorization accuracy with regards to the emotion labels, although there was a significant effect of accuracy with regards to the intensity labels. In particular, there was a significant main effect of group, and interactions of group with both emotion and intensity. The three-way interaction was not significant. This effect seems to be driven by significant group differences with anger at intensities two and three, and sad at intensities three, four and five. Results suggest that the late group were more sensitive for each of these comparisons.

Discussion

Overall, there were no significant effects of time since waking on emotional categorization in either of the two experiments, suggesting that time since waking does not affect emotional categorization, with dynamic faces. However, there were significant effects of group on the perceived intensity of emotional faces, when tested within-subjects. In particular, results suggest that the late group were more sensitive to emotionality in faces, with significant effects with angry and sad faces in the mid-intensity range. This suggests that intensity (expressed or rated) could perhaps be especially sensitive in assessing subject emotionality, a conclusion which is consistent with the results from Kyle et al. (2014), Van Der Helm et al. (2010), and results from the previous study. It has been suggested that intensity ratings follow on from emotional categorization, although manipulating the intensity of a stimulus increases the accuracy of recognition as well as intensity ratings (Utuma et al., 2009). There were also significant effects of group with the temporal parameters used for the 4D faces. In particular, there were significant effects with group for the peak latency and amplitude, the down rate, and the onset and offset latencies. However, there were no significant effects of group on emotional categorization or intensity labels in this task, and this discrepancy may be due to one of several factors.

Experiment One took place in the laboratory and was a between-subjects design, whereas Experiment Two took place online using a within-subjects design. This meant that there were significant differences between groups in the test environment. Conditioned arousal to the bedroom environment is thought to play a role in normal sleep and insomnia (Broomfield, Gumley and Espie, 2005; Espie, 2002). For example, sleep-onset insomnia patients have been found to show less de-arousal in the lead up to sleep when compared to controls (Robertson, Broomfield, and Espie, 2007), and the periods around awakening from sleep and going to bed may be especially relevant to people with insomnia (Harvey, 2002). Future studies should investigate the role of the test environment in order to assess whether this contributed towards the current results. However, it is possible that other uncontrollable differences in the testing environment contributed towards these results (e.g. ambient noise, lighting, location).

The use of online testing also meant that there were differences between studies with regards to the test times. The test times of early test sessions were similar, at close to four hours since waking in Experiment One and three hours since waking in Experiment Two. Proximity to waking is known to affect cognitive performance in the period immediately following awakening, a phenomenon known as sleep inertia. Sleep inertia

has typically been found to last 30 minutes after awakening, although performance can continue to improve for up to two hours (Tassi and Muzet, 2000). As both experiments tested subjects at around 3 or 4 hours since waking, it appears unlikely that sleep inertia effects were present in the early testing periods.

The late testing time of subjects was more dissimilar when comparing Experiments One and Two. In Experiment One, testing took place on average at around twelve hours since wake, whereas in Experiment Two testing took place at around fifteen hours since wake. This could be reflected in subject self-reports. In Experiment Two of this chapter, subjects reported significantly greater sleepiness and sleepiness/fatigue, and significantly less alertness, a result which suggests correspondence of subjective fatigue with performance. This would be in keeping with the suggestion of Carrier and Monk (2000) that alertness ratings can predict performance. The period of evening alertness, or wake maintenance zone, could also be relevant (Strongatz, Kronauer, and Czeisler, 1987). A recent study found performance on vigilance tasks to be significantly improved during the wake maintenance zone in comparison to earlier in the day (Shekleton et al., 2013). Furthermore, this period has been linked to the dim light melatonin onset, and those subjects who have crossed this threshold report significantly greater fatigue (Gorfine and Zisapel, 2009). It is possible that those subjects in Experiment Two were tested after their period of evening alertness. This would be consistent with the significant group differences in alertness and sleepiness/fatigue, but not the emotional task results. This would need to be tested further in additional studies which include physiological measurements.

In general, alertness is thought to be the result of interactions of sleep load and the circadian alerting signal (Cajochen, Blatter, and Wallach, 2004). Their interactions result in alertness increasing until the post-lunch period, with an additional evening peak in alertness which is followed by a decline in the lead-up to sleep (Cajochen, Blatter, and Wallach, 2004). Many studies have reported that cognitive performance mirrors core body temperature directly (Schmidt et al., 2007), especially when typical sleep/wake cycles are experimentally manipulated (Carrier and Monk, 2000). Forced desynchrony protocols can be used to identify the relative contributions of the circadian and homeostatic influences on performance (Schmidt et al., 2007), with body temperature a “gold standard” in the assessment of human circadian rhythms (Carrier and Monk, 2000), although other factors such as task difficulty contribute towards performance (Schmidt et al., 2007; Waterhouse, 2010). Simple monotonous tasks which are reliant on sustained attention and working memory are more affected by sleep loss than more complex tasks (Cajochen, Blatter, and Wallach, 2004). Time since waking has

been linked to prefrontal fatigue, whereas the circadian system is linked to a brain slowing (Cajochen, Blatter, and Wallach, 2004). In general the task time of Experiment Two was around 15 minutes, whereas Experiment One took longer to complete, at around 45 minutes. Performance rhythms seem to be affected by both the circadian timing system and time since waking independently, and alertness ratings may be able to predict performance as well as bodily temperature (Carrier and Monk, 2000).

A further relevant difference could relate to group differences in depression scores. In Experiment One, the depression scores of the early group were higher, with comparable scores between the late group and the participants taking part in Experiment Two. However, this factor seems unlikely to affect results as the scores of all participants were low, and below the threshold for subclinical levels of distress.

The early group in Experiment One were also more inclined towards eveningness, and a main effect of research in circadian rhythms in performance is congruency between diurnal preference and test times, with results superimposed upon alertness in general (Schmidt et al., 2007). In particular, extreme types are thought to be phase-shifted (Schmidt et al., 2007). However this factor was not thought to contribute towards results as extreme chronotypes were excluded, and test times of the remaining participants were individualized according to typical wake times.

Results suggest specific effects with regards to mid-intensity expressions of sadness and anger (Experiment Two). To summarize previous results, Chapter Three reported significant effects of sleep disruption the previous night, and depression, on happiness recognition. Chapter Four reported significant effects of insomnia on the recognition of high intensity expressions of sadness and low intensity expressions of surprise. In the current chapter (Chapter Five), Experiment One suggests that those subjects tested later tended to be less sensitive towards facial expressions, with regards to the temporal parameters of facial movements. Experiment Two suggests that those subjects tested later were more sensitive to emotional displays, with regards to mid-intensity expressions of sadness and anger. Results are thought to be a result of changes present at the late test session, as early test sessions are at comparable TSWs and beyond the point at which sleep inertia would be expected to be present.

With regards to how specific emotions are processed, Loughead et al. (2008) have looked at which brain regions were linked to the accuracy of identification of sadness, happiness, fear and anger. Healthy participants with a mean age of 27 viewed the emotion faces and neutral for 3 seconds, and were asked to indicate whether they

displayed the target or not. Greater activation of the amygdala for anger, and the thalamus, amygdala, inferior frontal regions, midfrontal regions and fusiform gyrus for fear, were found with correct identifications. Greater activation of the thalamus in particular was found with poorer recognition of happiness, and greater activation of the midfrontal regions were found with sadness recognition errors. In this task, bilateral frontal, amygdala, thalamus, fusiform gyrus and cuneus, cerebellum and right parahippocampal and angular gyri regions were active during the processing of all emotions, except the inferior frontal gyrus which did not respond in happy blocks. In another study, Blair et al. (1999) asked subjects to view sad and angry faces of varying intensity while making an incidental sex classification. They report increasing activation in the left amygdala, right temporal pole, right inferior temporal gyrus and right middle temporal gyrus with increasing intensity of sad expressions. With regards to angry faces, activations within the right orbitofrontal cortex (OFC) and anterior cingulate cortex (ACC) correlated with the increasing intensity of the angry faces.

The OFC has been linked to learning (Rolls, 2004), and the ACC has roles in emotional regulation and emotional appraisal (Etkin, Enger, and Kalisch, 2011), with a role of both in learning-based decision making (Rushworth et al., 2007). The amygdala has been found to contribute towards stress, emotion and fear (van Marle et al., 2009; Hartley and Phelps, 2010; Calder, Lawrence and Young, 2001; Adolphs, 2008). In general, the medial prefrontal cortex has been associated with behaviour, assessments of value, and self-reflection, giving it a role in social cognition (Amodio and Frith, 2006). Phillips et al. (2003) have reviewed the brain systems which are involved in emotion perception, in normal subjects and psychiatric illness. They implicate changes in the sensitivity of the amygdala and insula, ventrolateral prefrontal cortex, ventral anterior cingulate gyrus, and thalamus, ventral striatum, and the lability/regulation in interaction with the dorsolateral prefrontal cortex, dorsomedial prefrontal cortex, dorsal anterior cingulated gyrus, and hippocampus, as the neural structures involved in emotion perception. These areas are thought to be the neural substrates of emotion regulation, affective states, and the appraisal of emotion, and these processes are thought to be disrupted in psychiatric illness (Phillips et al., 2003). Future studies should investigate how sleep affects emotion recognition with regards to its brain correlates.

A possible limitation relates to the sample sizes in these two studies. However, differences in the tasks or groups are thought to be the most likely explanation for the current results. These sample sizes are also consistent with both psychophysics methods and the circadian literature. Moreover, the nature of the repeated sampling methods used made the testing of larger sample sizes impractical.

The current results may have been due to alterations in emotional sensitivity in the morning or in the evening, although changes in the evening are thought to be the most likely explanation. However, to assess how emotional sensitivity may change over the waking period we next assessed emotion recognition at four different times since waking. We also used the Ekman and Friesen (1976) faces to assess whether emotion recognition differences at different times since waking were attributable to the use of dynamic faces. In order to assess reaction time performance, we did not assess emotional intensity. However, performance was investigated more generally by the addition of emotional images and an object matching task.

Chapter Six

Emotion recognition in normal sleepers – is there a role of time since waking across the day?

Abstract

Sleep deprivation affects mood and emotional reactivity; results which have been supported by recent experimental paradigms. However, even within the waking day there are diurnal changes in mood and alertness, and how this may correspond to changes in emotional sensitivity is currently unclear. The current study aims to test how emotional sensitivity may change over the day, assessed via emotional categorization tasks. Subjective measures of mood and alertness were also used, in order to assess the correspondence of subjective reports with objective task performance. In a between-subjects design, participants completed a recognition task with emotional faces and images. A delayed-matching-to-sample task with everyday objects was also used as a visual comparison. Four time-since-waking groups were created, and these times ranged from roughly three hours since waking, to twelve hours since waking. No significant effects were found on emotional categorization with faces or images, or object recognition. Results suggest that there are no effects of time since waking on emotional categorization over the typical working day.

Emotion recognition in normal sleepers – is there a role of time since waking across the day?

Introduction

Good sleep is important to physical, mental, and emotional well-being (see, e.g., Baglioni, Spiegelhalder, Lombardo, and Riemann, 2010; Roth, 2007), and the adverse effects of stress on sleep are well known (Naidoo, 2009; Buckley and Schatzberg, 2005; Balbo, Leproult and Van Cauter, 2010). It is now well established that sleep disruption leads to changes in a person's mood state, which can amplify the effects of negative events and dampen the effects of positive events (e.g. Zohar et al., 2005). Similarly, sleep disruption before a traumatic event such as a motor vehicle accident increases the likelihood that a psychiatric illness will develop, even among trauma patients without a prior psychiatric disorder (Bryant et al., 2010). These previously healthy subjects have been found to be over three times more likely to develop a psychiatric disorder (Bryant et al., 2010). A previous history of insomnia has also been found to increase the likelihood of developing depression (Baglioni et al., 2011), further stressing the bidirectional relationships between sleep and mental health (Kahn, Sheppes, and Sadeh, 2013). As sleep disruption is common in the general population (see, e.g., Roth, 2007) it is therefore important to understand how sleep problems develop.

Recently, the effects of sleep loss on emotional task performance have been investigated. In such tasks, sleep deprivation has been found to affect responses to emotional images (e.g. Yoo et al., 2007) and faces (e.g. Van der Helm et al., 2010). These authors found that a lack of sleep reduces emotional intensity judgements of angry and happy faces. These effects were most evident in relatively ambiguous emotional expressions that were produced by morphing angry and happy faces with neutral faces, suggesting a specific impairment in recognizing subtle facial expressions of emotion (Van der Helm, Gujar, and Walker, 2010). Effects have also been reported on the ratings of emotional faces following naps, in contrast to a no-nap condition (Gujar et al., 2011). Such results have been used to suggest a role of sleep in "re-calibrating" the human emotional brain, and used as evidence that emotions become dysregulated without sleep (Gujar et al., 2011). These studies are important as they suggest that sleep disruption affects the ability to process emotion in healthy individuals, showing sleep and emotion are closely linked.

An understanding of the processes involved in normal sleep is important in order to understand how insomnia develops (c.f. Espie, 2002). Of particular relevance, both

sleep homeostatic and circadian processes have been implicated in the etiology of insomnia, and the relative contributions of these processes vary across the day (e.g. Richardson, 2007; Pigeon and Perlis, 2006; Benoit and Aguire, 1996). In the physiobiological model of Espie (2002), normal sleep is thought to be maintained and reinforced by sleep homeostasis, circadian timing, and sleep quality (Espie, 2002). These three processes are thought to result in a sleep pattern which is able to tolerate and accommodate stressors, and which defaults to good sleep (Espie, 2002). Normal sleep is therefore thought to occur automatically, without explicit conscious effort. In particular, stressors, whether behavioural, mental, or physiological, are not sufficient to inhibit good sleep, and de-arousal is not inhibited. However, the inhibition of de-arousal is itself thought to be sufficient to cause poor sleep, and additional factors can lead to insomnia (Espie, 2002). Predisposing, precipitating, and perpetuating factors are thought to contribute towards the transition from acute to chronic insomnia (Espie, 2007). The neurobiological model of insomnia also describes normal sleep-wake function, and suggests interactions of arousal, circadian rhythms, and the homeostatic sleep drive (Buysse et al., 2011). As such, circadian and homeostatic processes are important to both normal sleep and insomnia, and the relative contributions of each to sleepiness and arousal vary over the day.

Importantly, de-arousal is affected by emotion (c.f. Espie, 2002), and the brain areas identified in the neurobiological model are closely linked to emotion (Buysse et al., 2011) suggesting close linked between insomnia and emotion (c.f. Baglioni et al., 2010). Indeed, the maintenance of insomnia is thought to be affected by excessively negatively toned cognitive activity and its effects on daytime functioning (Harvey, 2002). Daytime variation in psychological and physiological parameters, and evidence of diurnal effects have been reported in mood (Buysse et al., 2007; Levitt et al., 2004; Ong et al., 2011), emotional brain activation (Hasler et al., 2012), and cardiovascular activity (Covassin et al., 2011). In particular, Buysse et al. (2007) have investigated daytime variations in mood in insomnia, identifying four factors which were related to negative mood, positive mood, sleepiness/fatigue, and alert cognition. The daytime effects of these measures were different between normal sleepers and insomnia patients, in particular, alert cognition showed a small increase over the day in insomnia, whereas normal sleepers evidenced a decline towards the evening. Group differences were most apparent at specific times, with divergent scores on positive mood and sleepiness/fatigue in the morning and evening, and on negative mood in the evening (Buysse et al., 2007). In a study by the same group, Levitt et al. (2004) found that there were significant differences between normal sleepers and insomnia patients at specific times of day. Control subjects were found to have significantly greater morning mood,

concentration, energy, and alertness; and greater alertness and energy at noon. These studies show that diurnal variations in mood are pertinent among people with insomnia, in keeping with evidence of variability in task performance over the waking period in normal sleepers (c.f. Valdez, Reilly and Waterhouse, 2008; Schmidt et al,. 2007; Waterhouse 2010; Folkard, 1990). For example, circadian rhythms affect performance, as do increasing wakefulness, and optimal performance often mirrors core body temperature (c.f. Valdez, Reilly and Waterhouse, 2008; Schmidt et al,. 2007; Waterhouse 2010; Folkard, 1990).

As such, research to date has established that 1) emotion and sleep are closely linked; 2) experimental tasks of emotional functioning can be sensitive to sleep-related changes and show diurnal variation, and 3) an understanding of sleep and emotion in healthy people under typical conditions is important in order to fully understand insomnia. Given the newness of research investigating the effects of sleep on emotion recognition, an important first step seems to be to clarify how time since waking may affect sensitivity towards emotional information among normal sleepers. In particular, how sensitivity towards emotional information may fluctuate over the working day seems to be pertinent. As emotion perception tasks have only recently been employed in people with insomnia (Kyle et al., 2014), and given the importance of homeostatic and circadian processes in normal sleep (c.f. Espie, 2002), we decided to assess normal sleepers in order to investigate possible diurnal effects in emotion perception.

In particular, the processes involved in emotion perception are thought to be linked to mood and emotional regulation (Phillips et al., 2003), and the emotional state of others is important for social interactions (e.g. Bruce and Young, 1998; Calder and Young, 2005). While we have previously used dynamic images, additional advantages of static images (Ekman and Freisen, 1976) include the extensive extant research with these stimuli; and the categorical perception of emotional faces has been established (e.g. Fugate, 2013). These stimuli were therefore used to assess whether previous results were due to the nature of the stimuli. Emotional images were also used as an emotional control task, and an image matching task was used to assess visual perception as well. Emotional scenes, such as the International Affective Pictures, or IAPS series (Lang, Bradley, and Cuthbert, 2008), are also commonly used within the study of emotion. Emotion faces and IAPS images have been compared directly, with faces rated as having lower subjective arousal and valence (Britton et al., 2006). Given the normal waking period lasts around 17 hours, with a typical sleep duration of 7 hours (Lund et al., 2010), participants were scheduled to complete the task at one of four times throughout the working day. Test times were individualized according to each subjects's

sleep schedule. It was hypothesized that emotions would show evidence of dysregulation over the working day, evidenced via an emotion recognition task. This would be consistent with results from Chapter 5.

Methods

Participants

Subjects were recruited from around campus, and ages ranged from 18 – 38 (*M* = 22.29, *SD* = 0.49). There were 54 females and 33 males, and 8% of subjects were nationals of European or American nations. An initial screening questionnaire consisting of the Pittsburgh Sleep Quality Index (Buysse et al., 1989) was used to exclude outright those subjects with evidence of sleep disruption. All participants reported good physical and mental health, without recreational drug use at testing. This study was conducted in accordance with the local Ethics Committee.

Additional exclusion criteria were:

- Morningness-Eveningness Questionnaire (Horne and Ostberg, 1976) scores < 31, or > 69, indicating an extreme preference.
- Insomnia Severity Index (Bastien, Vallieres, and Morin, 2001) scores > 14.
- Hospital Anxiety and Depression Scale (Zigmond and Snaith, 1983) scores > 10 on either subcomponent, indicating significant distress.
- Positive endorsement of "Do you have an insomnia complaint?"
- Toronto Alexithymia Scale (Bagby, Parker and Taylor, 1994) scores > 60.
- Total sleep time < 6 hours on the night before testing.
- Sleep efficiency scores < 80% on the night before testing.

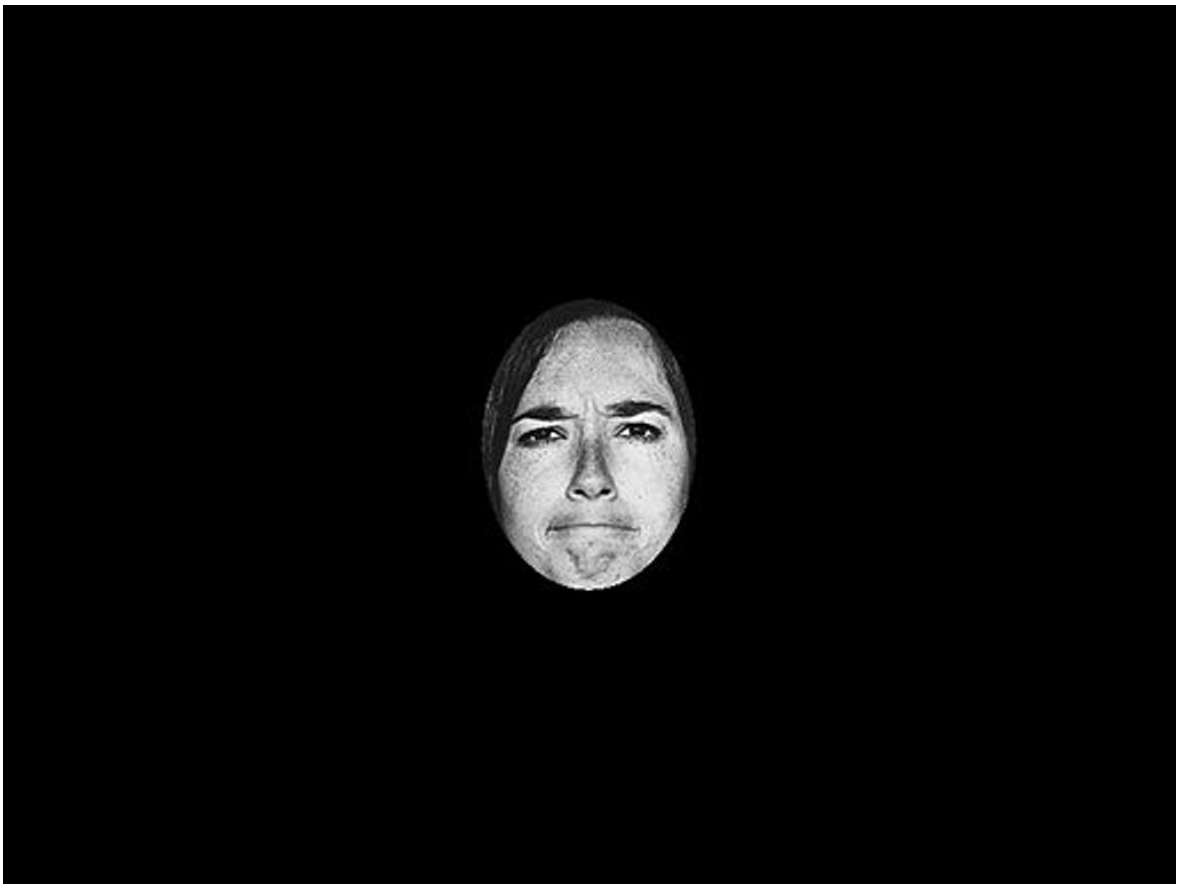
Stimuli and Measures

- Morningness-Eveningness Questionnaire (MEQ; Horne and Ostberg, 1976).
- Insomnia Severity Index (ISI; Bastien, Vallieres and Morin, 2001).
- Hospital Anxiety and Depression Scale (HADS; Zigmond and Snaith, 1983)
- Sleep Diary (Morin and Espie, 2004).
- Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989).
- Toronoto Alexithymia Scale (TAS-20; Bagby, Parker and Taylor, 1994).
- Karolinska Sleepiness Scale (KSS; Akerstedt and Gillberg, 1990).
- Global Vigor and Affect Schedule (GVAS; Monk, 1989).

Emotion faces

The face recognition task consisted of 40 Ekman and Friesen (1976) faces displaying expressions of sadness, happiness, anger and fear. Each face was repeated once, giving a total set of 80 faces, and each emotion was posed by 5 males and 5 females. Faces were greyscale, and cropped with an oval mask to remove extraneous visual cues. Average dimensions of faces were 5.15 by 7.15 cm.

Figure 24. Example facial stimuli



Emotion images

Emotion images were selected from the IAPS series (Lang, Bradley, and Cuthbert, 2008) following pilot tests, with 10 images chosen to depict each of sad, happy, angry, and fear based on subject responses. Images were repeated once, at maximum screen dimensions (28.6 by 21.8 cm), to give a total set of 80 images. When the mean arousal and valence ratings for each image were obtained from normative data (Lang, Bradley, and Cuthbert, 2008) and compared, an ANOVA suggested a significant effect of emotion on valence ratings (F (3, 36) = 51.04, p < 0.001), with Bonferroni tests suggesting significant differences between happy and the other three emotions (all p < 0.001). There was also a significant effect of emotion on arousal ratings (F (3, 36) = 9.08, p <

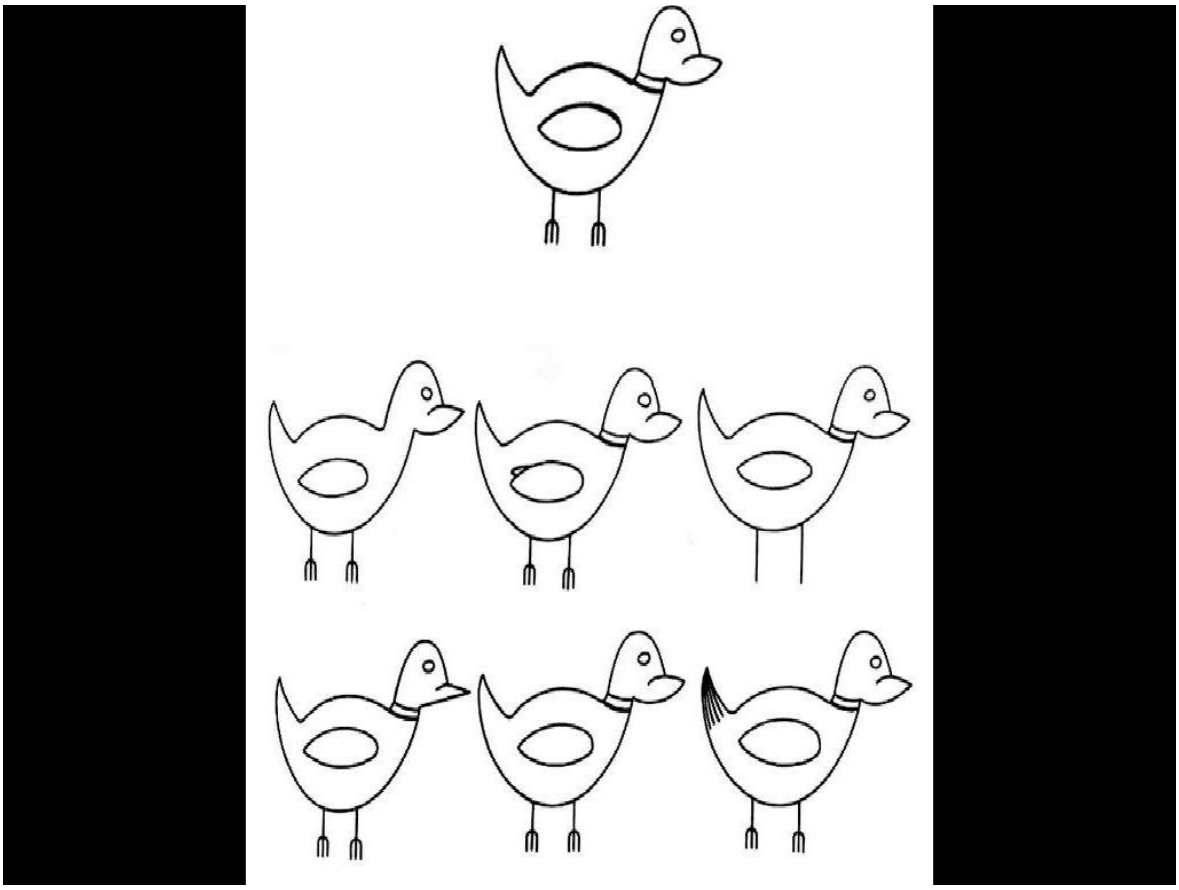
0.001). Bonferroni tests were significant between sad and anger ($p < 0.001$), and sad and fear ($p = 0.001$). As such, happy images were rated more positively than the other emotions, with sad images rated as less arousing than angry and fear.

Figure 25. Example image stimuli

Matching Familiar Figures Test (MFFT)

Stimuli are twenty greyscale line drawings of objects (e.g., a lamp, ship, or truck), with an image presented at the top of the screen. The identical image and five alternatives presented below in a three by two matrix. Participants were asked to respond with the numeric keypad (1 – 6) to identify the matching object, and stimuli were presented until response. Each image was presented once at maximum screen dimensions (28.6 by 21.8 cm). These images were developed by Kagan (1965) to assess cognitive style, and performance in this task has been found to correlate with the matching of unfamiliar faces (Burton, White and McNeill, 2010).

Figure 26. Example MFFT stimuli



Procedure

Participants were recruited via the School of Psychology subject pool, and word of mouth, and took part in exchange for £6 or course credits. They were initially emailed the PSQI, with normal sleepers asked to complete a week-long sleep diary. Sleep diary data formed the basis of test times, which were scheduled at 3, 6, 9 or 12 hours since median weekday wake-times. Participants then came in to the School of Psychology to complete the task, which consisted of emotion recognition from faces and images.

Participants were asked to recognize the emotion shown as quickly and accurately as possible from four choices, and faces were presented until response. Responses were made via the keyboard, and the keys ‘d’, ‘f’, ‘j’, and ‘k’ were labeled as angry, fear, happy and sad, respectively. Participants were familiarized with the key responses through 16 practice trials where they labeled emotion words.

Stimuli were presented on a black screen, preceded by a central fixation cross of 750 milliseconds, and followed by a blank screen of 500 ms. The emotion faces were presented randomly in two blocks with an optional 30 second break between them,

followed by the same procedure for the IAPS stimuli. Participants then completed the MFFT. Following this, participants completed the questionnaire battery.

The questionnaire battery was comprised of the state measures of the Karolinska Sleepiness Scale (KSS), mood questions, and sleep diary. The Toronto Alexithymia Scale (TAS-20), MEQ, HADS, and ISI were also given, and used to exclude clinical scorers on anxiety, depression, insomnia, and alexithymia, as well as extreme morningness-eveningness types.

Results

Firstly, the sleep of all subjects the night before testing is described below.

Table 13. Sleep diary results in the sample

	Mean	Median	Std Dev
SOL (mins)	11.82	10.00	9.80
WAKE (#)	.46	.00	.80
WASO (mins)	3.02	.00	6.52
TST (hours)	7.93	7.83	1.23
TiB (hours)	8.33	8.08	1.27
SE (%)	95.83	95.77	12.79
QUALITY (0-4)	2.94	3.00	.91
REST (0-4)	2.82	3.00	.80

Abbreviations: sleep onset latency (SOL), wake time after sleep onset (WASO), number of night-time awakenings (WAKE), total sleep time (TST), time in bed (TIB), sleep efficiency (SE), quality (QUAL); restful/restorativeness (REST).

Participants reported a normal sleep duration, with minimal evidence of sleep disruption as assessed by SOL, WASO, SE and WAKE. The time in bed of subjects was also normal, and subjects experienced their sleep as restful and of good quality

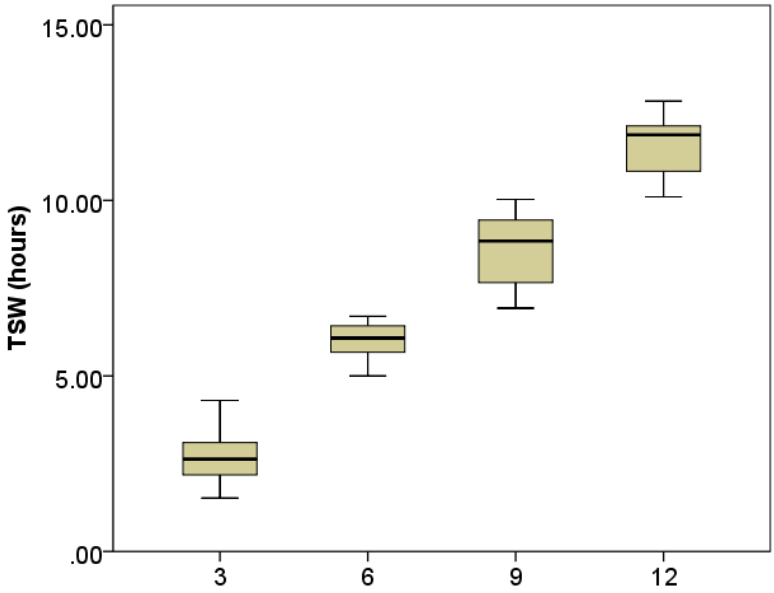
Manipulation check

1) The median weekday time-since-wake (TSW) was used to gauge subjects' habitual sleep schedule, and estimate test times at three, six, nine or twelve hours since waking. In a first step, test-day TSW was calculated, and cut-offs for four groups of equal size were obtained. Following visual inspection of the scatter plot of time since waking on the day of testing, subject groups were

finalized. As a result, the three hour group n = 22, the six hour group n = 24, the nine hour group n = 20, and the twelve hour group n = 21.

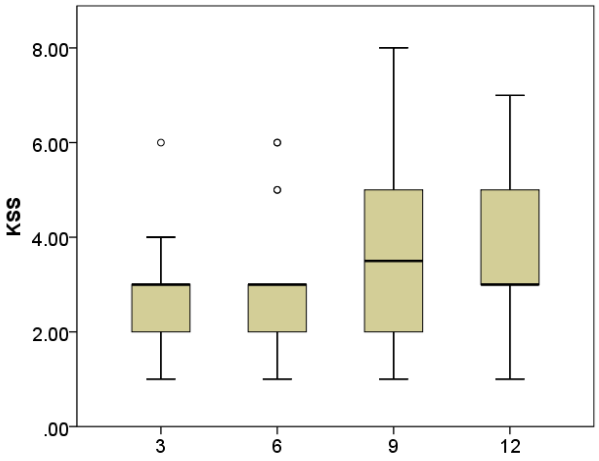
In order to validate group allocation, we then compared the four groups on their TSW. There was a significant effect of group on TSW (F (3, 86) = 515.27, p < 0.001), with follow-up Bonferroni comparisons suggesting all comparisons were significant.

Figure 27. Time since waking (TSW) between groups



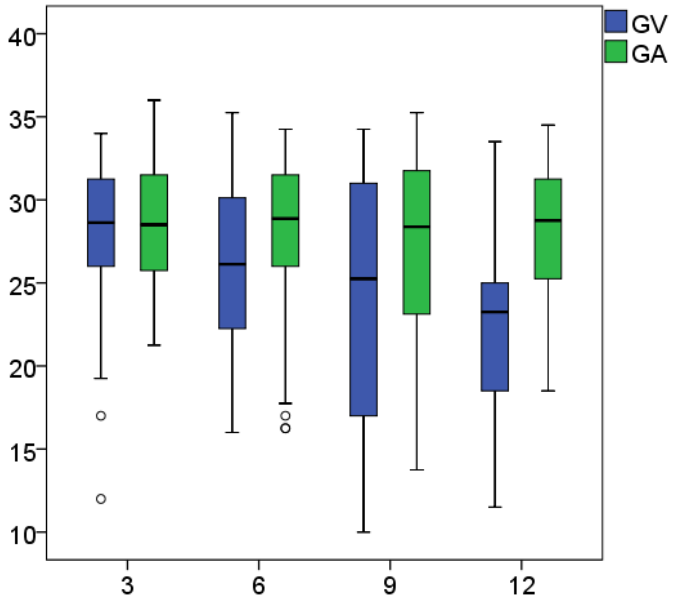
2) Next, the sleepiness levels of all groups, measured via the KSS, were compared. However, there were no significant group differences on reported sleepiness (*Kruskal-Wallis Test* (3) = 5.82, p = 0.12).

Figure 28. Karolinska Sleepiness Scale (KSS) scores between groups.



3) Sleep groups were then compared on subjective mood, with no significant group differences in global affect (F (3, 83) = 0.29, p = 0.83). However, there was a significant group difference in global vigor (*Kruskal-Wallis Test* (3) = 8.45, p < 0.05). There were significant differences between the 3 hour and 12 hour group (*Mann-Whitney U* = 107.50, p < 0.005). No other comparisons were significant (all p > 0.06).

Figure 29. Global vigor (GV) and affect (GA) scores between groups

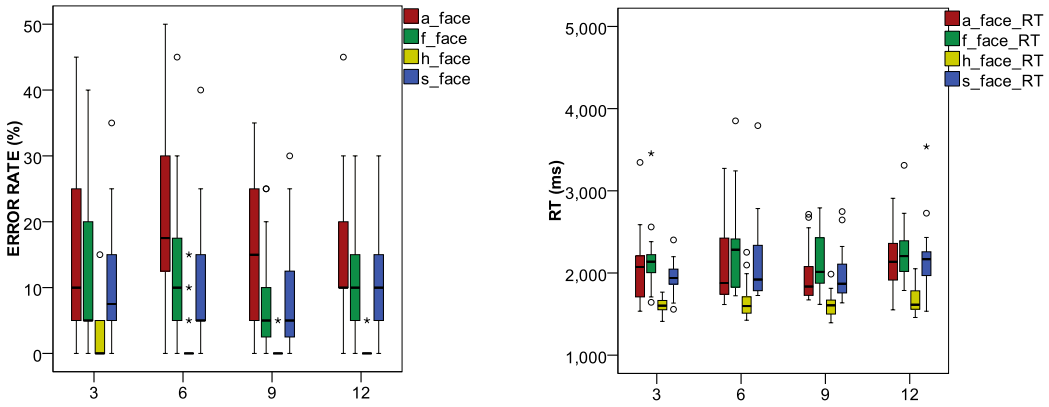


Emotion recognition from faces

Following visual inspection of the data, each of the four emotions were analyzed individually in nonparametric tests. Effect sizes can be found in Appendix 2.

Results suggest significant group differences with happy faces (*Kruskal-Wallis Test* (3) = 8.46, p < 0.05). However, no paired comparison was significant (all p > 0.16). No other comparisons were significant (all p > 0.26). There were no significant group differences for any emotion on reaction times (all p > 0.21).

Figure 30. Error rates and reaction times between groups



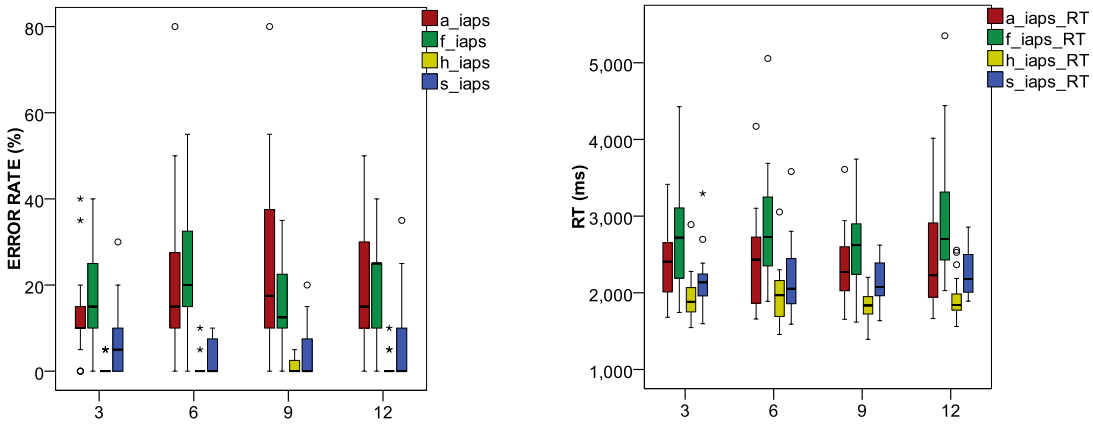
Abbreviations: anger (a); fear (f); happy (h); sad (s), reaction times (RT). A '*' indicates an outlier and a 'o' indicates a suspected outlier.

The effects with happy face recognition were followed up by regression models with total sleep time, time in bed, sleep onset latency, wake time after sleep onset, number of awakenings, sleep quality and sleep restorativeness; however no model was significant (all $p > 0.41$). As no results were significant the issue of multiple comparisons was not considered.

Emotion recognition from images

There were no significant group differences in the accuracy of recognition with any of the emotions (all $p > 0.12$), or on reaction times (all $p > 0.58$). Effect sizes can be found in Appendix 2. As no results were significant the issue of multiple comparisons was not considered.

Figure 31. Error rate and reaction times between groups



Abbreviations: anger (a); fear (f); happy (h); sad (s), reaction times (RT). A '*' indicates an outlier and a 'o' indicates a suspected outlier.

Image matching

There were no significant group differences in the matching-to-sample task, on accuracy (*Kruskal-Wallis test* (3) = 1.46, *p* = 0.68) or reaction times (*Kruskal-Wallis test* (3) = 1.91, *p*= 0.59). As no results were significant the issue of multiple comparisons was not considered. Effect sizes can be found in Appendix 2.

Discussion

Results from these tasks suggest that time since waking does not affect emotion recognition towards faces or images, or performance on an object-matching task. These results are against predictions from the previous chapter, and there are both theoretical (Espie, 2002; Valdez et al., 2008) and experimental (Buysee et al., 2007; Hasler et al., 2008; Hot, Leconte, and Sequeira, 2005) reasons to expect diurnal variation in emotional sensitivity. However, there were some indications of an increased error rate towards happiness in the earliest group, a finding which is consistent with results from Chapter Three which found effects of sleep on happiness categorization and reaction time. The TSW was also comparable to Chapter 5 Experiment One, which found that the early group were more sensitive to temporal parameters of the facial expressions. Also unlike Chapter 5 Experiment One was the evidence of significant differences between the earliest and latest groups on global vigour. There were no group differences on subjective mood or sleepiness in Chapter 5 Experiment One, although there were significant group differences on alert cognition and sleepiness/fatigue in Chapter 5 Experiment Two. In both, alert cognition was lower at a later test time. Results appear to confirm the importance of the dynamic stimuli which were used in the preceding chapter, especially given the presence of significant group differences in subjective mood. Several possible reasons for these results are discussed.

One possible reason for the lack of significant group differences could relate to a lack of significant groups differences in wakefulness or subjective mood between the four groups. However, there were significant group differences in the time since wake on the test days, with results suggesting that these group differences are significant between all four groups. Secondly, self-reported global vigour was significantly different between the earliest and latest sleep groups. However, there were no significant effects of sleep group on sleepiness or global affect, suggesting that the effects of the experimental manipulation on sleepiness were subtle. Previous studies have found diurnal variations in mood (e.g. Levitt et al., 2004), and this may have been expected to be reflected in

task performance. It is also possible that self-reported mood is not closely related to emotional task performance, and this could be relevant to the communicatory role of facial expressions of emotion (c.f. Gross and Barrett, 2011).

A second reason for the failure to find significant effects of time since waking on emotion recognition could relate to the tasks which were used. In particular, emotional categorization may not be sensitive to diurnal variation, and fluctuations in alertness over the waking period may be difficult to detect without particularly sensitive tasks (Killgore, 2010). In addition, as static faces tend to be recognized with high accuracy (e.g. Calvo and Lundqvist, 2008), dynamic facial expressions (e.g. Yu, Garrod, and Schyns, 2012) could perhaps be more sensitive, and have greater ecological validity. With regards to the effects of sleep on perceptual performance, the effects of sleep deprivation on visual tasks are subtle and can be absent when time is not constrained or less sensitive measures are used (Killgore, 2010; see also Williamson and Friswell, 2011). Those effects which are found tend to reflect deficits in top-down influences (Killgore, 2010), and it could be pertinent that emotional categorization or labeling seems to involve top-down control of the emotional brain (Hariri, Bookheimer and Mazziotta, 2000; Liebermann et al., 2007). This explanation appears most likely in light of the previous chapter, which reported effects with the 4D stimuli with regards to emotional intensity ratings and temporal parameters.

An additional reason for the failure to find significant effects could relate to behavioral performance measures, and it is possible that compensatory brain regions were employed to counteract any effects of sleep loss (Drummond et al., 2004), or the relevant brain areas were unaffected by time-since-waking. It could also be relevant that testing was scheduled within the period of the typical working day, and did not capture the de-arousal process which occurs in the lead-up to sleep (Robertson, Broomfield and Espie, 2007; Levitt et al., 2004). This could suggest that the effects of sleepiness or time since wake are only apparent at higher levels of homeostatic sleep pressure, such as in sleep deprivation paradigms, or when the circadian alerting signal has diminished in the evening.

In the psychomotor vigilance task, a gold-standard measure of sleep loss, performance defects have been found to become apparent at 16 hours of continual wakefulness, and increase thereafter (Killgore, 2010). While the groups differed significantly in global vigour, and time-since-waking, the maximum TSW fell below that which has been linked to performance impairments, with the maximum TSW group at around 14 hours. However, the clock times necessary to test a later TSW within the laboratory made late

testing times impractical. Neuroimaging techniques could also provide a more sensitive assessment. For example, Marek et al. (2010) used a Stroop paradigm to investigate aspects of attention, with four significant clusters of brain activation identified. These areas were particularly involved in the attentional orienting system, and included the frontal eye fields, an inferior parietal area, and the supplemental motor area.

It is alos possible that the sample size whas two low to detect differences in task performance. However, these results together with chapter three suggest that the static faces task is not adequately sensitive to group differences. As the testing times of this study and that of Chapter 5 Experiment 1 are comparable, it appears possible that the these test times are not sensitive to sleep-related chages in emotion recognition performance, with the later within-subjects design was able to identify.

As no significant effects of time of day were found on performance for all three tasks in the current chapter, it seems likely that emotional categorization performance is maintained across the daytime period. However, it is also possible that the current tasks were not sensitive to variations in emotionality. In keeping with this is the evidence of time of day effects with other methods. Hasler et al. (2008) report a significant diurnal pattern in socializing, laughing, and signing; behaviors associated with positive affect. Hot, Leconte, and Sequeira (2005) have found a significant linear trend in skin conductance response when unpleasant images are viewed across the day, in contrast to a significant quadratic trend with neutral images. As there is evidence of effects of time-since-waking in the previous chapter, results suggest that the current task was not sensitive to the effects of increasing wakefulness on emotional sensitivity due to the nature of stimuli and/or task.

Results in this thesis suggest that sleep disruption the previous night affects the recognition of happiness, although this result is not confirmed within the current sample of normal sleepers. There is also evidence that insomnia affects the recognition of high intensity expressions of sadness and low intensity expressions of surprise. When normal sleepers are tested at different times of day, there is evidence of effects of time-since-waking on sensitivity towards the temporal parameters of 4D faces, and in the recognition of intensity in mid-intensity expressions of sadness and anger. There are no effects of time-since-waking on emotional sensitivity across the day when reaction times and categorization are assessed in static faces. Given evidence of sleep variability in insomnia (Vallieres et al., 2005; Perlis et al., 2010; Buysse et al., 2010), and the multiple testing sessions used with the 4D faces, the effects of insomnia on emotional sensitivity at different times of day were not assessed further at this time. An additional

concern related to the results reported in Chapter 5, and the precise reasons for these effects. However, people with insomnia would be expected to assess their sleep upon awakening, and show increased arousal at bed-time in keeping with the cognitive model of insomnia maintenance (Harvey, 2002). Further work would need to be done in normal sleepers to clarify the links of sleep, mood and markers of arousal with emotion perception, and the variability within these brain systems (c.f. Phillips et al., 2003) and physiological markers. As daytime functioning is an area of concern for people with insomnia (Kyle, Espie, and Morgan, 2010), we next assessed its effects on additional tasks of social functioning.

Chapter 7

Does insomnia disorder affect mental state comprehension?

Abstract

Insomnia is a disorder characterized by subjective reports of daytime impairment, and this is a key diagnostic feature. Social interactions are crucial for everyday functioning, and impairments in this domain could contribute towards the daytime distress experienced by people with insomnia. Indeed, we have previously found that insomnia disorder affects emotion recognition performance. In the current chapter three tasks were used to investigate theory of mind performance. These were the mind in the eyes task, the false belief task, and a perspective-taking task. Participants were normal sleepers and people with insomnia. Results suggest no significant effects of group on accuracy on any task, although there were significant effects of insomnia on reaction times. In particular, people with insomnia tended to be faster in the false belief task, but slower in the perspective taking task. Results suggest that insomnia affects social functioning, and this could contribute towards the development and/or maintenance of this disorder.

Does insomnia disorder affect mental state comprehension?

Introduction

In a year, up to 33% of the population could experience sleep disruption or insomnia (LeBlanc et al., 2009), and daytime impairments in functioning are an important feature of an insomnia diagnosis (Edinger et al., 2004). Social functioning impairments are reported by people with insomnia (Kyle, Espie and Morgan, 2010; Kyle et al., 2013), however, tasks using objective measures of social functioning have only recently been used among people with insomnia. Kyle et al. (2014) have found that insomnia affects the perception of emotion from faces showing sadness and fear. In particular, people with insomnia rated emotional intensity in faces as lower than normal sleeper controls. However, successful social interactions are complex behaviours involving several components, including face and emotion recognition as well as theory of mind (Iarocci, Yager, and Elfers, 2007). Theory of mind and empathy are therefore important for interpersonal functioning and social competence (Iarocci, Yager, and Elfers, 2007; Zaki, Bolger, and Ochsner, 2008).

Theory of mind has been defined as a component of social competence which involves inferring the mental states of others, such as their beliefs, desires, feelings and intentions (Iarocci, Yager, and Elfers, 2007). Emotional expressions can lead to affective reactions and inferential processes (van Kleef, 2009), and this has been termed emotional contagion (Frith and Frith, 2012). The processing of emotion and the understanding of empathy seem to use the same neural processes (Decety and Jackson, 2006). Three forms of empathy are thought to be represented by neurocognitive circuits, namely cognitive empathy, emotional empathy, and motor empathy (Blair, 2005). Furthermore, the accuracy of emotion recognition has been linked to empathy in general, and both cognitive empathy (Zaki et al., 2008) and emotional empathy (Blair, 2005) in particular. The reason for this discrepancy seems to relate to the precise definitions of the components of empathy.

Motor empathy is the tendency to mimic others in social interactions, and likely involves the superior temporal cortex and mirror neuron systems (Blair, 2005). Zaki et al. (2008) describe the accuracy of emotion recognition as cognitive empathy, with affective empathy described as the experience of shared emotions. However, Blair (2005) defines emotional empathy as involving responses to emotional stimuli, which includes the recognition of emotional expressions. This suggests that emotional (and motor) empathy are more automatic than cognitive empathy (Blair, 2005), and Decety and Jackson

(2006) have similarly made a distinction between empathy driven by top-down or bottom-up processes.

Cognitive empathy has been defined as the ability to represent another person's mental state, or to engage in theory of mind, and could involve the medial prefrontal cortex (Blair, 2005). Tasks such as the Sally-Ann false belief task and the recognition of complex mental states assess this (e.g. Frith, 2001). People with high-functioning autism have been found to perform significantly more poorly in a task of identifying complex emotional states (Baron-Cohen et al., 2001), as do depressed individuals (Lee et al., 2005), and impairments have also been reported in social anxiety disorder (Hezel and McNally, 2014).

There seems to be agreement that empathy involves an affective response, a cognitive perspective-taking capacity, and emotion regulation (Decety and Jackson, 2006), with emotion generation and emotion regulation closely linked (Gross, Sheppes, and Urry, 2011). This corresponds to models of emotion recognition. Emotion perception is thought to involve three main states: appraisal of the stimulus, an affective state and then regulation of that affective state (Phillips et al., 2003).

A questionnaire measure exists which assesses the affective and cognitive components of empathy separately. Davies (1980) created the Interpersonal Reactivity Index (IRI), which measures two of these aspects of empathy. To identify the effect of empathy on behaviour, he believes that the different components of empathy should be considered separately, and his measure consists of four factors. The fantasy factor refers to the tendency to identify with fictional characters, and perspective taking refers to the tendency to take on another person's point of view. Empathetic concern refers to the tendency to experience compassion towards those undergoing adverse experiences, and the personal distress factor refers to the tendency to be upset when seeing that another person is undergoing a negative experience. This creates two measures of cognitive empathy (perspective taking and fantasy) and two measures of affective empathy (personal distress and empathetic concern) (e.g. Fontenelle et al., 2009). Affective empathy has been found to be influenced by obsessive-compulsive disorder (Fontenelle et al., 2009), and cognitive empathy scores are affected by post-traumatic stress disorder (Schmidt and Zachariae, 2009), depression (Wang et al., 2008) and bipolar disorder (Lahera et al., 2008). Moreover, disruption in the ability to 'disentangle [yourself] from others', or 'empathetic overarousal', is involved in psychopathology, and difficulties in 'self-other distinctiveness' can lead to personal distress (Decety and Jackson, 2006).

As such, the extent to which insomnia affects the ability to take another person's perspective and infer their mental state was the focus of the current experiment. This experiment aimed to quantify the subjective complaints that people with insomnia have with regard to social interactions (Kyle, Espie and Morgan, 2010) with objective task performance. People with insomnia completed a battery of theory of mind tasks and completed the Interpersonal Reactivity Index (Davis, 1980) in order to assess subjective and objective empathy. Consistent with the view that insomnia affects social functioning, participants with insomnia were expected to show impairments in these tasks. People with insomnia were also expected to report impairments in functioning and psychological distress, measured by the cognitive and affective components of the IRI.

Methods

Participants

Subjects were recruited from the subject pool at the School of Psychology, and took part in exchange for £15. Forty-three subjects took part, and were all native English speakers, who reported good physical health. Normal sleepers satisfied the following criteria at testing. This study was approved by the local Ethics Committee.

Normal Sleepers:

- PSQI scores of < 7
- ISI scores < 8
- No endorsement of an insomnia complaint
- No evidence of another sleep disorder
- No use of sleeping medication

Insomnia participants satisfied the following criteria at testing:

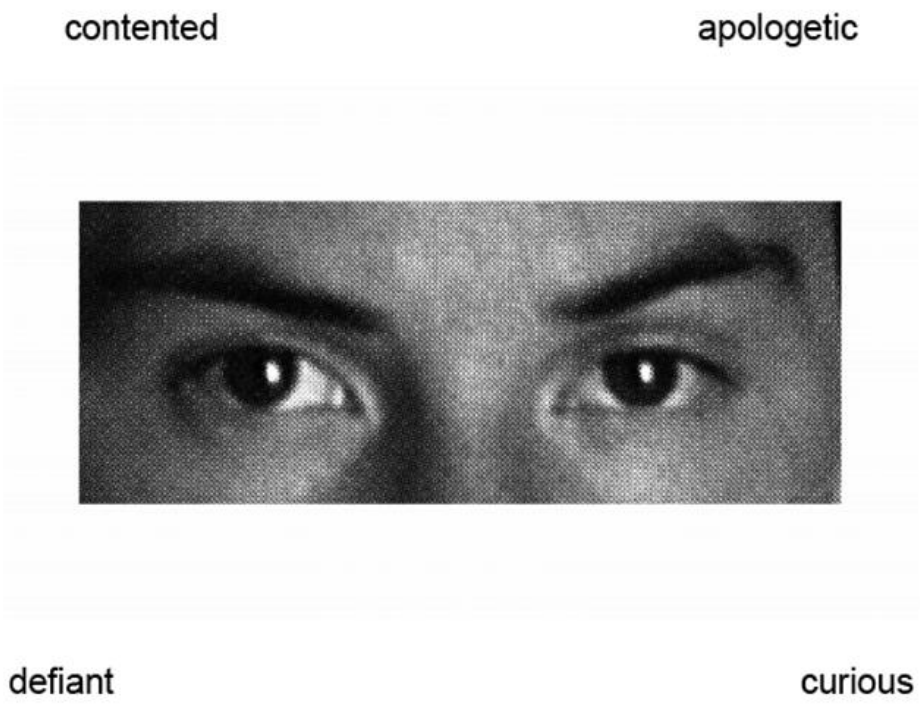
- PSQI scores of > 6
- Endorsement of an insomnia complaint

Stimuli

The 'Mind in the Eyes' Task was developed by Baron-Cohen et al. (2001). This task contains 36 images of complex mental states, which are defined as those involving attributions of beliefs or intention. Subjects are asked to select one of four words,

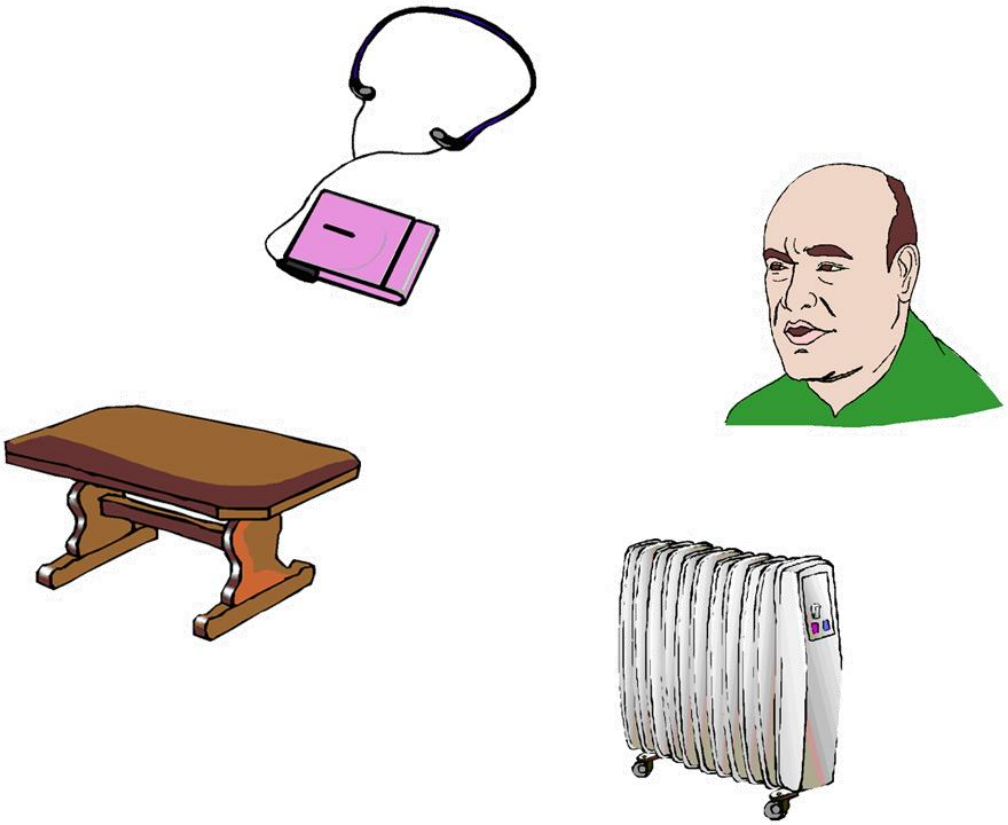
which best describes what the person in the picture thinks or feels. These words are presented in corners of the visual display, with the expression in the centre, and subjects respond via mouse clicks. Images were presented until response.

Figure 32. Example Mind in the Eyes task stimuli



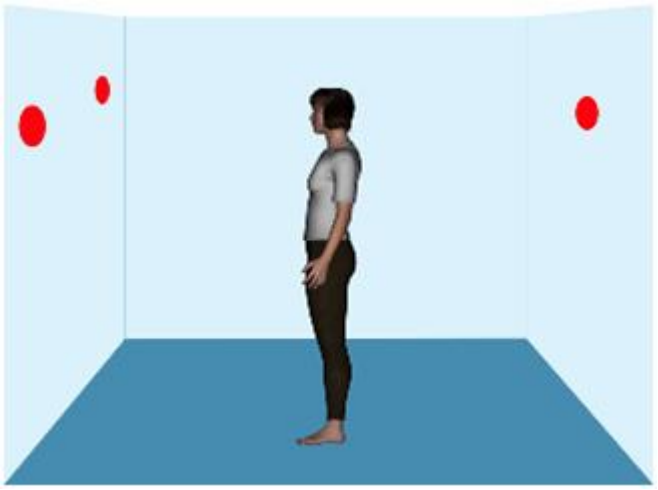
The False Belief Task used was developed by Ferguson, Scheepers, and Sanford (2010), and used their paradigm. This task requires subjects to listen to a series of sentences, and track characters' awareness of object locations. In a 'visual world' paradigm, each stage is represented visually by images of objects and characters. In the first stage, participants are told that Character A has put an object in a location, which Character B moves in stage two. In the final stage, in one condition, Character A has seen this move occur, whereas in the second condition he has not. The test question draws on this information to ask subjects e.g. where Character A will look for the object. There are two conditions overall, which are 'true beliefs' and 'false beliefs', with eight individual trials of each. Correct responding requires subjects to track both the situational and character information, which result in the two conditions. Two test session lists were created to balance task sentences between subjects. The behavioual dependent variables are recorded as mouse clicks on corresponding objects.

Figure 33. Example false belief task stimuli



The Perspective-Taking Task (Samson et al., 2010) asks subjects to adopt either their own perspective, or that of an avatar, creating two perspective conditions. They are then given a number, which represents how many dots can be seen, depending on the perspective taken. Subjects have to respond if this number of dots and the perspective match or mismatch via keyboard responses, creating two congruency conditions. In the test phase, an avatar is shown in a room facing a wall. There are dots displayed on the wall, and if an avatar can see them all ("they" condition), and the number of dots matches the number previously given, subjects respond with "match". If a subject is given "your" perspective, and they can see that there are a different number of dots on the walls than the number previously given, they respond "mismatch". In total 192 trials are shown, with equal numbers of both perspective conditions (you/they), and both consistency conditions (congruent/incongruent). Male and female avatars were selected to correspond with the participants' gender. Example stimuli are shown below.

Figure 34. Example perspective taking task stimuli



Questionnaires

The PSQI and ISI were given pre-test, and a sleep diary, DISS, STQ, HADS, MEQ, IRI and the FNE were given post-test, alongside a supplemental interview to confirm sleep group status.

Procedure

Potential participants were recruited via emailing the departmental subject pool, and those participants expressing an interest completed the pre-testing measures of the PSQI and ISI, as well as brief questions as to general health. Eligible subjects scored above or below threshold on the PSQI, with a corresponding presence or absence of an insomnia complaint. Insomnia was defined as a difficulty with getting to sleep, maintaining sleep, early morning awakenings, or non-restorative sleep, which adversely affects your daytime functioning, and subjects were asked if they thought they had insomnia (yes/no). Subjects were then invited to take part in the task battery, which was ordered via a latin square design to balance task order across subjects. Tasks were presented via the Eyelink1000 software, in a darkened room at a viewing distance of 85 cm. Screen diameters were 1280 x 1024 pixels, and the tasks took approximately 90 minutes to complete in total. Afterwards, participants completed the questionnaire battery and an additional semi-structured interview confirmed sleep groups.

Results

Group characterization

Of 21 NS (15 female, 6 male), the mean age was 22.57 (*SD* = 3.85). The 22 INS (14 female, 8 male) had a mean age of 23.00 (*SD* = 4.02), with no significant group differences in age (*T* (41) = - 0.36, *p* = 0.73), or gender (X^2 = 0.297, *p* = 0.586). There were a majority full-time students in both groups (19 of NS, 20 of INS), with group differences not significantly different (X^2 = 0.002, *p* = 0.961).

Table 14. Questionnaire results between normal sleepers and insomnia subjects

	GROUP					
	NS			INS		
	Mean	Median	Std Dev	Mean	Median	Std Dev
PSQI	3.29	3.00	1.31	11.14	11.50	2.46
ISI	2.71	3.00	1.82	14.68	15.00	2.97
MEQ	48.76	49.00	8.88	40.23	37.00	10.00
HADS-A	5.71	5.00	2.59	8.95	9.00	4.45
HADS-D	2.57	2.00	2.09	6.55	6.50	3.56
IRI-PT	20.33	21.00	4.35	20.23	21.00	4.55
IRI-FS	19.90	20.00	4.53	21.36	20.00	4.05
IRI-EC	21.62	22.00	4.72	21.68	22.00	4.36
IRI-PD	15.95	17.00	4.20	15.27	14.00	4.81
FNE	16.00	15.00	7.49	16.77	16.50	8.85
DISS-AC	61.82	64.00	15.26	44.65	43.20	18.50
DISS-NA	22.56	18.20	14.40	31.44	22.70	21.77
DISS-PA	53.95	54.80	14.47	42.95	42.30	17.63
DISS-SF	41.05	45.67	22.91	60.12	64.50	24.83

Abbreviations: Pittsburgh Sleep Quality Index (PSQI); Insomnia Severity Index (ISI); Morningness-Eveningness Questionnaire (MEQ); Hospital Anxiety and Depression Scale – Anxiety (HADS-A); Hospital Anxiety and Depression Scale – Depression (HADS-D); Interpersonal Reactivity Index – Perspective Taking (IRI-PT); Interpersonal Reactivity Index – Fantasy Scale (IRI-FS); Interpersonal Reactivity Index – Emapthic Concern (IRI_EC); Interpersonal Reactivity Index – Personal Distress (IRI-PD); Fear of Negative Evaluation (FNE); Daytime Impact of Sleep Scale – Alert Cognition (DISS-AC); Daytime Impact of Sleep Scale – Negative Affect (DISS-NA); Daytime Impact of Sleep Scale – Positive Affect (DISS-PA); Daytime Impact of Sleep Scale – Sleepiness/Fatigue (DISS-SF).

There were significant group differences on the PSQI (*T* (32.36) = - 13.16, *p* < 0.001) and ISI (*T* (41) = - 15.85, *p* < 0.001), MEQ (*T* (41) = 2.95, *p* = 0.005), HADS-A (*T* (34.08) = - 2.94, *p* < 0.01) and HADS-D (*T* (34.22) = - 4.49. *p* < 0.001). Sleep groups also showed differences in mood, assessed by the DISS (AC, *T* (41) = 3.31, *p* < 0.005; NA, *T* (36.60) = - 1.58, *p* = 0.12; PA, *T* (41) = 2.23, *p* < 0.05; SF, *T* (41) = - 2.62, *p* < 0.05). However, there

were no significant differences in the components of the IRI (PT, T (31) = 0.08, p = 0.94; FS, T (41) = - 1.11, p = 0.27; EC, T (41) = - 0.05, p = 0.96; PD, T (41) = 0.49, p = 0.63) or FNE (T (41) = - 0.31, p = 0.76).

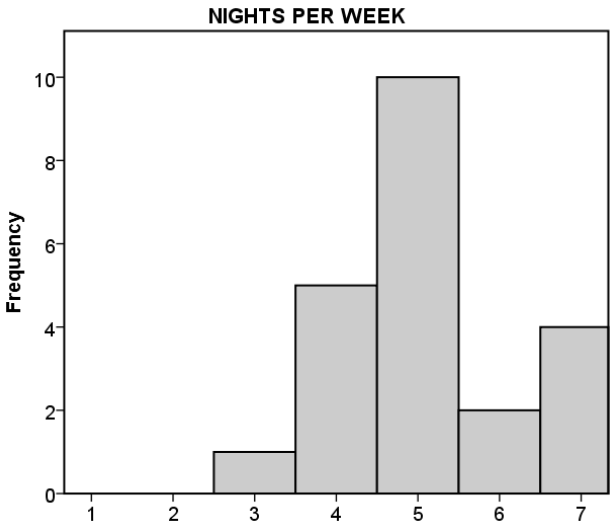
The severity of sleep-related insomnia symptoms was assessed via the first three questions of the ISI. With regards to difficulty falling asleep, normal sleepers reported none-mild symptoms, whereas a majority of insomnia participants reported symptoms which were mild-severe. All normal sleepers reported no difficulty falling asleep, whereas insomnia participants reported moderate symptoms most commonly. A majority of normal sleepers reported no problems waking up too early, and symptoms were mild-moderate for insomnia participants.

Table 15. Percentage of sleep onset, maintenance, and awakening problems between sleep groups.

		GROUP	
		NS	INS
		% (Count)	% (Count)
Difficulty falling asleep	None	86% (18)	0 (0)
	Mild	14% (3)	5% (1)
	Moderate	0 (0)	32% (7)
	Severe	0 (0)	45% (10)
	Very	0 (0)	18% (4)
		100% (21)	100% (22)
Difficulty staying asleep	None	100% (21)	4.5% (1)
	Mild	0 (0)	22.7% (5)
	Moderate	0 (0)	50.0% (11)
	Severe	0 (0)	13.6% (3)
	Very	0 (0)	9.1% (2)
		100% (21)	100% (22)
Problem waking up too early	None	76% (16)	22.7% (5)
	Mild	19% (4)	31.8% (7)
	Moderate	5% (1)	31.8% (7)
	Severe	0 (0)	4.5% (1)
	Very	0 (0)	9.1% (2)
		100% (21)	100% (22)

Additionally, the severity of sleep disruption in insomnia subjects was quantified by two measures: how long they had been experiencing poor sleep; and the number of nights per week their sleep was affected.

Figure 35. Frequency of insomnia symptoms in insomnia subjects



In a week, insomnia subjects experienced sleep disruption on five nights most commonly. All poor sleepers reported sleep disruption at least three nights a week, and around 70% reported sleep disruption at least five nights, meeting the frequency criterion for acute insomnia described by Ellis et al. (2012). This was reflected in the daytime effects of insomnia.

Table 16. Reported insomnia complaints between sleep groups (%)

		GROUP	
		NS	INS
		% (Count)	% (Count)
How satisfied/dissatisfied are you with your current sleep pattern?	very	14.3% (3)	18% (4)
	satisfied	33.3% (7)	41% (9)
	neutral	19.0% (4)	9% (2)
	dissatisfied	14.3% (3)	5% (1)
	very	19.0% (4)	27% (6)
		100% (21)	100% (22)
To what extent do you consider your sleep problem to interfere with your daily functioning?	not at all	76% (16)	0 (0)
	a little	24% (5)	5% (1)
	somewhat	0 (0)	36% (8)
	much	0 (0)	41% (9)
	very much	0 (0)	18% (4)
		100% (21)	100% (22)
How noticeable to others do you think your sleeping problem is in terms of impairing the quality of your life?	not at all	95% (20)	5% (1)
	a little	5% (1)	36% (8)
	somewhat	0 (0)	36% (8)
	much	0 (0)	23% (5)
		100% (21)	100% (22)
How worried/distressed are you about your current sleep problem?	not at all	90% (19)	9.1% (2)
	a little	10% (2)	0 (0)
	somewhat	0 (0)	63.6% (14)
	much	0 (0)	22.7% (5)
	very much	0 (0)	4.5% (1)
		100% (21)	100% (22)

A majority of normal sleepers reported that their sleep was not interfering with their daily functioning, whereas insomnia participants reported that their sleep was somewhat or much interfering. Normal sleepers also reported that their sleep problem was not noticeable to others, in contrast to insomnia participants who reported that their sleep problem was a little or somewhat noticeable. Normal sleepers were also not worried or distressed by their sleep problem, whereas a majority of insomnia participants were at least somewhat worried. However, the numbers of participants in both groups reporting sleep dissatisfaction were comparable, indicating a level of sleep dissatisfaction among normal sleepers and/or mild sleep dissatisfaction in insomnia participants. We then compared sleep groups on their reported sleep the previous night.

Table 17. Sleep diary results between normal sleepers (NS) and insomnia subjects (INS)

	GROUP					
	NS			INS		
	Mean	Median	Std Dev	Mean	Median	Std Dev
SOL (mins)	13.81	10.00	12.20	56.67	38.00	59.49
WAKE (#)	1.10	1.00	1.22	2.14	2.00	1.56
WASO (mins)	4.33	3.00	5.97	23.10	10.00	38.87
TST (hours)	8.20	8.00	1.58	5.90	6.00	1.34
TiB (hours)	9.05	8.50	1.46	9.35	10.00	2.04
SE (%)	91.49	91.30	17.99	64.58	62.50	14.70
QUALITY (0-4)	3.38	3.00	.67	2.05	2.00	.84
REFRESH (0-5)	3.38	3.00	.86	2.45	2.00	.80
TSW (hr:ms)	7:05	7:07	2:14	6:45	6:50	2:16

Abbreviations: sleep onset latency (SOL), wake time after sleep onset (WASO), number of night-time awakenings (WAKE), total sleep time (TST), time in bed (TIB), sleep efficiency (SE), restful/restorativeness (REST), quality (QUAL).

There were significant group differences on test day on SOL (T (21.68) = - 3.23, p < 0.005), WAKE (T (40) = - 2.43, p < 0.05), WASO (*Mann-Whitney U* = 321.00, p < 0.05), TST (*Mann-Whitney U* = 53.00, p < 0.001), SE (*Mann-Whitney U* = 45.00, p < 0.001), QUALITY (T (41) = 5.73, p < 0.001), REFRESH (T (41) = 3.65, p = 0.001). However, there were no significant group differences in TiB (T (41) = - 0.57, p = 0.58), or TSW (T (37) = 0.47, p = 0.64).

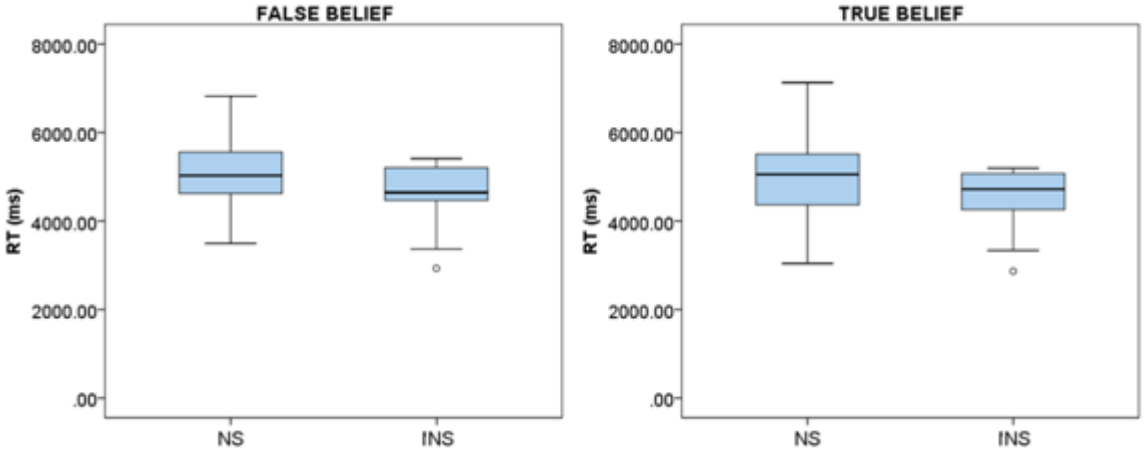
Mind in the Eyes

There were no significant group differences on accuracy (*Mann-Whitney U* = 166.00, p = 0.11) or reaction times (*Mann-Whitney U* = 156.00, p = 0.07). Effect sizes can be found in Appendix 2.

False Belief Task

There were no significant group differences in accuracy, in either condition (FB, *Mann-Whitney U* = 228.50, p = 0.78; TB, *Mann-Whitney U* = 200.00, p = 0.54). However, groups did differ significantly on reaction times (FB, *Mann-Whitney U* = 128.00, p < 0.05; TB, *Mann-Whitney U* = 135.00, p < 0.05). The false belief (FB) results are significant after accounting for these two comparisons (0.05/2 = 0.03), although the true belief (TB) results are not. Effect sizes can be found in Appendix 2.

Figure 36. False belief and true belief reaction times between groups



'°' indicates a suspected outlier. Abbreviations: Normal Sleepers (NS); Insomnia (INS).

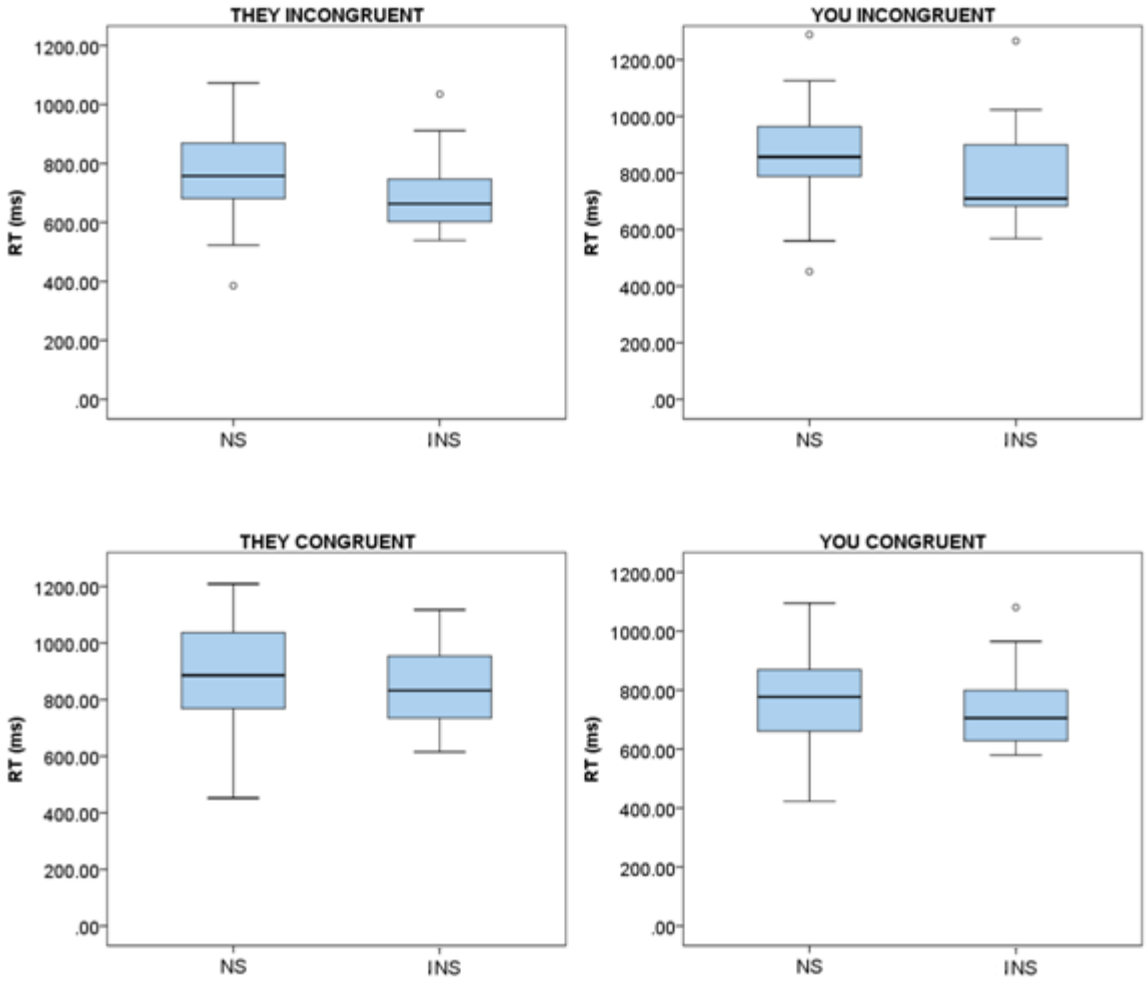
Perspective Taking Task

There were no significant group differences on accuracy in the THEY^{X}INCONGRUENT condition (T (35) = 1.74, p = 0.09) or THEY^{X}CONGRUENT condition (*Mann-Whitney U* = 176.50, p = 0.87). Similarly, there were no significant group differences in the YOU^{X}INCONGRUENT condition (*Mann-Whitney U* = 234.00, p = 0.06) or in the YOU^{X}CONGRUENT condition (*Mann-Whitney U* = 208.50, p = 0.26).

When reaction times were compared, there was a significant main effect of group (F (1, 140) = 4.15, p < 0.05), and a significant main effect of congruency (F (1, 140) = 12.55, p = 0.001). There was no significant main effect of perspective (F (1, 140) = 0.08, p = 0.78), or interactions of perspective with congruency (F (1, 140) = 1.73, p =0.19), perspective with group (F (1, 140) = 0.10, p = 0.76), or congruency with group (F (1, 140) = 0.01, p = 0.93). The three-way interaction was not significant (F (1, 140) = 0.29, p = 0.59).

Follow-up t-tests were then carried out between groups, with no significant differences in any of the four combinations. For congruent trials with 'their' perspective, (T (35) = 1.19, p = 0.24), for 'you' results were (T (35) = 0.89, p = 0.34). Incongruent trials with 'their' perspective resulted in (T (35) = 0.64, p = 0.53), and for 'you' results were (T (35) = 1.34, p = 0.19). As such the issue of multiple comparisons was not considered. Effect sizes can be found in Appendix 2.

Figure 37. Perspective taking task reaction time scores between groups



Discussion

Insomnia subjects showed no impairments in the Eyes task, on either accuracy or reaction times. Both groups also performed similarly on the False Belief task, with regards to accuracy, although there was evidence that insomnia participants were faster to respond in both conditions. Similarly, sleep groups did not differ in accuracy in the perspective taking task, although there was evidence of a significant main effect of group on reaction times. In general, poor sleepers took longer to respond, although this effect was not specific to any particular condition. There were no significant effects of sleep group on any aspect of subjective empathy.

The effects of insomnia on reaction times have previously been reported by Altena et al. (2008). These authors found that people with insomnia were faster to respond on a simple vigilance task but slower to respond on a complex vigilance task in comparison to control subjects, when performance ratios were assessed in a sample of older adults. This finding appears relevant to the current study, where insomnia patients were faster

to respond in the false belief task, but slower to respond in the perspective taking task. This could reflect differences in task demands. For example, the false belief task comprised three sentences, the last of which asked subjects to identify where a particular character would look for an object. This segmentation of the task into three discrete sentences and screens could have aided comprehension, and it is also possible that tasks of this nature are more familiar to participants. In contrast, in the perspective taking task participants were given two cues, and were then asked to identify whether they were a match or mismatch. This task was therefore less akin to how social interactions occur in real life, and could therefore have been more cognitively demanding. These results would also seem to be in keeping with the role of hyperarousal in insomnia (e.g. Riemann et al., 2010; Bonnet and Arand, 2010).

Perhaps surprisingly, subjects with insomnia did not report alterations in subjective empathy. Sleep loss has previously been reported to affect self-reported emotional intelligence (Killgore et al., 2008; Killgore, 2013) but not objective emotional intelligence (Killgore, 2013). As such, the current results appear dissimilar to those found in total sleep deprivation, with effects on task functioning but not on subjective reports. The current results therefore do not seem likely to reflect the effects of sleep loss, despite several group differences in sleep parameters. However sleep, arousal and stress are clearly linked (Meerlo et al., 2008), and the regulation of arousal is thought to play a key role in insomnia (c.f. Espie, 2002; Harvey et al., 2002; Riemann et al., 2010). Future studies could investigate potential differences in prefrontal functioning and prefrontal-amygdala connectivity via neuro-imaging techniques.

In insomnia, poor neurobehavioral performance has been linked to increased psychopathology (i.e. depression, anxiety, insomnia, physical health-related quality of life), which could relate to general alterations in pre-frontal functioning (Sheckleton et al., 2014; Altena et al., 2008). As sleep groups differed in several components of subjective mood and psychological distress, it is possible that the current results in insomnia subjects were linked to general psychopathology. Indeed, anxiety and depression are prevalent in insomnia, and 91% of people with persistent insomnia also score highly on measures of anxiety and depression (Jansson and Linton, 2007).

Importantly, there were no significant group differences in performance accuracy despite effects on reaction times. This suggests that insomnia is not linked to impairments in mentalizing as such, but that alterations in aspects of timing could underlie subjective impairments. This could also be relevant to the fatigue reported by insomnia patients (Kyle, Morgan and Espie, 2010), and the role of cognitive evaluations

of daytime functioning in people with insomnia (Harvey, 2002). It has also been suggested that sleep loss may be countered by the compensatory recruitment of additional brain areas, although the role of this in the current study would need to be investigated further. Differences in task duration could also have contributed towards results, and it is also possible that the false belief task, with the combined use of audio-visual stimuli was more engaging.

Another relevant factor relates to the nature of insomnia within this group. Average scores on the ISI were 15, which is the threshold for a clinical level of sleep disruption, with a maximum possible score of twenty-eight. However, these scores appear comparable to those found in a clinical setting, with Kyle et al. (2014) reporting ISI scores of an average eighteen. With regards to the severity of sleep disruption, forty-five percent reported severe problems falling asleep, with problems very severe in a further eighteen percent and moderate for thirty-two percent. Furthermore, insomnia participants reported sleep disruption three nights per week or greater. Over ninety percent of insomnia participants were distressed by their sleep problem, with ninety-five percent reporting that their sleep problem interfered with their daily functioning at least moderately. There were no significant group differences in time in bed. Insomnia participants therefore met the criteria for insomnia disorder described by Edinger et al. (2004). However, the subjective distress appeared three points higher in the current sample (HADS-A and HADS-D), perhaps suggesting greater arousal in the current sample than that of Kyle et al. (2014). Also relevant is that both sleep groups reported similar levels of sleep dissatisfaction, despite no reported insomnia complaint. This appears to suggest that arousal and distress contributed towards the current results. It is also possible that with more subjects these effects could become more apparent. However, the sample size selected is consistent with the published experimental literature in insomnia disorder.

Overall, results from this study suggest that insomnia affects theory of mind performance. In particular, reaction times are significantly different in insomnia participants than normal sleepers, with no significant group differences in accuracy. Insomnia participants appear faster in a false-belief task, but slower in a perspective taking task. Results suggest that the skew is affected by insomnia, with fewer longer responses in the false belief task and fewer faster responses in the perspective taking task. There were no differences in self-reported empathy. Results appear in keeping with the role of arousal in insomnia (Altena et al., 2008; Bonnet and Arand, 2010; Riemann et al., 2010), and the importance of daytime functioning complaints (Kyle, Espie and Morgan, 2010; Harvey, 2002). Future studies should investigate the role of

arousal in theory of mind task performance in insomnia using neuroimaging techniques, such as prefrontal hypoactiviation (Altena et al., 2008) and its behavioural correlates.

As a result of the experimental work in this thesis, the issue arose as to how normal sleepers are selected and screened, as there are differences between normal sleepers, poor sleepers, and insomnia disorder (Espie, 2002). In particular, it became apparent that the methods for selecting normal sleepers are typically less rigorous than those for insomnia disorder participants, despite published standards on this issue (Edinger et al., 2004). As data from both insomnia disorder subjects and controls affects result, it seemed important to investigate this issue in greater depth. The next chapter systematically reviewed the literature in order to address this issue.

Chapter 8

How are normal sleeping controls selected? A systematic review of cross-sectional insomnia studies and research agenda

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LB conceived the study, conducted the analysis and wrote the paper, with the other authors contributing feedback primarily regarding style.

Abstract

There appears to be some inconsistency in how normal sleepers (controls) are selected and screened for participation in research studies for comparison with insomnia patients. The purpose of the current study is to assess and compare methods of identifying normal sleepers in insomnia studies, with reference to published standards. We systematically reviewed the literature on insomnia patients which included control subjects. The resulting 37 articles were systematically reviewed with reference to the five criteria for normal sleep specified by Edinger et al. (2004). In summary, these criteria are: evidence of sleep disruption; sleep scheduling; general health; substance/medication use; and other sleep disorders. We found sleep diaries, PSG, and clinical screening examinations to be widely used with both control subjects and insomnia participants. However, there are differences between research groups in the precise definitions applied to the components of normal sleep. We found that none of reviewed studies applied all of the Edinger et al. criteria, and 16% met four criteria. In general, screening is applied most rigorously at the level of a clinical disorder, whether physical, psychiatric, or sleep. While the Edinger et al. criteria seem to be applied in some form by most researchers, there is scope to improve standards and definitions in this area. Ideally, different methods such as sleep diaries and questionnaires would be used concurrently with objective measures to ensure normal sleepers are identified, and descriptive information for control subjects would be reported. Here, we have devised working criteria and methods to be used for assessment of normal sleepers. This would help clarify the nature of the control group, in contrast to insomnia subjects and other patient groups.

How are normal sleeping controls selected? A systematic review of cross-sectional insomnia studies and research agenda

Introduction

Given the significance of sleep to well-being (Luyster et al., 2012), consistency in how research participants are selected is important. Indeed, this is accepted among clinicians, with diagnostic systems used to identify different sleep disorders (Edinger et al., 2004; DSM-IV, 1994; ICSD, 2005). While it is acknowledged that adherence to consensus categorization systems is important with clinical groups, such high standards have not always been applied to the selection of normal sleepers (controls). As a result, the precise definitions, and consequently methods, applied to identify normal sleepers are variable within sleep research. The purpose of the current study was to investigate exactly how control subjects are assessed, in comparison to insomnia patients. The selection of control subjects is important, as group differences may be caused by these subjects rather than the patient group, if normal sleepers are not well defined and selected. Furthermore, consistency in how normal sleepers are defined is important in order to compare results between studies. These results have broader implications for the selection of normal sleepers or control subjects within sleep research overall.

A definition of normal sleepers (controls) has been provided, and five criteria have been identified. The research diagnostic criteria (RDC) for normal sleepers specifies that normal sleepers should show no evidence of sleep disruption (Criterion A), and that the timing of sleep should be both regular and conventional (Criterion B; Edinger et al., 2004). As such, both the quality of sleep and its timing are thought to be important in defining normal sleepers. However, these components of normal sleep are not always applied in practice. For example, the Pittsburgh Sleep Quality Index, or PSQI (Buysse et al., 1989), and the Insomnia Severity Index, or ISI (Bastien, Vallieres and Morin, 2001), have been used to categorize participants as poor and normal sleepers (Ong et al., 2011; Bower et al., 2010; Ree, Pollitt and Harvey, 2006; Barclay and Gregory, 2010; Jones et al., 2005). In this approach, those participants scoring below threshold are categorized as normal sleepers. Others seem to define acceptable levels of sleep disruption, or to select healthy subjects based on the absence of insomnia disorder rather than the presence of normal or good sleep. However, such differences in methods may lead to different groups being used as a comparison, with some subjects better sleepers than others. Furthermore, evidence of sleep disruption is only one component of research diagnostic criterion for control subjects (Edinger et al., 2004).

The second component of research diagnostic criterion for control subjects includes two elements; firstly, that sleep timing is conventional (Edinger et al., 2004). Some authors specify habitual bed times and rise times as inclusion criteria. This is also pertinent to circadian rhythm sleep disorders, and an individual's preference for morningness or eveningness is relevant to their sleep scheduling. The morningness-eveningness questionnaire (MEQ) was developed to assess diurnal preference (Horne and Ostberg, 1976), and has been used to identify morning and evening types (Archer et al., 2003; Goel, Kim and Lao, 2005; Taillard et al., 2003; Phipps-Nelson et al., 2003; Carney et al., 2004). Furthermore, the RDC also specifies that timing of sleep is stable. Sleep diaries can be used to monitor adherence to a sleep schedule (Taillard et al., 2003; Vandewalle et al., 2007; Yoo et al., 2007), and assess reported sleep patterns and habits, as well as their variability (Ferguson et al., 2010; Buysse et al., 2010; Dahlgren et al., 2009). They provide information about the daily timing of sleep, as well as measures of sleep continuity (e.g. wake after sleep onset), and its qualitative experience, and sleep diaries are regarded as the "gold-standard" in measuring subjective sleep experience (Carney et al., 2012). However, while a routine sleep schedule is thought to be important to normal sleep (Buysse et al., 2011; Espie, 2002), there seems to be a lack of clarity as to how much variability in sleep scheduling is acceptable in practice.

To fully understand the development and maintenance of sleep disorders, such as insomnia, it is necessary to understand the processes in normal sleep (Buysse et al., 2011; Espie, 2002; Espie et al., 2006). However, this is hampered when the methods of assessment of normal sleepers differ, and this seems especially pertinent when research subjects are recruited from a student population, whose sleep can be irregular, and of poor quality (Lund et al., 2010). A majority of potential participants (i.e. normal sleepers) might be expected to show a moderate level of vulnerability towards poor sleep or insomnia, in keeping with a normal distribution, e.g. Yiend (2010).When insomnia subjects and normal sleepers are compared on the effects of poor sleep, the daytime effects of poor sleep are similar, although more severe for insomnia patients (Espie et al., 2012), and both groups use comparable criteria to judge sleep quality (Harvey et al., 2008). However, in insomnia patients the daytime effects associated with sleep seem especially important, both in theory (Espie, 2002; Harvey, 2002) and to patients themselves (Espie et al., 2012; Kyle, Espie, and Morgan, 2010).Current research is aimed at investigating the etiology of insomnia disorder, e.g. the development of chronic insomnia from acute insomnia (Ellis et al., 2014), and this suggests the importance of additional factors in the development of insomnia disorder. For example, insomnia patients might experience the effects of sleep disruption more severely, or report more frequent nights of poor sleep (Yiend, 2010), and changes in sleep

architecture could contribute towards this transition (Ellis et al., 2014). Furthermore, in keeping with a normal distribution (Yiend, 2010), some normal sleepers could show evidence of sleep disruption, while not quite endorsing insomnia, e.g. Espie, (2002). Normal sleepers also could be different from good sleepers, who would be expected to report good sleep without sleep disruption. Although investigating the differences between good sleep and normal sleep is beyond the scope of the current paper, understanding definitions applied to control subjects seems an important first step. As such, we have conducted a systematic review on how control subjects are assessed for study inclusion within insomnia research. We then outline recommendations for assessing normal sleep, and suggest methods of assessment.

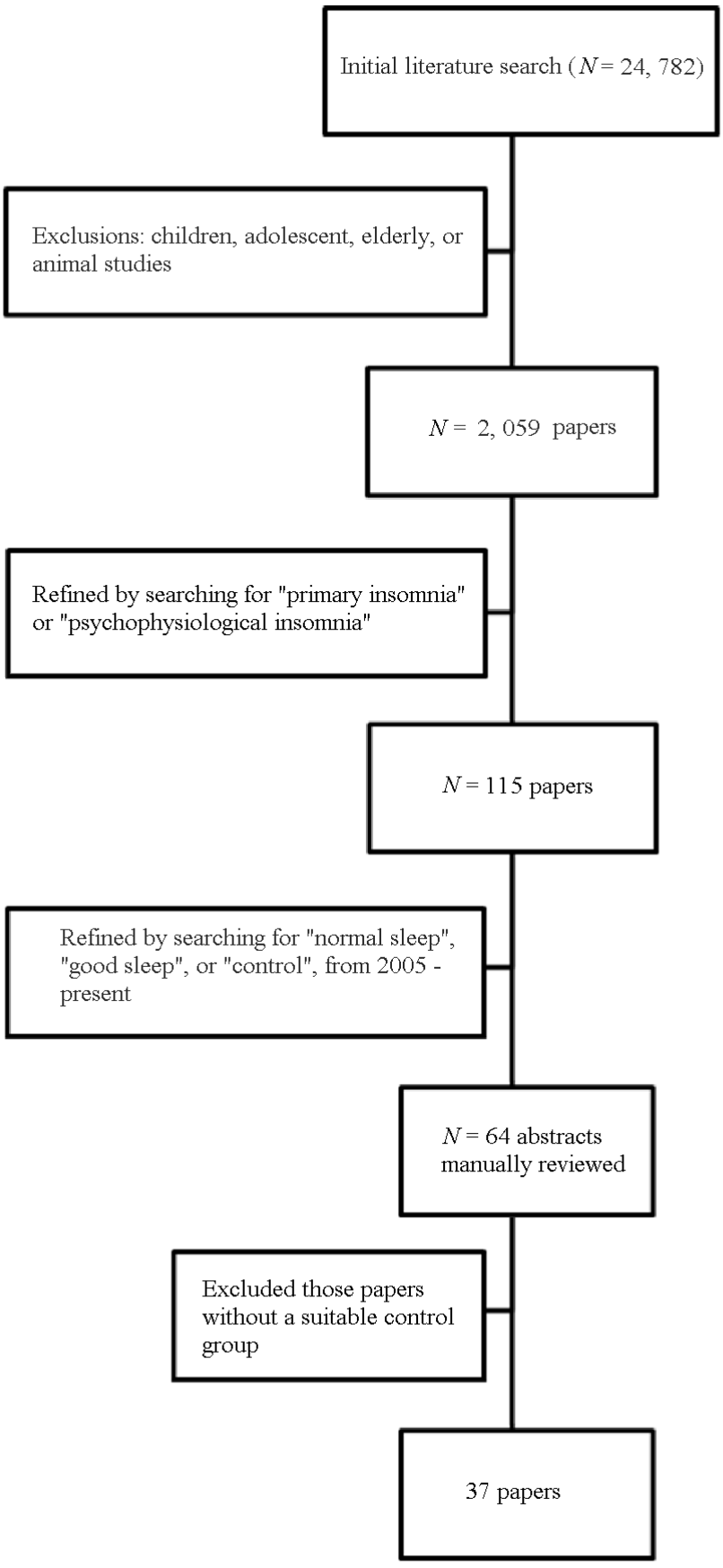
Methods

A literature search was conducted within six key sleep-society affiliated journals. In particular, Sleep is the official publication of the Associated Professional Sleep Societies, the Journal of Sleep Research is published on behalf of the European Sleep Research Society, and Sleep Medicine is the official journal of the World Association of Sleep Medicine and International Pediatric Sleep Association. Behavioral Sleep Medicine is official journal of the Society of Behavioral Sleep Medicine, Chronobiology International is the official journal for the International Society for Chronobiology, the American Association for Medical Chronobiology and Chronotherapeutics, and the Society for Light Treatment and Biological Rhythms. The Journal of Biological Rhythms is the official publication of the Society for Research on Biological Rhythms. The Journal of Clinical Sleep Medicine, an official publication of the American Academy of Sleep Medicine, was not included due to a lack of institutional access. The literature search was confined to these journals, as they were expected to apply more stringent criteria towards how sleep groups are defined. The anticipated effect of this was to bias the literature search towards more conservative or stringent methodologies with regards to sleep.

The “Web of Knowledge” (http://wok.mimas.ac.uk/) search engine was used to access database entries for these journals. Key search terms were “poor sleep” or “insomnia”, and a large number of results was found initially (24, 782 search results). These results were filtered by selecting article types which were published in English, and we selected those studies based on adults (see Figure 38). We further refined these results to identify those papers where an insomnia sample was compared against controls, and 64 abstracts were then manually reviewed (Figure 38). These papers were all published from 2005 – present, following the publication of the RDC in 2004. As the focus of this

review was on methods of assessment, sample size was not considered as an exclusion criterion.

Figure 38. Literature search strategy



Those papers without a suitable control group were excluded (for example, intervention studies), giving a final sample of 37 (Table 18.). All papers included an insomnia patient group, and the majority (30) used patients with primary insomnia. Data were extracted by selecting those methods relevant to each of the 5 criteria in the RDC. In general, specific details as to insomnia, and methods of sleep assessment, were coded within Criterion A. Information relevant to circadian rhythm sleep disorders and test time, as well as work and travel, was contained within Criterion B. In keeping with the RDC, methods relevant to physical and psychiatric health, medication use and substance abuse, and sleep disorders in general, were coded separately under Criteria C, D and E. All data were coded as described in the original papers; and not subject to interpretation at initial encoding.

Table I8. Summary of papers meeting inclusion criteria

	1st Author	Year	Control N	Age	Gender	Insomnia patients
1	Bastien	2013	30	35.8 (9.1)	18F, 12M	Psychophysiological insomnia, paradoxical insomnia
2	Huang	2012	48	38 (12)	28F, 20M	Primary insomnia
3	Israel	2012	22	26.5 (7.3)	19F, 3M	Primary insomnia
4	Morgan	2012	17	36 (9)	9F, 8M	Primary insomnia
5	Corsi-Cabrera	2012	10	25.6 (4.6)	5F, 5M	Primary insomnia
6	Forget	2011	12	44.3 (9.4)	7F, 7M	Primary insomnia
7	De Zambotti	2011	8	23.23 (3.24)	5F, 3M	Primary insomnia
8	Nissen	2011	53	46.9 (4.65)	32F, 21M	Primary insomnia
9	Spiegelhalder	2011	46	37.3 (11.4)	27F, 19M	Primary insomnia
10	Manconi	2010	288	58.5 (7.23)	176F, 112M	Primary insomnia

11	Winkelman	2010	15	38.8 (5.3)	6F, 9M	Primary insomnia
12	Deuschle	2010	827	54.6 (17.2)	455F, 372M	Primary insomnia
13	Spiegelhalder	2010	30	48.3 (12.9)	21F, 9M	Primary insomnia
14	Parrino	2009	20	45 (8)	16F, 4M	Paradoxical insomia
15	Lanfranchi	2009	13	42 (9)	9F, 4M	Primary insomnia
16	Buysse	2008	25	30.6 (7.4)	15F, 10M	Primary insomnia
17	Winkelman	2008	16	37.6 (4.5)	7F, 9M	Primary insomnia
18	Feige	2008	100	41.12 (13.99)	54F, 36M	Primary insomnia
19	Spiegelhalder	2008	20	38.6 (10.1)	12F, 8M	Primary insomnia
20	Bastien	2008	16	36.81 (10.19)	10F, 6M	Psychophysiological insomnia
21	Edinger	2008	84	48.6 (16.8)	41F, 43M	Primary insomnia
22	Sagaspe	2007	13	45 (12)	5F, 8M	Psychophysiological insomnia
23	Orff	2007	17	36.1 (7.1)	13F, 4M	Primary insomnia
24	Riemann	2007	8	46.3 (14.3)	5F, 3M	Primary insomnia
25	Robertson	2007	15	27.7 (7.05)	8F, 7M	Psychophysiological insomnia
26	Yang	2007	15	34.3 (12.9)	10F, 5M	Primary insomnia
27	Buysse	2007	18	27.2 (7.9)	15F, 3M	Primary insomnia
28	MacMahon	2006	20	28.2 (10.1)	11F, 9M	Primary insomnia
29	Ouellet	2006	14	30.00 (10.05)	5F, 9M	Insomnia syndrome (DSM-

						IV and ICSD)
30	Nissen	2006	7	44.9 (4.1)	4F, 3M	Primary insomnia
31	Marchetti	2006	30	23.2 (1.69)	15F, 15M	Psychophysiological insomnia
32	Carney	2006	104	47.3 (16.8)	52F, 52M	Primary insomnia
33	Lineberger	2006	88	45.39 (16.59)	44F, 44M	Primary insomnia
34	Rioux	2006	11	48.00 (7.86)	5F, 6M	Primary insomnia
35	Salin-Pascual	2006	6	26.6 (5.0)	4F, 2M	Primary insomnia
36	Thacher	2006	10	34.7 (7.9)	7F, 3M	Primary insomnia
37	Devoto	2005	7	22.6 (2)	4F, 3M	Primary insomnia

Results

Criterion A

We recorded how control groups were defined with regards to Criterion A, i.e. “the individual has no complaints of sleep disturbance or daytime symptoms attributable to unsatisfactory sleep”. Firstly, the definitions applied to control subjects are summarized. These definitions varied, from “healthy”, “normal/good sleepers”, and “typically good sleepers”, and included descriptions such as no subjective complaints of sleep difficulties or insomnia, or sleep or insomnia complaints. More detailed definitions included that subjects characterize their sleep as restorative or refreshing, sleep satisfaction, relatively imperturbable sleep, and falling asleep as soon as your head touches the pillow. Additional specifications included that subjects report no history of sleep disorders or insomnia, either current or in the past, and objective sleep thresholds were also used. Sleep questionnaires can be used to quantify sleep-related thresholds, and 5% of studies reported cut-off scores or descriptive information for the Pittsburgh Sleep Quality Index (PSQI), with the Insomnia Severity Index (ISI) similarly used by 30% of papers. Many studies (51%) reported sleep diary parameters of control subjects, and a majority of studies (65%) reported descriptive sleep information from PSG measures, with 2 studies (5%) reporting actigraphy-derived sleep parameters.

In terms of meeting criteria A, we required papers to have explicit exclusion criteria based on both measures of sleep disruption (i.e. sleep continuity), and daytime effects (e.g. report sleep as restorative). In total 8% of papers met this criterion.

Criterion B

Criterion B is defined as an individual having "a routine standard sleep/wake schedule characterized by regular bedtimes and rising times". To assess this, we recorded information relevant to sleep timing within the articles. Four studies (11%) reported average bed times/rise times, and the range of subjects' sleep timing was reported by 30% of studies, either descriptively, or as inclusion criterion (e.g. to confirm consistency of habitual sleep patterns with a specified sleep laboratory schedule). One study reported an actigraphy-derived measure of circadian phase, and another reported a measure of diurnal preference. Other relevant exclusion criteria included shift working patterns and long range travel, as well as circadian rhythm sleep disorders or abnormal usual sleep schedules. With regards to meeting criteria for sleep timing, adherence to this criterion was defined by explicit exclusion criteria for sleep timing, i.e. bed times and rise times, and met by 30% of papers.

Criterion C

Criterion C is defined as "no evidence of a sleep-disruptive medical or mental disorder".

Table 19. Summary of adherence to RDC Criteria. '+' indicates full adherence to criteria, and '-' indicates apparent non-adherence.

	1st Author	Year	Criterion A	Criterion B	Criterion C	Criterion D	Criterion E
1	Bastien	2013	-	-	+	+	+
2	Huang	2012	-	-	+	-	+
3	Israel	2012	-	-	+	+	+
4	Morgan	2012	-	-	+	+	-
5	Corsi-Cabrera	2012	-	+	-	-	+
6	Forget	2011	-	-	+	-	+
7	De Zambotti	2011	-	+	+	+	-
8	Nissen	2011	-	-	+	-	-
9	Spiegelhalder	2011	-	+	+	-	+
10	Manconi	2010	-	-	-	-	-
11	Winkelman	2010	-	+	+	+	-

12	Deuschle	2010	-	-	-	-	-
13	Spiegelhalder	2010	-	-	-	-	-
14	Parrino	2009	-	-	-	-	+
15	Lanfranchi	2009	-	+	+	+	+
16	Buysse	2008	-	-	+	+	+
17	Winkelman	2008	-	+	+	+	+
18	Feige	2008	-	+	+	+	+
19	Spiegelhalder	2008	-	-	-	-	-
20	Bastien	2008	+	-	+	+	+
21	Edinger	2008	-	-	+	-	+
22	Sagaspe	2007	-	+	-	-	+
23	Orff	2007	-	+	+	+	+
24	Riemann	2007	-	-	+	-	-
25	Robertson	2007	-	-	+	+	-
26	Yang	2007	-	+	+	+	+
27	Buysse	2007	-	-	+	+	+
28	MacMahon	2006	-	-	-	-	-
29	Ouellet	2006	-	+	+	-	-
30	Nissen	2006	-	-	+	-	-
31	Marchetti	2006	+	-	-	-	-
32	Carney	2006	-	-	+	-	+
33	Lineberger	2006	-	-	+	+	+
34	Rioux	2006	+	-	+	-	-
35	Salin-Pascual	2006	-	-	-	-	-
36	Thacher	2006	-	-	+	+	+
37	Devoto	2005	-	-	-	-	-

A majority of studies applied general medical examinations, which were used to assess health, and the absence of signs or symptoms of a disorder (e.g. blood screening tests). Twelve studies (32%) also reported BMI scores, and 56% used or reported data from additional questionnaire screening measures for symptoms of mental health conditions, such as depression or anxiety. At least one medical condition was excluded for by 84% of studies, and specific disorders listed included unstable hypertension, thyroid disorders, seizure disorders, neurodegenerative disease, chronic pain, significant head trauma or loss of consciousness, cardiovascular or respiratory disease, diabetes, dementia, multiple sclerosis, pregnancy, hepatitis, cancer, Parkinson's disease, rheumatoid arthritis, and gastroesophagael reflux disease, asthma, chronic obstructive pulmonary disease. At least one psychiatric disorder was excluded by 86% of studies, which

included mood disorders, psychotic disorders, anxiety disorders, eating disorders, somatoform disorders, and substance abuse disorder (this latter is considered in more detail under Criterion D). When this criterion was judged via exclusions for medical and psychiatric disorders, 70% of papers met this criterion.

Criterion D

Criterion D is defined as "no evidence of sleep disruption due to a substance exposure, use, abuse, or withdrawal". Generally, articles assessed subjects for evidence of a disorder which would be relevant to this criterion. In total, 76% of articles reported exclusion criteria as to medication use, and most commonly selected those subjects who were either not on medication, not using medication affecting sleep, or taking CNS-active agent, psychotropic agents, or hypnotics. Drug abuse or dependence was excluded by 57% of studies, with alcohol, caffeine, or nicotine consumption mentioned by 59% of articles. When D was defined as explicit exclusions for substance abuse and medication use, 43% of studies met criterion.

Criterion E

The final criterion is "no evidence of a primary sleep disorder". A number of studies (59%) reported PSG screening for sleep apnea and limb movements in control subjects. Evidence of sleep disruption, or other sleep disorders, was assessed by 76% of articles and included evidence of current disorder, evidence of symptoms, and/or past (or family) history. In addition, other disorders (e.g. nocturia, enuresis, bruxism) were mentioned occasionally. When this criterion was defined as explicit exclusion criteria for sleep disorders in conjunction with PSG, 54% of papers met criteria.

In total, no papers were judged to meet all five criteria. 16% met four criteria, 24% of papers met three criteria, 27% met two criteria, and 14% met one criterion, with 19% of papers meeting none.

Discussion

Overall, the selected articles screened subjects well for potential disorders (whether physical, psychiatric, or sleep). However, the criteria applied to control subjects differed between studies, and information relevant to criteria seemed to be used to describe subjects groups, rather than as explicit a priori exclusion criteria as such. There are also differences between laboratories as to how exactly subjects are identified, and Criteria A and B seem to require clarity. While Edinger et al. (2004) define A as "no complaints of sleep disturbance, or daytime symptoms attributable to poor sleep", there is a lack of consensus as to how exactly this should be defined. For

example, some specify sleep diary criteria, while others use questionnaire cut-offs, and/or a lack of 'sleep complaint', or absence of insomnia disorder as such. We would interpret A as comprising three main elements, firstly, whether an individual is experiencing sleep disturbance (i.e. via sleep duration or sleep continuity measures). Secondly, whether subjects are satisfied with their sleep, and experience good sleep quality. In addition to this, the experience of adverse sleep-related daytime effects would lead to the exclusion of control subjects.

Criterion B, defined as "a routine standard sleep/wake schedule characterized by regular bedtimes and rising times" (Edinger et al., 2004) seems to comprise several components, in particular, the habitual timing of sleep, and its stability. Evidence of circadian rhythm sleep disorders often seemed to be used to assess this criterion, overlapping with Criterion E. While a majority of authors reported use of sleep diaries, which can be used to assess this, it is often unclear exactly how these components are defined in practice. Many authors defined normal sleep timing parameters, although normality would seem to depend on the study sample, and would be affected, for example, by age (Ohayon et al., 2004). Sleep timing, chronotype, and sleep quality seem to be interlinked (Barclay et al., 2005; Monk et al., 2003; Barclay et al., 2010). Furthermore, while the stability of sleep timing seems to be important for control subjects, this component seems to be rarely directly addressed, other than by shift work, and it is unclear whether any exclusions were made based on sleep timing stability as such. The variability, or stability, of sleep timing could also contribute to good sleep (e.g., Espie, 2002), and differences in sleep timing between the working week and the weekend could contribute to variability in sleep timing (Monk et al., 2003), and social jetlag (Wittman et al., 2006; Roenneberg et al., 2003). Questionnaire measures such as the Sleep Timing Questionnaire (Monk et al., 2003) could be used to quantify the components of sleep timing, as could measures derived from sleep diary parameters. We would define B as conventional (for a particular population) bed times and rise times, which are consistent ($\pm$ one hour, at least four days a week).

For the remaining criteria, there seems to be some ambiguity as to the precise definitions. Furthermore, clear definitions are needed in order to standardize methods and measures. For example, we would interpret C as a currently diagnosable serious medical or mental disorder. We would define Criterion D by the abuse of substances (e.g. alcohol, caffeine, nicotine, or drugs), or by the use of prescription medication. Lastly, we would define E as a currently diagnosable sleep disorder, i.e. narcolepsy/cataplexy, periodic limb movements or restless legs, parasomnias, circadian rhythm sleep disorders, and insomnia disorder, and in conjunction with PSG screening

(e.g. for sleep apnea and periodic limb movements). Moreover, the presence of a significant health disorder, without associated sleep disruption, may be unlikely given the overlap of sleep with general health (Luyster et al., 2012 ; Spiegel et al., 2009). While the final three components (C, D and E) seem particularly applicable within a medical setting, they may not be appropriate or necessary for use in all research settings, to ensure normal sleepers are selected.

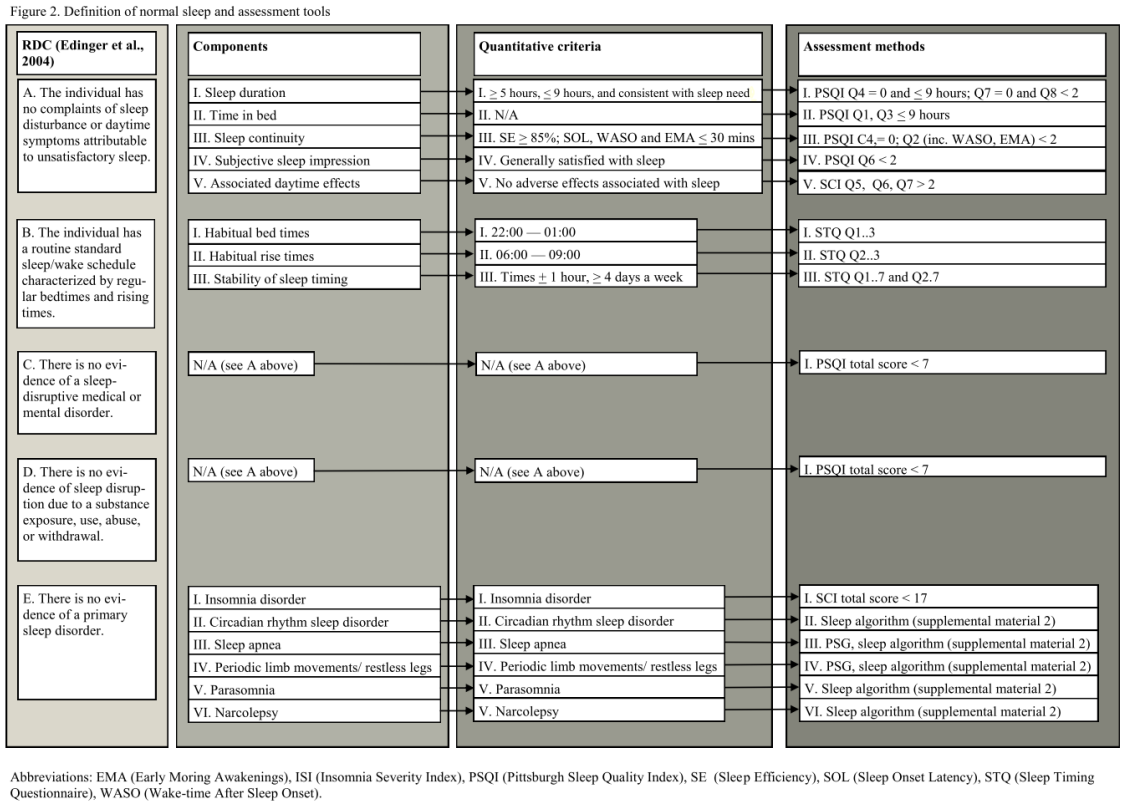
Defining normal sleep: A research agenda

All of these components can be assessed in different ways, such as by simple self-reports (e.g. do you have insomnia?), a personal history (e.g. a previous diagnosis), evidence of symptoms (e.g. screening measures or PSG), and a diagnostic clinical interview by trained experts. The precise levels of assessment applied would depend on, for example, the number of subjects to be tested, access to resources (e.g. PSG, laboratory facilities), and the experience of the researcher (e.g. in diagnosing the presence of a disorder). To aid standardization across the field, we suggest that precise definitions, exclusion criteria, and descriptive information, are reported as much as possible. Furthermore, the use of standardized methods, such as the consensus sleep diary (Carney et al., 2012), will help aid comparisons between studies, as the precise contents of sleep diaries can vary between laboratories. In addition, Edinger et al. (2004) recommend reporting information as to the methods of recruitment and types of individuals, and the criteria for normal sleep may need to be tailored, for example, in elderly subjects (Ohayon et al., 2004). Here we suggest specific assessment tools thought sufficient to identify normal sleepers, favouring questionnaire methods and aiming to reduce the burden on participants as much as possible (see Figure 39).

Means, standard deviations, and ranges are also recommended to be reported for common sleep measures, as well as quantitative thresholds, and the measures from which these are derived. Indeed, we found sleep diaries to be often used, although full descriptive information, as stated above, was not always reported. Edinger et al. (2004) report that most insomnia studies describe control subjects as being without sleep complaints or insomnia. Exclusions were found to be made for medical disorders which commonly affect sleep (~50%), symptoms of psychiatric disorder (~42%), psychoactive agents (~23%), evidence of sleep timing disruption (~15%), normal sleep values (~8.5%), or primary sleep disorders (<4%), and over 85% of samples were selected based on less than three of these criteria; results which also appear broadly consistent with the present review. However, in our sample of primary or physiological insomnia patients and controls, these values are higher overall, and in both studies sleep timing measures

are among the least reported. Here we suggest a definition of normal sleep, for use with control subjects in contrast to patients, and in studies of healthy sleepers (e.g. sleep deprivation paradigms). Furthermore, this may be of particular importance in student populations, whose sleep has been described as “erratic” (Lund et al., 2010).

Figure 39. Suggested criteria and assessment of normal sleepers



With regards to specific criteria as to normal sleep parameters, studies of insomnia have previously defined criteria for normal sleepers (Espie et al., 2012; Kyle et al., 2014). For example, papers may define sleep parameter thresholds for insomnia subjects, such as a sleep-onset latency or wake-time after sleep onset duration of greater than 30 minutes, total sleep time less than six hours, and sleep efficiency less than 85% (Kyle et al., 2014; Lineberger et al., 2006), and it could be possible to extrapolate criteria for normal sleepers from such reports. Furthermore, studies of sleep deprivation and epidemiological studies provide evidence of the effects of sleep manipulations, and of normal ranges within the general population. In the absence of existing specifications for normal sleep, we would suggest the following definition and possible measurement tools.

Firstly, an individual does not meet criteria for an existing sleep disorder (i.e. insomnia disorder, circadian rhythm sleep disorder, sleep apnea, narcolepsy/cataplexy, periodic limb movements or restless legs syndrome, or a parasomnia). We suggest that the

presence of periodic limb movements or restless legs syndrome, and sleep apnea should be assessed via polysomnography. Espie (Wilson et al., 2010) has developed a screening algorithm for CRSD, parasomnias, restless legs syndrome or periodic limb movements, sleep apnea, and narcolepsy. The ISI (Bastien, Vallieres and Morin, 2001) can be used to assess the severity of insomnia symptoms in those with a sleep complaint, and sleep complaints together with daytime sleepiness may be indicative of a circadian rhythm sleep disorder (CRSD; Doghamji et al., 2004). The sleep disorders questionnaire can also be used to assess sleep apnea, narcolepsy, and restless legs syndrome or periodic limb movements (Douglass et al., 1994). The diagnosis of narcolepsy without cataplexy has been described in greater depth in 2014 (Baumann et al., 2014). In order to reduce the questionnaire burden on research participants, we suggest that the brief screening algorithm developed by Espie should be used to identify the likely presence of narcolepsy, parasomnias, and circadian rhythm sleep disorders, and used to confirm a lack of sleep apnea, periodic limb movements or restless legs syndrome. However, the questions here are minimal, and PSG would provide a higher level of evidence. Furthermore, actigraphy can be used to assess CRSD, and the Sleep Condition Indicator (Espie et al., 2014) can be used to screen for insomnia disorder.

Secondly, an individual should report no adverse daytime effects associated with poor sleep, at least within the previous week. Questionnaire measures could also be used to assess this, such as question 7 of the ISI (Bastien, Vallieres and Morin, 2001), or component 7 of the PSQI (Buysse et al., 1989). Thirdly, an individual should report general satisfaction with their sleep, which can be assessed via the subjective components of a sleep diary (Carney et al., 2012), component 1 of the PSQI (Buysse et al., 1989), or question 4 of the ISI (Bastien, Vallieres and Morin, 2001). We suggest that question 6 of the PSQI – 'During the past month, how would you rate your sleep quality overall?' should be used to assess general sleep satisfaction. For no adverse daytime effects of poor sleep, PSQI questions 7, 8 and 9 could be used, with complaints less frequently than once or twice a week. Alternatively, ISI question 3 could be used to assess this, as could questions 5, 6, and 7 of the Sleep Condition Indicator (Espie et al., 2014). Fourthly, we suggest specific definitions of sleep parameters which we would suggest are indicative of normal sleep. In particular, we suggest thresholds for sleep duration, sleep continuity, time in bed, and sleep timing.

Typical sleep duration criteria are included, in keeping with a recent description of sleep health (Buysse, 2013), and as short sleep duration/sleep restriction is linked to negative effects on health (Luyster et al., 2012; Spiegel et al., 2009; Meerlo, Sgoifo and Suchecki, 2008) and mortality (Kripke et al., 2011). Individuals with insomnia, who also

have a short sleep duration, also seem to experience a more severe disorder (depression, heart and metabolic health; Vgontzas et al., 2013). As an excessive sleep need, or time in bed, can be indicative of mood disorders (Kaplan and Harvey, 2009), we would define normal sleep by a sleep duration of less than nine hours a night, and more than five hours a night (in the absence of diminished sleep continuity). Furthermore the amount of sleep typically achieved should be consistent with sleep need (c.f. Luyster et al., 2012; Buysee, 2014), and this is affected by factors such as age (Lund et al., 2010; Ohanyon et al., 2004).

Sleep restriction also affects the ability to judge sleep need well (Van Dongen et al., 2003). For example, a study with "naturally short sleepers" found that many potential subjects reported that their short sleep duration was associated with work or care-giving, or poor physical or mental health (Monk et al., 2001). Indicators of naturally short sleep duration included that these sleepers did not seem to be making up for lost sleep at weekends, and had identical Epworth Sleepiness Scale (ESS) scores as control subjects. These subjects slept on average for 6 hours a night or less, and were found to show significantly greater evidence of hypomanic symptoms (Monk et al., 2001). These studies taken together indicate that there are few people who are naturally short sleepers, and those who are evidence signs of mood disruption. However, these guidelines will require testing and ultimately there will be a trade-off between sensitivity and specificity. We would suggest that sleep timing be assessed via questions one and three of the PSQI, or via the Sleep Timing Questionnaire (Monk et al., 2003; STQ). Sleep duration could be assessed via the PSQI, with question 8 (daytime sleepiness) used to assess whether sleep need is being met. These measures combined implicitly set limits on time in bed.

On measures of sleep continuity, sleep onset latency, wake time after sleep onset, and early morning awakenings should each be less than 30 minutes, with a sleep efficiency of greater than 85%. These components can be assessed via sleep diary (Carney et al., 2012) or the PSQI (Buysse et al., 1989). With regards to sleep scheduling, ordinarily, the timing of sleep should be consistent with a 9am-5pm work pattern. We would suggest a typical bed-time of between 22:00-01:00, with a rise time of 06:00-09:00. Furthermore, these times should not vary markedly, with sleep times consistent, within an hour, most days a week. The Sleep Timing Questionnaire (Monk et al., 2003), and sleep diaries (Carney et al., 2012), can be used to assess sleep timing and stability. However, ideally, all components of normal sleep could be captured by the use of a single measure, and sleep diaries are not always practical. While a comprehensive definition of good sleep in contrast to normal sleep is beyond the scope of the present review, we suggest

documenting applied criteria, to allow for future work in this area (see Figure 3). Furthermore, these components are consistent with those recently identified by Buysse, (2014) (sleep duration, efficiency/continuity, timing, alertness, and satisfaction), as being important for sleep health. A sleep health questionnaire was also described (Buysse, 2014).

Additional criteria might be needed for the screening/selection of good sleepers, such as the endorsement of good sleep, alongside the absence of complaint. Good sleepers and normal sleepers could be somewhat different subject groups, and this could be worth investigating further. For example, three hypotheses may be made as to their differences. Firstly, good sleepers may be less likely to report or experience sleep disruption. Secondly, the effects of sleep loss on daytime functioning could be less severe, or minimal, for good sleepers. Thirdly, good sleepers could have a general resilience against poor health and towards well-being. For example, the Ford Insomnia Response to Stress Test (FIRST) can be used to assess vulnerability towards sleep disruption (Drake et al., 2004), and the importance of sleep adaptability has been recognized theoretically (Espie, 2002; Kaplan and Harvey, 2009). Understanding this resilience to poor sleep/insomnia could have important implications for individuals and organizations, where sleep disruption may be expected.

As a result of this review, we have developed the Revised Research Criteria for Defining Normal Sleeper Controls (Figure 40.) for use with control subjects. Here, we suggest four main components of normal sleep, i.e. sleep quality, sleep timing, sleep duration, and general health. These correspond broadly to each of the 5 components previously identified (Edinger et al., 2004). We would define sleep quality with three subcomponents, which are: sleep duration and continuity, subjective sleep impression, and its impact on functioning. Sleep timing includes habitual bed and rise times, and their impact, as well as sleep timing stability. With regards to other sleep disorders, four key sleep disorders are most relevant to screen for, i.e. narcolepsy/cataplexy, sleep disordered breathing or sleep apnea, parasomnias, and restless legs and periodic limb movements. Insomnia disorder and circadian rhythm sleep disorders can be assumed to be covered within sleep quality and sleep timing. Under general health, we have combined C and D of the RDC, and included mental and physical health with medication use and substance abuse. However, extreme levels of substance abuse would overlap with mental health (i.e. at the level of a substance abuse disorder), and substance abuse would include illicit drugs as well as, for example, nicotine, alcohol, and caffeine.

Figure 40. Revised research diagnostic criteria for selecting normal sleeper controls

In summary, while results suggest that in general the methods of assessing normal sleepers cover the key components of normal sleep as specified by Edinger et al. (2004), there is variability in the exact procedures used by different laboratories. However, an important limitation of the present results is that review papers used a well-defined insomnia sample, and results may differ if a broader inclusion criterion was used. It should also be noted that some of the studies reviewed may have begun before the publication of the RDC in 2004, and were published afterwards. Nonetheless, even within the current sample important issues in identifying controls were identified. Different fields could also apply different definitions to normal sleepers/control subjects, and we agree with Edinger et al. (2004) that "due to this lack of standardization, synthesizing results of multiple … studies is a difficult if not impossible task". As a first step, greater reporting of descriptive sleep information would aid in clarifying the exact nature of control groups. If screening of sleep disruption and timing can be clarified, additional methods could be redundant, and this would help reduce the burden on controls. While existing measures, such as the Pittsburgh Sleep Quality Index (Buysse et al., 1989) and Insomnia Severity Index (Bastien, Vallieres and Morin, 2001), provide ranges for a lack of sleep disruption, these measures are not used consistently. Furthermore, as predominately global measures, these questionnaires do not tend to be reported at the item-level, and do not address all components of the RDC (Edinger et al., 2004). The use of this criterion for normal sleep would help clarify how the components of the RDC are assessed, aiding the understanding of how insomnia develops, as well as the nature of good sleep itself, by helping to standardize this field.

Chapter 9

General Discussion

Synthesis

Study one suggests that the previous night's sleep affects emotion identification from static faces, with regards to happiness recognition. Depression scores also contributed towards the accuracy of recognizing happiness, although effects of sleep group were not significant. There were no effects of sleep on a measure of subjective empathy. Results from Study Two suggest that insomnia disorder is linked to impairments in dynamic emotion recognition, with the categorization of high-intensity expressions of sadness and low-intensity expressions of surprise. However there were no significant effects on reaction times or intensity ratings. These results appear comparable with previous results by Kyle et al. (2014) who found impaired perception of intensity in patients with psychophysiological insomnia. These effects were found with the intensity ratings of static fearful and sad faces. Psychological distress was found to contribute towards these results.

In general, poor sleep and depression contribute towards happiness recognition, and poor sleep is associated with greater levels of psychological distress. Insomnia also affects the recognition of sadness and surprise, with no effects on intensity ratings or reaction times. However, there was evidence that emotional understanding was affected by insomnia. In patients with psychophysiological insomnia, faces displaying sadness and fear are rated as less intense, with no effects on categorization accuracy, and psychological distress contributes towards results. These results taken together suggest that emotion recognition is impaired with insomnia, in particular, the perception of intensity or recognition of faces with varying levels of intensity is affected. Poor sleep in general does not seem to affect results, although measures of the previous night's sleep and depression were found to affect happiness recognition. This is perhaps in keeping with a study by Van Der Helm et al. (2010) which found sleep deprivation was associated with impaired intensity ratings in the mid-intensity range of faces depicting anger and happiness. Sleep loss may contribute towards mood dysregulation and insomnia, resulting in the greater impairments in emotion perception in subjects with insomnia disorder (Experiment Two and Kyle et al., 2014), and may be in keeping with a role of hyperarousal in insomnia disorder. Future studies would be required to investigate the precise interactions of sleep disruption, insomnia, and psychological distress with emotion recognition. For example, the relationships between

sleep and arousal could become altered as insomnia disorder develops, and emotion perception could contribute towards this and/or indicate it.

We then sought to better characterize the relationship of sleep with emotion recognition in normal sleepers. Chapter 5 Experiment One found that time since waking affected sensitivity towards temporal parameters, and intensity ratings of mid-intensity expressions of sadness and anger when dynamic faces were used. In Chapter 5 Experiment One, the early group were more sensitive to the temporal parameters, whereas in Experiment Two the early group rated the emotional faces as less intense. Differences in the direction of results are attributed to the time of testing in the late group, with associated differences in test setting and physiological phase. These results were not replicated with emotion recognition in static faces across the day (Chapter 6), suggesting that the effects of time since waking are subtle, and sensitive to task characteristics. We suggest that testing participants in the early evening or later requires that results be interpreted with caution, as results may be different when testing takes place during the typical working day. Such testing would ideally include measures of physiological phase. Chapter 7 investigated whether the previous results with emotion face recognition extend to additional tasks of theory of mind. Results suggest that insomnia disorder is associated with speeded responses on the false belief task but slowed performance on the perspective taking task. There were no effects on accuracy or subjective empathy. Results are interpreted as reflecting the dysregulation of arousal in insomnia disorder, with evidence of both hyperarousal and fatigue in these participants. The variability in arousal of normal sleepers also needs to be considered when testing participants later on in the day. These results are in keeping with reports of subjective daytime impairments in insomnia disorder (Kyle, Espie and Morgan, 2010). Results also suggest that future studies would benefit from a well-characterized control group and specific test times; otherwise the directionality of results may be misleading.

In general, results suggest that sleep affects emotion perception in both normal sleepers and insomnia disorder (see Table 20 below).

Table 20. Summary of thesis results

	Normal sleepers (students)	**General (student) population**	**Insomnia disorder (student sample)**	**Insomnia disorder (UGSC sample)**
Results	- No effects of test time in reaction times of normal sleepers to emotional images or faces, despite group differences in reported global vigour, when static faces are used. Some evidence that the early (three hour) group make more error with happiness (Chapter 6). - Significant group differences in sensitivity towards temporal parameters, with the early group more sensitive. No effects on categorization labels or intensity ratings, when dynamic stimuli are used (repeated testing). No group differences on subjective mood or sleepiness (Chapter 5, Experiment One). - Significant group differences in intensity ratings, of mid-intensity sad and angry faces, with the early group less sensitive. No significant effects with categorization labels, when dynamic stimuli are used (repeated testing. Significant group	- Previous nights' sleep and depression scores affect happiness recognition, with effects of previous nights' sleep on happiness reaction times. There were no significant effects on subjective empathy. No effects of sleep groups derived from PSQI scores sampled as a continuous variable (Chapter 3).	- Significant effects of insomnia disorder on emotion categorization, when dynamic faces are used. In particular these subjects were less accurate in recognizing sad faces of high intensity and surprise faces of low intensity. There were no significant effects of intensity judgements or reaction times. Regression analyses suggest effects of sleep and mood on happiness recognition (Chapter 4). - Significant effects of insomnia disorder on theory-of-mind task performance. In particular the insomnia subjects were faster to respond in the false belief task, but slower to respond in the perspective-taking task. There were no effects on accuracy (Chapter 7).	- Psychophysiological insomnia was linked to lower intensity ratings of sad and fearful faces. There were no effects on categorization (dynamic faces). Results were not linked to sleepiness (vigilance task performance or ratings) (Kyle, Beattie et al., 2014). - Primary insomnia linked to perceived social effects and exclusion, as well as a lack of understanding (Kyle, Espie and Morgan, 2010).

	differences in reported alert cognition and sleepiness/fatigue (Chapter 5 Experiment Two).			
Summary	Emotion perception changes between morning and evening, with late evening subjects more sensitive with regards to intensity ratings of mid-intensity sad and angry 4D faces. This seems to correspond to differences in subjective alertness. Early evening participants seem to be less sensitive with regards to sensitivity towards temporal parameters in 4D faces, with no group differences in alertness. Some evidence of more errors when categorizing happy static faces in the early group, with significant group differences in global vigour.	Depression scores and the previous nights' sleep affect happiness recognition error rates, with effects of the previous nights' sleep on happiness recognition reaction times.	Impaired categorization accuracy of 4D high intensity sad faces and low intensity surprise faces. Also significant differences in reaction times to theory-of-mind tasks.	Lower intensity ratings of sad and fearful static faces. Reported impairments in social functioning.

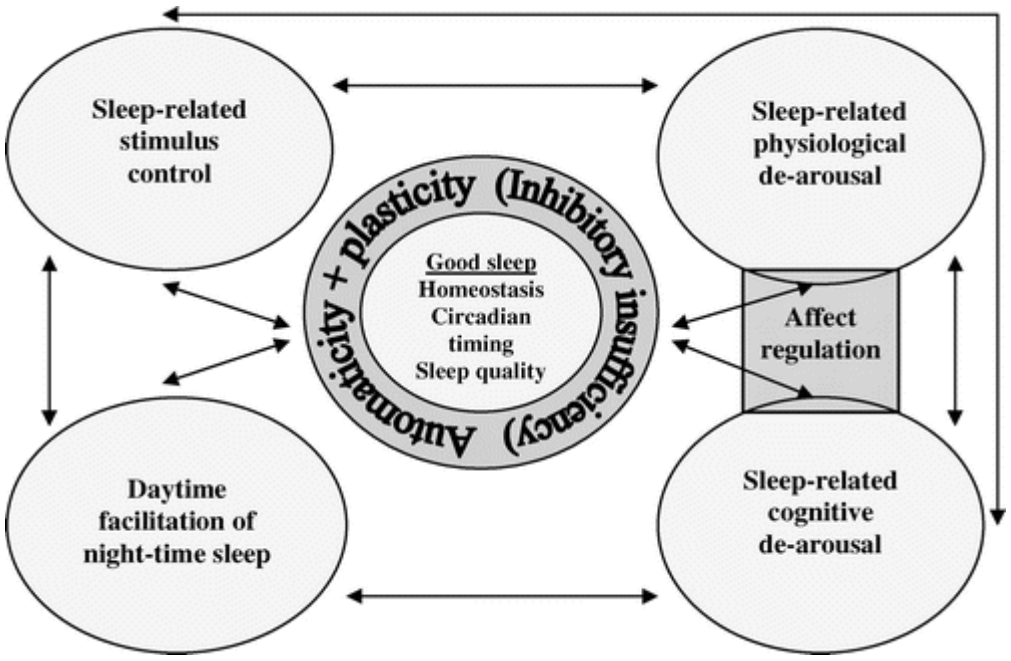
Results suggest that participants tested in the late evening are *more* sensitive when making intensity judgements of mid-intensity sad and angry dynamic faces (Chapter 5 Experiment 2). This could indicate that sleep is important in "re-calibrating" the human emotional brain (Gujar et al., 2011). The psychobiological model suggests that normal sleepers de-arouse in the lead up to sleep (Espie, 2002), and the current results may suggest that if this fails to occur the sensitivity towards specific emotions (sad and angry) is heightened. Indeed, acute stress has been found to increase amygdala activation to all emotional faces, decreasing its specificity but increasing sensitivity (Van Marle et al., 2009). Espie (2002) suggests that the inhibition of de-arousal is sufficient to result in poor sleep, and insomnia disorder may be the result of additional factors.

The maintenance of insomnia has been linked to increasingly negatively toned cognitive activity (Harvey, 2002), REM sleep instability (Riemann et al., 2012), and attentional bias towards sleep (Espie et al., 2006). In insomnia participants, hyperarousal is thought to play a key role (Riemann et al., 2010; Bonnet and Arand, 2010), and these subjects show a smaller decline in metabolism in several brain areas between waking and non-REM sleep (Nofzinger et al., 2004). These subjects also showed waking hypo-metabolism in the frontal cortex when compared to controls (Nofzinger et al., 2004). We found these subjects to make more categorization errors with dynamic high intensity sad faces and low intensity surprise faces (Chapter 4), and to make lower intensity judgements towards static sad and fearful faces (Kyle et al., 2014). As such, hyperarousal in insomnia may manifest as behavioural impairments in emotion recognition, although the manipulation/testing of intensity appears to be crucial in this.

These results therefore appear consistent with insomnia theories, whereby sleep disruption and poor sleep affect the inhibition of de-arousal, and likely create a vicious cycle whereby poor sleep is perpetuated (Espie 2002; Espie et al., 2006). An insomnia complaint is thought to develop in a minority of people who experience poor sleep, and the processes by which this occurs are still being delineated. However, "excessively negatively toned cognitive activity" is thought to contribute towards insomnia maintenance (Harvey, 2002), and the results in this thesis suggest one way in which this could occur. Specifically, people with insomnia may increasingly attend to their sleep in an attention-intention-effort process, which paradoxically inhibits sleep (Espie et al., 2006). Attention towards the daytime consequences of poor sleep is thought to perpetuate the emotional dysregulation in insomnia (Harvey, 2002), and people with insomnia report concerns with daytime functioning (Kyle, Espie, and Morgan, 2010). The results of this thesis appear to support a role of sleep in socio-emotional functioning in normal sleepers, poor sleepers, and people with insomnia. This could therefore

represent one pathway by which insomnia becomes a chronic disorder. Future studies may wish to consider the emotional consequences of these effects, and their role in emotional dysregulation.

Figure 41. A psychobiological model of good sleep. Insomnia is proposed as resulting from chronic inhibition of one or more of the component processes. Copied with permission by Espie, (2002) from Annual Review of Psychology (53).



This is consistent with patient reports of impaired functioning (Kyle, Espie and Morgan, 2010), and evidence of alterations in reaction times when performing theory of mind tasks (Chapter 7). Also relevant is the association in Kyle et al. (2014) of performance with a measure of psychological distress, rather than measures of sleepiness or sleep diary measures. This appears in contrast to our results in normal sleepers, where subjective mood/sleepiness differences were found in tasks with alterations in emotion intensity recognition (Chapter 5 Experiment 2) and evidence of impaired happiness recognition (Chapter 6). Chapter 3 with subjects in the general student population also found the previous nights' sleep to be linked to happiness recognition, although depression scores also contributed. However, it should be noted that the late group in Chapter 5 Experiment One were *less* sensitive towards the temporal parameters in dynamic faces, with no effects on recognition. Future studies should assess the links of temporal parameter sensitivity with emotion recognition, and these subjects are anticipated to have been tested at a different physiological phase than those subjects in Chapter 5 Experiment Two.

Also of interest is the result that happiness recognition (accuracy and reaction times) from static faces in a general population sample is linked to the previous nights' sleep (Chapter 3). Happiness is typically associated with high recognition rates (Calvo and Lundqvist, 2008). Happiness recognition would be expected to be linked to depression, and indeed depression was a predictor of reaction times with happy faces (Chapter 3). Relatedly, there was evidence that those normal sleepers tested early in the day made more errors when recognizing static happy faces (Chapter 6). This might suggest that that the effects of sleep in normal sleepers impact upon mood. Phillips et al. (2003) suggest that stimuli are appraised leading to an affective state, and that emotional regulation affects both the affective state and stimuli appraisal. The effects of sleep disruption on mood are well known (Baglioni et al., 2010), and the sleep deprivation of healthy subjects impairs emotional regulation (Yoo et al., 2007). For example, sleep disruption prior to trauma increases the likelihood that healthy subjects will develop a psychiatric disorder (Bryant et al., 2010). We also report evidence that sadness recognition is impaired in insomnia (Chapter 4; Kyle et al., 2014). There is some evidence that the recognition of both happy and sad faces is impaired in depression (Bourke, Douglas and Porter, 2010). Although the links of emotion and sleep are complex (Kahn, Sheppes and Sadeh, 2013), there is evidence that insomnia and depression are linked (Staner, 2010), and insomnia can predict the occurrence of depression (Baglioni et al., 2011). In general, alterations in emotion recognition and theory of mind performance may constitute a perpetuating factor by which insomnia is maintained (c.f. Harvey, 2002).

The role of intensity also appears particularly important. In general, recognition accuracy of static faces was high, and this is consistent with the literature (Calvo and Lundqvist, 2008). As a consequence, task difficulty has often been increased by researchers investigating emotion recognition and psychopathology (e.g. Csukly et al., 2009; Gilboa-Schechtman et al., 2008; Yoon, Joorman and Gotlib, 2009; Surguladze et al., 2004). For example, Cooper, Rowe, and Penton-Voak (2008) criticize the use of a 10 second emotion face presentation time, failing to repeat a previously reported effect under a more natural testing condition. Adolphs (2002) also suggests that emotions can be combined in different ways, and methods of increasing task difficulty could impair ecological validity. Yu, Garrod and Schyns (2012) have created a 4D platform of emotional expressions taking account of physiological constraints. Dynamic faces have been linked to greater arousal (Sato and Yoshikawa, 2007) and may therefore be better able to identify differences in normal sleepers and insomnia participants (Espie, 2002; Bonnet and Arand, 2010; Riemann et al., 2010). It has been suggested that intensity decisions follow on from emotion categorization, and greater intensity is greater

recognition (Utuma et al., 2009). Future studies should investigate normal emotion recognition within this 4D platform in greater depth.

Limitations

Study participants

Sleep quality is a general term which is often applied to how sleep is perceived, or sleep continuity. There is no agreed definition of 'sleep quality', and indeed this could have a different meaning for different individuals (Krystal and Edinger, 2008). As such, this can be assessed in different ways, which are dependent upon how this concept is defined. Poor sleep quality is prevalent among students, with one third reportedly staying up until 3am at least once a week (Lund et al., 2010). Delayed sleep phase disorder is also common in young adults (c.f. Crowley, Acebo, and Carskadon, 2007). As such, there was a high baseline level of sleep disruption within the current study population, and this was reflected in a higher cut-off for poor sleep on the PSQI. Normal sleepers and poor sleepers also show similarities in which factors they use to judge their sleep quality, although there are also some differences (Harvey et al., 2008). As such, it may be the case that the normal sleepers in the current sample had had previous transient experiences of poor sleep. This could be relevant for understanding the aetiology of insomnia, and could be looked at in greater depth in future studies. However, the insomnia disorder participants showed significant differences in several measures suggesting that these participants were a distinct group. Future studies could also explore the trajectory of sleep complaints in these subjects, in order to assess whether these individuals go on to develop a chronic insomnia problem.

The experience of sleep disruption is common, and has been investigated over time in the general population. Cluster analysis suggests that there are four patterns of insomnia symptoms over the adult lifespan (Green et al., 2012). Some participants report no problems getting to sleep or maintaining sleep over time, whereas others report a low likelihood of sleep latency problems but a fairly high chance of sleep maintenance difficulties. The third category of participants reported low probabilities of either sleep problem at initial sampling, but increasingly reported the presence of both sleep latency and maintenance problems at later sampling. A fourth class reported fairly high probabilities of both sleep latency and maintenance problems throughout the study (Green et al., 2012). As such, it appears that some people are consistent good sleepers over time, while others show chronically disrupted sleep. There seems to be a vulnerability to developing insomnia symptoms over the adult lifespan in the remaining

two groups, and it seems possible that the sleep and circadian effects of ageing contribute to this.

With regards to the current sample, the flexibility of the study lifestyle may contribute towards sleep disruption, as the sleep of students has been described as erratic (Lund et al., 2010). This could contribute towards the high baseline levels of sleep disruption within this sample. Insomnia disorder may also manifest differently within these subjects. In particular, the severity of insomnia seems to be somewhat lower in the current student sample, and they may be able to employ a greater range of coping strategies due to lifestyle flexibility. Future studies could therefore make greater use of student subjects in order to understand protective and vulnerability related factors in the development of chronic insomnia. This could potentially have important ramifications, as insomnia is known to precede depression (Baglioni et al., 2010), and the early prevention of insomnia could prevent future health disorders.

Understanding of emotion

Emotion has been called one of the most contentious research areas in psychology (Gross, Sheppes and Urry, 2011). With regards to emotion recognition tasks, selecting emotion labels has been described as a cognitive task, with evidence of top-down effects on the emotional brain, whereas matching faces has been described as a perceptual task (Hariri, Bookheimer and Mazziotta, 2000; Liebermann et al., 2007). Emotional labeling could be linked to emotional re-appraisal (Liebermann et al., 2007), and such tasks are akin to real-life, where the appraisal and evaluation of incoming stimuli includes deciding which emotion is being displayed from several options. A limitation of the current studies is how task instructions influenced the neural response to stimuli. Furthermore, while links between emotion perception, affect, and affect regulation have been described (e.g. Phillips et al., 2003), further work is necessary in order to evidence the precise nature of their inter-relationships. Of particular interest is the correspondence of subjective mood with emotion recognition performance, at brain and behaviour levels of response.

Multiple measures of emotionality

Facial EMG can measure subtle levels of facial expressivity (see Dimberg, 1990), and has been used to assess individual differences in emotionality (Neta, Norris and Whalen, 2009; Dimberg and Thunberg, 2007). Other physiological measures can also be used to assess emotion (e.g. Thayer and Lane, 2000; Amstadter, 2008), and the

use of multiple measures of emotion would allow assessment of their inter-relationships. This could help to clarify the nature of the emotional dsyregulation in insomnia, and the role of hyperarousal. There are also differences between people in their emotional ability, which can be linked to alexithymia (Lumley et al., 2005). Effects of mood induction on emotion task performance also have been reported (Lee et al., 2008; Chepenik, Cornew, and Farah, 2007; Trevisani, Johnson, and Carver, 2008; Schmid and Mast, 2010). Future studies could consider the role of posed and natural expressions of emotion, and their effects on both expressed emotional displays and emotional recognition. Furthermore, this would be relevant to the role of emotional displays in social signalling and experienced affect.

Role of the circadian system

While sleep loss and sleep disruption have been found to affect emotion perception, the circadian system could also exert effects, and this area has been rather less studied than the effects of sleep loss. The role of light in emotion perception, and blue light, which plays an important role in the circadian system, has been found to affect how emotional information is processed (Vandewalle et al., 2010). In particular, this spectral quality of ambient light increases brain reactivity to angry voices, whereas green light does not, without any effects with neutral stimuli (Vanderwalle et al., 2010). Hot, Leconte and Sequeira (2005) presented subjects with images of neutral scenes and high arousal negatively-valenced scenes, and found a time of day effect on the skin conductance response (SCR) to aversive images. They found a significant linear trend to unpleasant images, and a significant quadratic trend to neutral images, across the day. Circadian effects have been investigated via measures of diurnal preference, with Paradee et al. (2008) reporting a significant effect of chronotype-congruent test times in overall emotion recognition performance among a group of rehabilitation patients. Emotion performance has also been assessed at different times of day in repeated-sampling of the skin-conductance response (SCR). Future studies could consider in greater depth the role of the circadian system in emotion recognition and social interactions, in both normal sleepers and people with insomnia

Future Directions

Facial expressions in context

The emotional context of facial expressions affects how they are recognised (Righart and de Gelder, 2008; Aviezer et al., 2008). The effects of such cues may be especially pertinent in psychopathology, such as anxiety (Blanchette, Richards, and Cross, 2007) and depression (Blysma, Morris and Rottenberg, 2008). Future studies could therefore investigate the role of the emotional setting in emotion recognition. Facial expressions are processed in conjunction with other cues, and bodily cues, such as gaze (Ewbank, Fox, and Calder, 2010; Bindemann, Burton and Langton, 2008) and bodily signals (Aviezer, Trope, and Todorov, 2012), contribute to the emotional setting. The integration of these cues likely involves multi-modal processing (e.g. Calder and Young, 2005). The social context also affects emotional expressivity, and this depends on whether an individual is alone or with other people (Schmidt, Cohn and Tian, 2003).

Effects of pharmacology

The administration of pharmacological agents can also shed light on the mechanisms by which effects occur. Future studies could also explore the effects of pharmacology on emotion recognition, as insomnia is often comorbid with other conditions. Modafinil has also been found to improve complex emotion recognition after sleep deprivation, although learning could have contributed towards these results (Huck et al., 2008). Several research groups have sought to examine the role of neurotransmitter pathways in emotion perception, both in healthy subjects and psychiatric disorders. For example, impulsivity has been linked to the ventral striatum, and the dopamine pathway, as well as its related functional polymorphisms (Hariri, 2009). Furthermore, individual differences in temperament and trait affect are important in complex human behaviours such as social interactions, and could contribute towards the development of psychological disorders (Hariri, 2009). Several studies report beneficial effects of antidepressants on emotion task performance (Savaskan et al., 2008; Harmer et al., 2009; Tranter et al., 2009). For example, the effects of experimental manipulations of serotonin and dopamine levels on cognition have been investigated in recovered patients (Hayward et al., 2005; McTavish et al., 2005; Merens, Booij and Van Der Does, 2008). Similar manipulations have also been employed with healthy adults (Harmer et al., 2003; Arce et al., 2008; Paulus et al., 2005), and those with family history of affective illness (Van Der Veen et al., 2007). Stress paradigms have also been used in conjunction with manipulations of neurotransmitter levels (Firk and Markus, 2008), and

such an approach could be pertinent in the context of insomnia as a disorder of hyperarousal (Riemann et al., 2010; Bonnet and Arand, 2010). These studies could also perhaps help to aid in the treatment of insomnia.

Alternative tasks of social functioning

Social competence includes face recognition, emotion recognition, and theory of mind, in coordination with contextual and environmental factors (Iarocci, Yager, and Elfers, 2007). Explicit mentalizing is thought to be linked to the ability to reflect upon mental states, in the self (introspection) and others (Frith and Frith, 2012). Such processes are relevant to social emotions and decision making (Singer, 2012). While a number of tasks of decision making have been investigated with sleep deprived participants, to date few have been applied to poor sleeper groups. Such studies are also necessary in order to address the complexity of social interactions, and how sleep could contribute to this.

Performance in Insomnia

While insomnia is defined on the basis of subjective reports of poor sleep quality, which occurs despite adequate sleep opportunity (Edinger et al., 2004), and associated daytime effects (e.g. Ustinov et al., 2010), finding objective differences in task performance has been difficult for researchers (e.g. Orff et al., 2007; Varkevisser et al., 2007). This has led to the suggestion that the reported performance deficits in insomnia could be more linked to the perception of compensatory 'effort' in maintaining performance, than actual performance impairments as such (Orff et al., 2007; Varkevisser et al., 2007). Others have suggested that performance deficits are subtle (Shekleton, Rogers and Rajaratnam, 2010; Fortier-Brochu et al., 2012), and that sensitive tasks have yet to be found (Espie and Kyle, 2008). In keeping with this, a recent meta-analysis suggests that people with insomnia show effects on memory tasks in particular (Fortier-Brochu et al., 2012). Results from the current studies suggest that emotion recognition and theory of mind is affected in insomnia disorder, indicating that experimental tasks drawn from other areas of psychology could be used to identify performance impairments.

Conclusions

Results suggest that sleep affects emotion recognition in both normal sleepers and people with insomnia. In normal sleepers emotion recognition and sensitivity towards temporal parameters are affected by time since waking, although time since waking and the nature of stimuli seem to affect the direction of results. The previous nights's sleep and depression scores also affect happiness recognition in the general (student) population. These results suggest subtle effects of sleep on socio-emotional functioning, and could have implications for the development of psychiatric disorders such as depression. Emotion recognition is also impaired in insomnia disorder, with specific effects towards high intensity expressions of sadness and low-intensity expressions of surprise. Insomnia also affects reaction times in theory of mind tasks. Results are consistent with previous reports of social functioning impairments in primary insomnia (Kyle, Espie, and Morgan, 2010), as well as results of the systematic review, suggesting that socio-emotional functioning is impaired in insomnia disorder. Future studies should extend these results by using neuroimaging measures. These results are interpreted within the context of the etiology of insomnia, with reference to theories as to role of arousal and hyperarousal.

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Appendix One: Measures and Stimuli

The Insomnia Severity Index (ISI) assesses the severity of insomnia symptoms over the past week, with 5 questions which measure sleep disturbances and daytime effects (Bastien et al., 2001). A total score below 8 indicates no significant insomnia, scores 8 - 14 suggest sub-threshold insomnia, and scores 15 - 28 indicates clinical insomnia. In insomnia patients, this measure correlates with assessments of sleep continuity, with 3 factors identified- daytime effects, sleep disruption severity, and sleep satisfaction (Bastien et al., 2001). The reliability of the ISI has been assessed by Bastien et al., (2001), who found evidence of contenst validity, sensitivity, with concurrent validity and internal consistency.

The Morningness-Eveningness Questionnaire (MEQ) has 19 questions to assess diurnal preference (Horne and Ostberg, 1976). Scores range from 16 – 86, with lower scores indicating greater eveningness. A four-factor structure, of peak time, morning affect, retiring and rising has been identified (Caci et al., 2009). This measure appears valid, with significant correlations with sleep timing variables (Horne and Ostberg, 1976). A Turkish version of this measure shows high reliability over time, with a test-retest reliability co-efficinet of 0.84 (Punduk, Gur and Ercan, 2005)

Sleep diaries assess sleep patterns in detail (Carney et al., 2012), and participants completed a sleep diary on the day of testing rating their sleep for the previous night (Morin and Espie, 2004). This consisted of 11 questions assessing their sleep, as well as questions relating to subjective sleep quality. This measure has construct validity as assessed by focus groups (Carney et al., 2012). However, the reliability of sleep diaries needs to be investigated further due to the nature of sleep, with its instability violating an assumption of test-re-test reliability (Carney et al., 2012).

The Pittsburgh Sleep Quality Index (PSQI) measures sleep quality over the past month, and scores range from 0 – 21 (Buysse et al., 1989). Nineteen items comprise seven components (subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction). This measure discriminates between those with insomnia and good sleepers with good sensitivity and specificity, at a global score > 5 (Buysse et al., 1989; Backhaus et al., 2002). Due to the high levels of sleep disruption within a student population (Lund et al., 2010), the threshold for poor sleep was set at > 6. This was been found to result in a slightly lower sensitivity of 93.4% and 100% specificity for clinically significant sleep disruption (Backhaus et al., 2002). Among primary insomnia patients, the test-retest reliability is 0.087 (Backhaus et al., 2002).

The Hospital Anxiety and Depression Scale (HADS; Zigmond and Snaith, 1983) has 14 questions to measure anxiety and depression over the past week. Scores range from 0 – 21, with scores below 8 considered normal, scores 8 – 11 subclinical, and scores 12 or over suggesting clinical levels of anxiety or depression. A two-factor structure has typically been supported (Cosco et al., 2012). The scoring of this measure has been found to be reliable by Zigmond and Snaith (1983), who also report high internal consistency. Patient and interview ratings were also correlated.

The Interpersonal Reactivity Index (IRI; Davis, 1980) measures cognitive and affective empathy with two subscales for each (i.e., perspective-taking, fantasy scale, empathetic concern, personal distress). Twenty-eight items comprise this measure, and the four components are scored independently. This multidimensional approach was validated by factor loadings and a factor analysis suggesting the validity of this approach. These subscales were also found to be internally reliable, with temporal stability over time.

A 4D stimuli platform was used to generate dynamic stimuli, and is described in detail by Yu, Garrod and Schyns, (2012). Four certified Facial Action Coding System (FACS) coders were used to model 41 core Action Units (AUs). These movements were then translated into functions, which could be used to reproduce each movement. From these functions, which represent each action unit, six temporal parameters were derived. These parameters correspond to peak amplitude, peak latency, onset latency, offset latency, acceleration, and deceleration. These values formed the basis of each facial animation. However, these animations were organized so as to only present physiologically possible facial expressions. Furthermore, additional constraints limited the range of the degrees of freedom, and parameters were not independent (see Yu, Garrod, and Schyns, 2012). Parametric values were therefore selected for each AU pseudo-randomly, creating 1200 facial animations, which were shown on eight individual facial identities (four male and four female).

Appendix 2: Effect sizes

Chapter 3: Effect sizes of emotion recognition performance and empathy

	NS		PS		Effect sizes
	Mean	Std Dev	Mean	Std Dev	Cohen's D
ANGER ACC	76.19	18.21	72.22	13.07	-0.27
FEAR ACC	66.79	16.18	64.48	21.47	-0.18
SAD ACC	75.48	13.24	74.21	17.21	-0.12
DISGUST ACC	67.14	18.55	67.46	19.70	0.02
SURPRISE ACC	82.26	10.85	81.55	11.38	-0.08
HAPPY ACC	98.57	2.66	98.02	3.12	-0.26
ANGER RT	2259.19	329.46	2210.33	356.18	-0.19
FEAR RT	2712.13	666.98	2551.36	468.74	-0.30
SAD RT	2271.36	320.48	2223.07	375.63	-0.19
DISGUST RT	2471.93	804.97	2194.45	461.53	-0.43
SURPRISE RT	2363.17	365.17	2265.38	356.79	-0.34
HAPPY RT	1866.20	220.53	1772.29	158.75	-0.54
IRI PT	18.63	3.80	16.71	5.19	-0.64
IRI FS	16.89	4.90	16.57	5.17	-0.08
IRI EC	20.00	3.77	20.48	4.23	0.16
IRI PD	12.06	5.26	13.05	4.79	0.24

	Early-Late		Effect sizes
	Mean difference	Pooled Std Dev	Cohen's D
HAPPY INTENSITY 1	-0.14071	0.46639	-0.30
SURPRISE INTENSITY 1	-0.01071	0.36613	-0.03
FEAR INTENSITY 1	0	0	N/A
DISGUST INTENSITY 1	-0.18124	0.35034	-0.52
ANGER INTENSITY 1	-0.23571	0.43571	-0.54
SAD INTENSITY 1	0.066087	0.58194	0.11
HAPPY INTENSITY 2	-0.28889	0.67905	-0.43
SURPRISE INTENSITY 2	-0.00388	0.7138	-0.01
FEAR INTENSITY 2	-0.10121	0.29614	-0.34
DISGUST INTENSITY 2	-0.03737	0.55663	-0.07
ANGER INTENSITY 2	-0.46296	0.72591	-0.64
SAD INTENSITY 2	-0.31868	0.56376	-0.57
HAPPY INTENSITY 3	0.12698	0.66479	0.19
SURPRISE INTENSITY 3	-0.01339	0.73103	-0.02
FEAR INTENSITY 3	-0.02143	0.60718	-0.04
DISGUST INTENSITY 3	-0.31798	0.75509	-0.42
ANGER INTENSITY 3	-0.42308	0.78174	-0.54
SAD INTENSITY 3	-0.38995	0.78353	-0.50
HAPPY INTENSITY 4	0.09899	1.0193	0.10
SURPRISE INTENSITY 4	0.001463	1.0031	0.00
FEAR INTENSITY 4	-0.21511	1.0579	-0.20
DISGUST INTENSITY 4	-0.05584	0.90424	-0.06
ANGER INTENSITY 4	-0.04448	0.96602	-0.05
SAD INTENSITY 4	-0.40502	0.99065	-0.41
HAPPY INTENSITY 5	-0.05356	1.0768	-0.05
SURPRISE INTENSITY 5	0.00149	1.0024	0.00
FEAR INTENSITY 5	-0.23894	1.0858	-0.22
DISGUST INTENSITY 5	0.1616	0.98463	0.16
ANGER INTENSITY 5	0.018356	0.93537	0.02
SAD INTENSITY 5	-0.28999	0.98452	-0.29

	3h Group		12h Group		
	Mean	Std Dev	Mean	Std Dev	Cohen's D
Anger face	14.77273	12.48592	15.00000	10.95445	-0.02
Fear face	12.04545	12.01775	9.52381	7.05421	0.25
Happy Face	2.04545	3.67070	0.23810	1.09109	0.66
Sad face	10.22727	8.79357	11.19048	9.98809	-0.10
Anger IAPS	12.72727	9.84732	20.00000	16.04681	-0.55
Fear IAPS	17.27273	11.72373	20.47619	12.83596	-0.26
Happy IAPS	0.90909	1.97386	0.95238	2.55883	-0.02
Sad IAPS	8.40909	8.22111	7.14286	9.81981	0.14
Anger face RT	2060.38636	410.63890	2187.69048	362.94061	-0.33
Fear face RT	2152.36364	369.33312	2249.92857	356.04425	-0.27
Happy face RT	1601.40909	92.03967	1677.78571	166.43111	-0.57
Sad face RT	1936.59091	206.66920	2146.07143	423.96890	-0.63
Anger IAPS RT	2418.72727	489.22226	2475.09524	647.07756	-0.10
Fear IAPS RT	2798.61364	714.56707	2943.45238	869.80947	-0.18
Happy IAPS RT	1931.34091	295.83632	1930.59524	276.85152	0.00
Sad IAPS RT	2158.84091	360.72923	2274.52381	325.57835	-0.34
MFFT ER	11.59091	17.95407	20.95238	24.57738	-0.44
MFFT RT	5291.22727	9428.51306	9423.33333	10130.85082	-0.42

	3h Group		9h Group		
	Mean	Std Dev	Mean	Std Dev	Cohen's D
Anger face	14.77273	12.48592	14.50000	10.87005	0.02
Fear face	12.04545	12.01775	7.50000	7.86398	0.44
Happy Face	2.04545	3.67070	0.25000	1.11803	0.65
Sad face	10.22727	8.79357	8.75000	8.56477	0.17
Anger IAPS	12.72727	9.84732	23.50000	20.39737	-0.68
Fear IAPS	17.27273	11.72373	15.25000	11.17740	0.18
Happy IAPS	0.90909	1.97386	1.25000	2.22131	-0.16
Sad IAPS	8.40909	8.22111	4.25000	5.91052	0.58
Anger face RT	2060.38636	410.63890	1974.05000	337.75645	0.23
Fear face RT	2152.36364	369.33312	2154.42500	386.21685	-0.01
Happy face RT	1601.40909	92.03967	1608.20000	142.24778	-0.06
Sad face RT	1936.59091	206.66920	1979.65000	306.61681	-0.17
Anger IAPS RT	2418.72727	489.22226	2349.67500	474.30798	0.14
Fear IAPS RT	2798.61364	714.56707	2585.07500	496.69646	0.34
Happy IAPS RT	1931.34091	295.83632	1844.25000	173.99172	0.35
Sad IAPS RT	2158.84091	360.72923	2134.45000	288.15639	0.07
MFFT ER	11.59091	17.95407	11.00000	20.30038	0.03
MFFT RT	5291.22727	9428.51306	4890.22500	8624.53011	0.04

	3h Group		6h Group		
	Mean	Std Dev	Mean	Std Dev	Cohen's D
Anger face	14.77273	12.48592	20.62500	13.04778	-0.46
Fear face	12.04545	12.01775	12.50000	10.83473	-0.04
Happy Face	2.04545	3.67070	1.25000	3.68605	0.22
Sad face	10.22727	8.79357	8.54167	9.26414	0.19
Anger IAPS	12.72727	9.84732	21.04167	18.35514	-0.56
Fear IAPS	17.27273	11.72373	24.58333	15.73674	-0.52
Happy IAPS	0.90909	1.97386	0.62500	2.24214	0.13
Sad IAPS	8.40909	8.22111	3.33333	4.34057	0.78
Anger face RT	2060.38636	410.63890	2122.33333	453.95675	-0.14
Fear face RT	2152.36364	369.33312	2315.56250	542.31227	-0.35
Happy face RT	1601.40909	92.03967	1661.25000	220.97895	-0.35
Sad face RT	1936.59091	206.66920	2117.22917	473.48103	-0.49
Anger IAPS RT	2418.72727	489.22226	2414.16667	597.20222	0.01
Fear IAPS RT	2798.61364	714.56707	2837.22917	701.37192	-0.05
Happy IAPS RT	1931.34091	295.83632	1954.35417	329.23774	-0.07
Sad IAPS RT	2158.84091	360.72923	2186.75000	455.10042	-0.07
MFFT ER	11.59091	17.95407	12.08333	16.14585	-0.03
MFFT RT	5291.22727	9428.51306	7287.75000	9312.13595	-0.21

	6h Group		9h Group		
	Mean	Std Dev	Mean	Std Dev	Cohen's D
Anger face	20.62500	13.04778	14.50000	10.87005	0.78
Fear face	12.50000	10.83473	7.50000	7.86398	1.72
Happy Face	1.25000	3.68605	0.25000	1.11803	8.39
Sad face	8.54167	9.26414	8.75000	8.56477	1.70
Anger IAPS	21.04167	18.35514	23.50000	20.39737	0.03
Fear IAPS	24.58333	15.73674	15.25000	11.17740	0.63
Happy IAPS	0.62500	2.24214	1.25000	2.22131	10.19
Sad IAPS	3.33333	4.34057	4.25000	5.91052	3.86
Anger face RT	2122.33333	453.95675	1974.05000	337.75645	-4.81
Fear face RT	2315.56250	542.31227	2154.42500	386.21685	-4.46
Happy face RT	1661.25000	220.97895	1608.20000	142.24778	-8.36
Sad face RT	2117.22917	473.48103	1979.65000	306.61681	-4.81
Anger IAPS RT	2414.16667	597.20222	2349.67500	474.30798	-4.27
Fear IAPS RT	2837.22917	701.37192	2585.07500	496.69646	-4.15
Happy IAPS RT	1954.35417	329.23774	1844.25000	173.99172	-6.73
Sad IAPS RT	2186.75000	455.10042	2134.45000	288.15639	-5.43
MFFT ER	12.08333	16.14585	11.00000	20.30038	0.72
MFFT RT	7287.75000	9312.13595	4890.22500	8624.53011	-0.54

	6h Group		12h Group		
	Mean	Std Dev	Mean	Std Dev	Cohen's D
Anger face	20.62500	13.04778	15.00000	10.95445	0.46
Fear face	12.50000	10.83473	9.52381	7.05421	0.32
Happy Face	1.25000	3.68605	0.23810	1.09109	0.36
Sad face	8.54167	9.26414	11.19048	9.98809	-0.28
Anger IAPS	21.04167	18.35514	20.00000	16.04681	0.06
Fear IAPS	24.58333	15.73674	20.47619	12.83596	0.28
Happy IAPS	0.62500	2.24214	0.95238	2.55883	-0.14
Sad IAPS	3.33333	4.34057	7.14286	9.81981	-0.51
Anger face RT	2122.33333	453.95675	2187.69048	362.94061	-0.16
Fear face RT	2315.56250	542.31227	2249.92857	356.04425	0.14
Happy face RT	1661.25000	220.97895	1677.78571	166.43111	-0.08
Sad face RT	2117.22917	473.48103	2146.07143	423.96890	-0.06
Anger IAPS RT	2414.16667	597.20222	2475.09524	647.07756	-0.10
Fear IAPS RT	2837.22917	701.37192	2943.45238	869.80947	-0.14
Happy IAPS RT	1954.35417	329.23774	1930.59524	276.85152	0.08
Sad IAPS RT	2186.75000	455.10042	2274.52381	325.57835	-0.22
MFFT ER	12.08333	16.14585	20.95238	24.57738	-0.43
MFFT RT	7287.75000	9312.13595	9423.33333	10130.85082	-0.22

	9h Group		12h Group		
	Mean	Std Dev	Mean	Std Dev	Cohen's D
Anger face	14.50000	10.87005	15.00000	10.95445	-0.05
Fear face	7.50000	7.86398	9.52381	7.05421	-0.27
Happy Face	0.25000	1.11803	0.23810	1.09109	0.01
Sad face	8.75000	8.56477	11.19048	9.98809	-0.26
Anger IAPS	23.50000	20.39737	20.00000	16.04681	0.19
Fear IAPS	15.25000	11.17740	20.47619	12.83596	-0.43
Happy IAPS	1.25000	2.22131	0.95238	2.55883	0.12
Sad IAPS	4.25000	5.91052	7.14286	9.81981	-0.35
Anger face RT	1974.05000	337.75645	2187.69048	362.94061	-0.61
Fear face RT	2154.42500	386.21685	2249.92857	356.04425	-0.26
Happy face RT	1608.20000	142.24778	1677.78571	166.43111	-0.45
Sad face RT	1979.65000	306.61681	2146.07143	423.96890	-0.45
Anger IAPS RT	2349.67500	474.30798	2475.09524	647.07756	-0.22
Fear IAPS RT	2585.07500	496.69646	2943.45238	869.80947	-0.50
Happy IAPS RT	1844.25000	173.99172	1930.59524	276.85152	-0.37
Sad IAPS RT	2134.45000	288.15639	2274.52381	325.57835	-0.45
MFFT ER	11.00000	20.30038	20.95238	24.57738	-0.44
MFFT RT	4890.22500	8624.53011	9423.33333	10130.85082	-0.48

		NS		INS		
		Mean	Std Dev	Mean	Std Dev	Cohen's D
GROUP NS	ME_ACC	.80	.05	.77	.09	0.41
	ME_RT	5325.29	1490.18	4902.74	2159.47	0.23
	FB_ACC	97.02	5.46	97.02	6.74	0.00
	TB_ACC	94.05	10.91	94.05	7.52	0.00
	FB_RT	5181.57	788.98	4599.08	652.78	0.81
	TB_RT	5065.72	895.72	4514.50	651.04	0.71
	PT_ACC_THEY_C	.88	.30	.80	.39	0.23
	PT_ACC_THEY_I	.92	.07	.87	.09	0.62
	PT_ACC_YOU_C	.96	.04	.96	.06	0.00
	PT_ACC_YOU_I	.91	.10	.95	.06	-0.49
	PT_RT_THEY_C	759.83	172.14	699.39	132.31	0.39
	PT_RT_THEY_I	882.25	191.56	846.71	140.24	0.21
	PT_RT_YOU_C	782.12	179.71	734.37	143.13	0.29
	PT_RT_YOU_I	861.49	198.44	778.83	176.25	0.44