

Investigating the Relationship between Sleep and Postpartum Depression

**A longitudinal study examining the relationships between subjective and
objective sleep during the perinatal period and postpartum depression**

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A thesis submitted in partial fulfilment of the requirements of
Bournemouth University for the degree of Doctor of Philosophy

October 2013

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Lauren Kita

Research has suggested that a bi-directional relationship exists between sleep disruption and depression. Not only is poor sleep a commonly reported symptom in those with depression, some aspects of sleep have also been shown to predict the onset of depression. Despite sleep problems being a commonly reported occurrence throughout the perinatal period, the field of perinatal sleep research remains in its relative infancy. However, recent studies suggest that sleep disturbances during this time may increase the risk of developing postpartum depression. Currently, research in this area is limited by studies that have failed to control for depressive symptoms at baseline, relied upon subjective, often retrospective, measures of sleep, and have only measured symptoms of postpartum depression in the early postpartum period. Few studies have used polysomnography, considered the 'gold standard' of sleep, and no studies to date have specifically compared the relationship between subjective and objective sleep. Therefore, the major aim of this thesis was to gain a better understanding of the specific aspects of sleep that were most relevant to postpartum depression. In order to address this aim, studies were carried out to: explore the aspects of sleep most relevant to major depressive disorder; examine differences in sleep between pregnant and non-pregnant women; investigate the relationships between subjective and objective measures of sleep; explore longitudinal changes in sleep, fatigue and depression throughout the perinatal period, and finally; examine which aspects of sleep at which time-point were most relevant to the development of postpartum depression. Overall this thesis found that women experience significant changes to their sleep throughout the perinatal period. While the sleep of third trimester women is considerably poorer than that of non-pregnant women (both objectively and subjectively), the most significant changes occur in the transition between late pregnancy and the early postpartum period. Furthermore, increased amounts of sleep and reports of difficulty falling asleep during late pregnancy predicted the development of postpartum depressive symptoms. This suggests that certain aspects of sleep during late pregnancy may serve as markers for women at risk of developing postpartum depression.

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Acknowledgments

First and foremost I would like to thank my supervisors, Dr Andrew Mayers and Professor Siné McDougall, for their continued support and encouragement. Thank you both for picking me up during my moments of melt-down, and for maintaining your faith in me throughout the past three years.

Many thanks to those that have helped me along the way, particularly in learning polysomnography. Thank you to Lyn Davies who sadly passed away this year. Lyn was always a phone's reach away whenever I had any technical problems (of which there were quite a few!). I would like to thank Julia Chapman and Sean Higgins for allowing me to spend invaluable time at the Sleep Disorders Centre at Guys' and St Thomas' Hospital in London. I am also grateful to Claudia Maldonado for her early sleep mentorship, and to Liz Griffiths for helping me to navigate my way through the long-winded NHS ethical approval process!

Thank you to all of the women that kindly volunteered to take part in my (somewhat demanding!) study, without you this would not have been possible. I am especially grateful to Rhiannon Barrington for her assistance with data collection, and for keeping me cheery during the many evenings that we spent together setting up sleep studies.

I would like to thank Bournemouth University for enabling me to do this PhD and for providing a supportive community. Also thanks to Santander for allowing me the funds to attend my first international sleep conference in America, which truly enlightened my passion for sleep research, a field in which I hope to devote the rest of my career.

Finally, I would finally like to thank my family. Particularly my boyfriend, Dudley, for putting up with me during the past three years and for being such a positive person. Thank you to my mother and father for believing in me and for investing in my education, which has allowed me to get where I am today. I promise to get a 'real job' some day!

Introduction and Overview

We spend around a third of our lives asleep (Wilson & Nutt, 2008). While the specific function of sleep is debated, lack of sleep is associated with a range of negative outcomes. This includes changes in mood, cognitive impairment and disruption to bodily systems (Durmer & Dinges, 2005; McEwen, 2006). There are two main types of sleep: rapid eye movement sleep (REM) and non-rapid eye movement sleep (NREM; Aserinsky & Kleitman, 1953; Rechtschaffen & Kales, 1968), which appear to have different functions. NREM is further divided into four stages with each stage representing a deeper form of sleep. The different stages of sleep have a characteristic form, including specific brain waves, eye movements and muscle tone.

The stages of sleep can be measured objectively using polysomnography (PSG). This permits the examination of both 'normal' and 'abnormal' sleep. Although there is no single definition of how much sleep an individual should obtain, healthy sleep includes steady transitions through each of the stages (Rechtschaffen & Kales, 1968). In contrast, atypical sleep patterns may include changes to the phasing of sleep stages, more arousals from sleep, changes to the duration of sleep, and can result in excessive daytime sleepiness. These features may indicate the presence of a sleep disorder. Sleep can also be measured subjectively. However, the way that sleep is perceived may not necessarily reflect actual objective sleep, and may be affected by other factors such as depression (Argyropoulos et al., 2003; Rotenberg, Indursky, Kayumov, Sirota, & Melamed, 2000; Unruh et al., 2008).

The aim of this thesis is to examine the relationship between sleep and postpartum depression. However, before reviewing this relationship it is important to first gain an understanding of the importance and function of sleep, as well as the different ways that it can be measured. Chapter 1 therefore provides an overview of sleep, including a description of 'normal' sleep and the ways in which sleep can be measured, with a specific focus on differences between subjective and objective sleep. The second part of this chapter focuses on sleep changes throughout the perinatal period. Chapter 2 examines both major depressive disorder and postpartum depression and describes research that has examined the relationship between sleep and depression, along with possible theoretical underpinnings. Chapter 3 provides a systematic literature review of studies which have specifically examined the relationship between sleep and postpartum depression, leading onto the aims and

objective of this thesis. Chapter 4 details a preliminary study that was carried out in order to delineate which aspects of subjective sleep were most relevant to depression in a general sample, before specifically focusing on the perinatal period. Chapter 5 describes the methods for the main study: investigating the relationship between sleep and postpartum depression. This includes a justification of the rationale for the study design and the measures adopted, a description of the procedure, and information regarding participant characteristics.

Given that this was a large study with several different components, the results of the study are described in three parts over Chapters 6, 7 and 8. Chapter 6 describes differences in sleep architecture between pregnant and non-pregnant women, as well as the relationships between subjective and objective sleep. Chapter 7 presents the longitudinal findings examining changes in sleep, fatigue and depression throughout the perinatal period. Chapter 8 examines the cross-sectional and longitudinal relationships between sleep and perinatal depression, followed by a series of regression analyses that identified which aspects of sleep were most predictive of postpartum depression scores. Finally, Chapter 9 integrates the findings from these chapters and ends with a discussion of their implications, both in relation to clinical practice and future research.

CHAPTER 1: SLEEP

"If sleep does not serve an absolutely vital function, then it is the biggest mistake the evolutionary process has ever made."

Allan Rechtschaffen, University of Chicago Sleep Laboratory
Smithsonian, November 1978

1.1 Overview of Chapter

This chapter provides an overview of sleep, the ways in which sleep can be measured, and the relationship between subjective and objective measures of sleep. Given what we know about the complexity of sleep, a range of measures should therefore be used for its measurement within research. Throughout this thesis it is argued that many studies exploring the relationship between sleep and postpartum depression overlook this complexity, and therefore our current understanding of this relationship is limited.

The second part of the chapter focuses on how sleep patterns can change during the perinatal period. Women experience significant changes to their sleep patterns during both pregnancy and the postpartum period, as a result of a number of physical, psychological and hormonal factors. However, research within this area is limited by few studies, limited measures of sleep, small sample sizes and inconsistent methodologies. Chapter 3 will review the literature of studies that have examined how poor sleep during pregnancy may be related to the development of PPD. Following this will be a description of studies that set out to expand our understanding of this relationship.

1.2 Sleep Physiology

History of Electroencephalography (EEG)

The studying of brain waves was first discovered by Berger (1929), through the invention of EEG. Berger (1929) discovered that the brain produces different waves that could be used to study individual differences in brain activity. The process of EEG involves a series of electrodes being positioned on the scalp, which monitor levels of neuronal activity in the cerebral cortex. These electrodes can be positioned in accordance with the international

10-20 electrode placement system, using pre-defined measurements (Klem, Lüders, Jasper, & Elger, 1999). Initially, this activity was recorded on paper but nowadays is displayed on a computer screen. Brain activity can be classified into four different types of activity, defined by amplitude (the measurement of the top or bottom half of the wave) in microvolts (mV) and frequency (the number of waves produced each second) in hertz (Hz; Cooper, 1994):

- 1 Alpha rhythm: typically occurs during states of resting or quiet wakefulness with eyes closed. The frequency is between 8 and 14 Hz, amplitude is less than 50 mV.
- 2 Beta rhythm: occurs in several stages of sleep mainly in anterior brain regions. The frequency is more than 13 Hz and amplitude is less than 30 mV.
- 3 Theta rhythm: appears during brain activation. The frequency is 4-8 Hz with low amplitude.
- 4 Delta rhythm: observed in all brain regions during slow-wave sleep (SWS). The frequency is 4 Hz or less with wide amplitude.

Polysomnography (PSG)

The five stages of sleep can be analysed in detail using polysomnography (PSG). This involves the use of three measures that are combined to enable accurate identification of each stage (Rechtschaffen & Kales, 1968). The five stages of sleep are each characterised by a particular type of wave dominance (Loomis, Harvey, & Hobart, 1937). Rapid eye movement sleep (REM) was first identified by Aserinsky and Kleitman (1953) and this stage was later associated with dreaming (Dement & Kleitman, 1957). The latest PSG manual from the American Academy of Sleep Medicine (AASM) offers specific guidance around both the placement of electrodes and scoring of sleep (Iber, Ancoli-Israel, Chesson, & Quan, 2007). The three measures used in PSG are described below.

- 1 Electroencephalography (EEG): As described, this involves a series of electrodes being placed on the scalp to measure electrical activity in the brain. Electrodes are placed in occipital, central and frontal locations, as well as a reference electrode on each mastoid (behind the ear) and two ground electrodes. Careful measurements are first carried out according to the 10-20 method to ensure consistent and accurate recordings

(Klem et al., 1999).

- 2 Electrooculography (EOG): This is used to identify eye movements seen in phasic REM sleep. Electrodes are placed next to each eye to record movement.
- 3 Electromyography (EMG): This is used to detect muscle movement, particularly for the identification of muscle atonia during REM sleep. The latest AASM recommendations suggest that three electrodes are placed on the chin (Iber et al., 2007).

1.2.1 Stages of Sleep

Collectively, EEG, EOG and EMG activity are used to identify stages of sleep. Polysomnographic sleep is typically scored by assigning a stage to each 30 second epoch of the night. Sleep stages go from zero to four; with zero indicating a period of wakefulness. These stages are followed by a period of REM sleep. Stages one to four are known as non-REM sleep (NREM), each increasing in depth. Each stage of sleep is dominated by a particular type of wave, as shown in Figure 1.

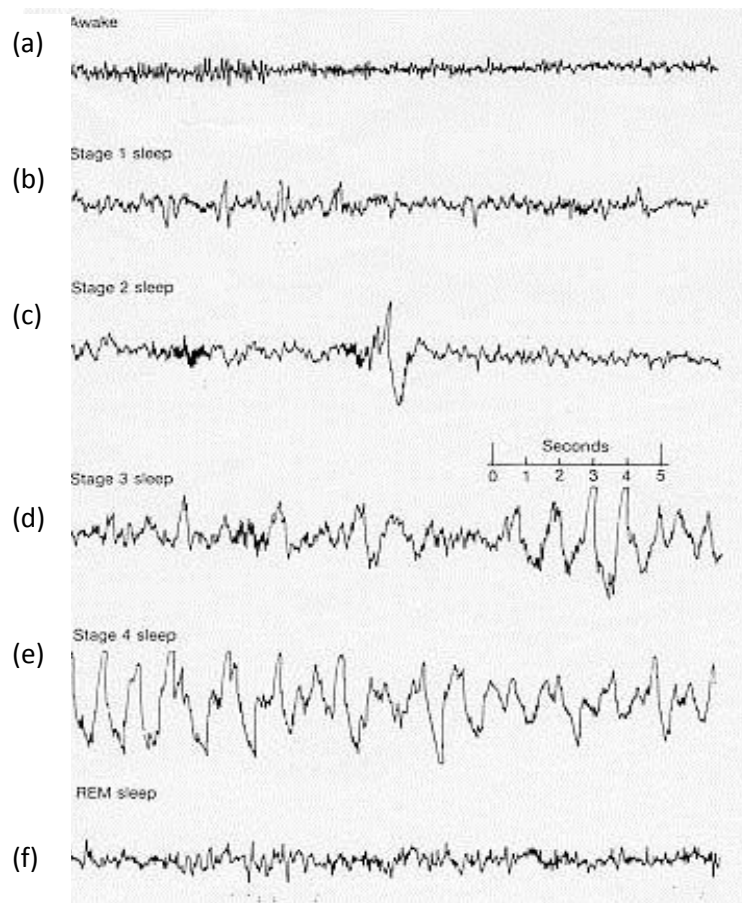


Figure 1. Brain waves during the different stages of sleep (from www.sleepdex.org)

The stages of sleep are described below (Iber et al., 2007):

- Stage 0 (wakefulness; see Figure 1a)
 - EEG: Low-voltage, mixed frequency of activity, with periods of quiet wakefulness associated with alpha wave activity, particularly when a person is drowsy and eyes are closed.
 - EOG: Control of eye movements is voluntary; blinks and deflections present. Slow rolling eye movements when a person is drowsy with eyes closed.
 - EMG: Varies according to level of activity and muscle relaxation, with variable amplitude tonic activity.

- Stage 1 (transition from wakefulness to sleep; see Figure 1b)
 - EEG: Gradually slows down, alpha waves disperse and are substituted with theta activity. Negative short waves that are isolated or come in bursts appear, recorded from Cz and nearby, known as vertex sharp waves.
 - EOG: Slow, rolling eye movements.
 - EMG: Slightly reduced from wakefulness, low-voltage tonic activity. May be accompanied by abrupt muscular contractions of the extremities known as myoclonic hypnagogic jerks, which may wake the patient and may be accompanied by hallucinations of falling.
 - Accounts for around 2-5% sleep in healthy adults (Lee-Chiong, 2008).
- Stage 2 (see Figure 1c)
 - EEG: Presence of spindles (rhythmic waves between 12-14 Hz and 20-30 mV) and K- complexes (rapid negative wave followed by positive wave which is slower and higher in amplitude).
 - EOG: Some slow eye movements may be observed but gradually disappear.
 - EMG: Body movements rare, tonic activity reduced.
 - Accounts for around 45-55% sleep in healthy adults (Lee-Chiong, 2008).
- Stage 3* (see Figure 1d)
 - EEG: Slow-wave (delta) activity increases to approximately 20-50% of each epoch until a synchronized pattern appears. Spindles and K-complexes may still be visible at this stage.
 - EOG: No eye movements or activity.

- EMG: Similar, or slightly less than stages 1 and 2.
- Accounts for around 10% sleep in healthy adults (Lee-Chiong, 2008).
- Stage 4* (see Figure 1e)
 - EEG: More than 50% of each epoch is slow-wave delta activity.
 - EOG: No eye movements or activity.
 - EMG: Very low-amplitude tonic activity may occur.
 - Accounts for around 10% sleep in healthy adults (Lee-Chiong, 2008).

* Stages 3 and 4 are also known as 'slow wave sleep' (SWS). According to the AASM Manual for the Scoring of Sleep and Associated Events, stages 3 and 4 are now combined (Iber et al., 2007); after concluding that there was no biological significance or validity in the subdivision of SWS (Silber et al., 2007). This has replaced the former system of Rechtschaffen and Kales (1968). This international scoring system is used for the analysis of sleep within this thesis.

- REM sleep (see figure 1f)
 - EEG: Small amplitude, mixed frequency which resemble stage 1. May have 'sawtooth' waves, frequency in the theta range.
 - EOG: Rapid eye movements in opposing directions.
 - EMG: Reduced to lowest level of recording; suppression of muscle tone apart from occasional twitches in the face and extremities.
 - Accounts for around 20-25% sleep in healthy adults (Lee-Chiong, 2008).

1.2.2 Normal Sleep

Sleep is a dynamic process. Throughout the night we go through cycles of NREM and REM sleep. Healthy individuals enter sleep through NREM sleep (stages 1, 2, 3 & 4) followed by a period of REM sleep. This transition through the sleep stages is known as a sleep cycle. Each

cycle lasts between 90-120 minutes, and most healthy adults experience between 3-5 cycles during a night's sleep (Lee-Chiong, 2008). However, percentage of slow-wave sleep decreases with age (Wilson & Nutt, 2008). Each cycle does not necessarily contain all stages of sleep. The first part of the night is dominated by stages 3 and 4 (slow-wave sleep), and sleep becomes gradually lighter with more REM sleep as the night progresses. Figure 2 shows a simplified hypnogram of a healthy individual's sleep. This individual fell asleep around 22.30pm and woke up at approximately 7am. All of their slow-wave sleep occurred before 2am and periods of REM sleep became longer throughout the night. This individual experienced five sleep cycles over the course of the night.

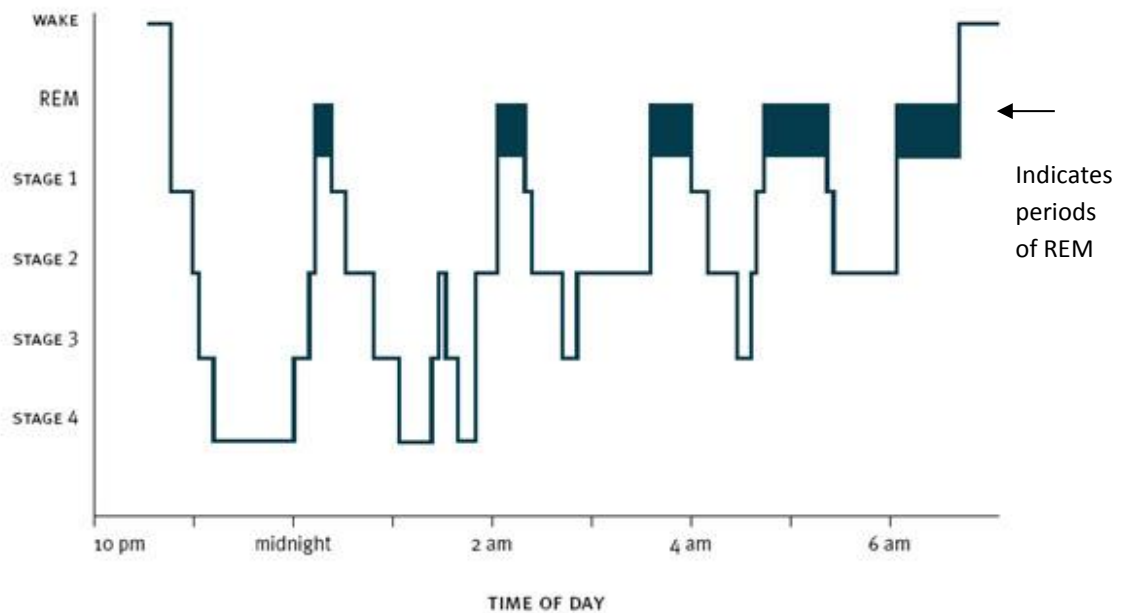


Figure 2. Simplified hypnogram of a healthy individual's sleep (from Gander, 2003)

1.2.3 Sleep-Wake Regulation: Circadian Rhythms

According to Borbély (1982), there are two processes that control our sleep-wake cycle: the circadian process (known as Process 'C') and the homeostatic process (known as Process 'S'). Circadian processes are regular rhythms of light and dark cycles that regulate our lives, and play an important role in sleep (Colten & Altevogt, 2006). They typically work around approximately a 24 hour period and affect many bodily functions, including regular changes in core body temperature, hormonal secretions, heart rate, renal output, gut motility and

melatonin secretion (Cooper, 1994). The circadian rhythm system in humans and mammals is controlled by the suprachiasmatic nucleus (SCN), located within the anterior hypothalamus (Wilson & Nutt, 2008). Circadian rhythms are present in every cell and organ in the body. These are known as peripheral clocks (Hastings, Maywood, & Reddy, 2008).

The physiological processes within the body adapt to these night and day rhythms. Melatonin, known as the darkness hormone, induces sleepiness and increases in dim light (Colten & Altevogt, 2006). Core body temperature reaches its lowest point during sleep (Colten & Altevogt, 2006). Sleep is usually initiated during the period of increased melatonin and decreased body temperature (Dijk, Duffy, Riel, Shanahan, & Czeisler, 1999). In this way, the circadian system can be thought of as a finely tuned system, allowing us to function most efficiently in our environment.

However, when left to 'free-run,' (e.g. if light and dark and other environmental cues are removed) our daily rhythms tend to conform to slightly longer than 24 hours (Sack, Brandes, Kendall, & Lewy, 2000). This means that our system continually has to be reset to the 24-hour cycle. Zeitgebers are processes that entrain the circadian system. One of the most important of these is light, which falls on the retina and send impulses to the SCN (Wirz-Justice, Benedetti, & Terman, 2009). Light is also a key factor in the production of melatonin, having a suppressive effect. This is the mechanism of bright light therapy; used to treat circadian rhythm sleep disorders and seasonal affective disorder (Dawson & Encel, 1993). Other zeitgebers include social patterns, such as regular social activities including work, sleep and meal times, all of which affect the circadian system (Wirz-Justice et al., 2009).

When circadian rhythms are disrupted, for example due to jet lag or shift work, this is associated with negative outcomes such as decreased cognitive functioning and adverse health outcomes (Zee & Goldstein, 2010). When an individual attempts to sleep during an inappropriate time of the circadian cycle, such as when melatonin is declining and body temperature is rising, the resulting sleep, if any, is likely to be shorter with more awakenings (Dijk et al., 1999).

The second process involved in sleep-wake regulation is the homeostatic process (process 'S,' Borbély, 1982). While circadian processes control the timing of sleep based on 24-hour biological, psychological and physiological cycles; homeostatic processes determine the

propensity for sleep, based on amounts of prior wakefulness. Slow-wave sleep is a measure of homeostatic pressure, and is increased after periods of sleep deprivation (Kubitz, Landers, Petruzzello, & Han, 1996). Process 'S' reaches its highest point around 16 hours after waking, and is lowest upon natural waking (Wilson & Nutt, 2008). Optimal sleep quality will occur when sleep onset takes place at the optimal circadian phase (night-time) and once enough wake-time has passed so that sleep propensity (homeostatic pressure) reaches a sufficient threshold to allow for rapid sleep onset. While this thesis does not examine specific biological markers of the circadian process, given that sleep in itself is a circadian process, it is important to consider how circadian rhythms may play a role in depression, since the relationship between sleep and depression is the key topic of this thesis.

In recent years there has been a growth of research exploring circadian rhythm disturbances in depression (Boyce & Barriball, 2010; Germain & Kupfer, 2008; Gorwood, 2010; Monteleone, Martiadis, & Maj, 2011; Salgado-Delgado, Tapia Osorio, Saderi, & Escobar, 2011). Current knowledge surrounding the symptomatology and treatment efficacy in depression supports a circadian component. Early morning awakenings, diurnal mood changes, changes in sleep architecture¹ and changes to the timing of temperature nadir and peak cortisol levels, are all commonly reported in depression (Boyce & Barriball, 2010). Each of these processes involves a circadian component. Mood variation throughout the 24-hour cycle in healthy individuals is related to the interaction between duration of prior wakefulness and circadian phase (Boivin et al., 1997). It is therefore not surprising that the circadian and sleep-related disturbances related to depression can have detrimental effects on mood (Germain & Kupfer, 2008).

The phase-shift hypothesis postulates that mood disturbances result from a phase delay or advance of the SCN and related circadian rhythms that control temperature, cortisol, melatonin and REM sleep, relative to other rhythms (Germain & Kupfer, 2008). Reduced REM latency (explored further in Chapter 2), early morning awakenings, and early morning

¹ Sleep architecture refers to the basic structural organisation of sleep, measured using polysomnography. This includes transition between stages (REM and non-REM sleep), proportions of time spent in each stage, cycles of sleep, and timings.

increase in adrenocorticotrophic hormone (ACTH) have been reported in depression, supporting a phase advance hypothesis (Monteleone et al., 2011).

The dim light melatonin onset (DLMO) is a way of measuring a person's circadian phase (Lewy & Sack, 1989), and is considered to be the time at which the biological 'sleep gate' opens (Shochat, Luboshitzky, & Lavie, 1997). DLMO can be measured by taking salivary samples every 30 minutes from 6pm until bedtime. The interval between DLMO and timing of mid-sleep (known as the phase angle difference) can provide a marker of internal circadian alignment (Lewy, 2007). Lewy (2007) reported a phase angle difference of 6 hours in a group of healthy subjects, while depressive severity in those with seasonal affective disorder was associated with significant deviation from this score, in both directions. In contrast, others have found a negative correlation between depression scores and phase angle difference in MDD, supporting the idea of a phase delay (Emens, Lewy, Kinzie, Arntz, & Rough, 2009).

Recent research has examined whether changes to circadian alignment occur throughout the perinatal period (a detailed discussion of perinatal sleep is provided later in the chapter). Given that sleep pattern changes are likely to change patterns of exposure to light and dark, it is also likely that they alter the circadian phase. Previous research has found later rise times in the postpartum period compared to pregnancy, indicative of a phase delay (Wolfson, Crowley, Anwer, & Bassett, 2003). In a preliminary study of 12 women, Sharkey, Pearlstein and Carskadon (2013) found significant changes in circadian phase position and phase angle between DLMO and bedtime between the third trimester of pregnancy and 6 weeks postpartum. Most women experienced phase delays of DLMO in the postpartum period compared to pregnancy, despite bedtime remaining stable between time-points. As a result, the phase angle between DLMO and bedtime also shortened in the postpartum period, indicating that new mothers were falling asleep closer to the endogenous time of melatonin secretion compared to pregnancy. Furthermore, the authors found strong correlations between circadian measures (including DLMO and phase angle) and depressive symptoms during pregnancy and at 2 and 6 weeks postpartum. Later circadian phase and eveningness preference were most consistently associated with increased depression. This suggests that women experience changes in sleep-related processes during the perinatal period, which may contribute to the development of postpartum depression. Gaining a better understanding of

the relationship between sleep and postpartum depression is a key aim of this thesis and is addressed in later chapters.

1.3 Functions of Sleep

The exact function of sleep remains unclear, yet it is evident that sleep is important for many vital functions, such as energy conservation, thermoregulation (control of bodily temperature) and tissue recovery (Maquet, 2001). Animal studies of sleep deprivation have provided key evidence that sleep is vital for survival. A study by Everson, Bergmann, and Rechtschaffen (1989) found that total sleep deprivation in rats resulted in death; with survival time ranging from 11-32 days. Sleep deprivation was also associated with weight loss, scrawny appearance, increased food intake, increased energy expenditure, decreased body temperature and altered blood plasma levels of norepinephrine and thyroxin.

Studies of human sleep deprivation are understandably rare. However, research suggests that the cumulative effect of long-term sleep loss is associated with a number of adverse health outcomes, including increased risk of hypertension (Gottlieb et al., 2006) diabetes and obesity (Knutson, Spiegel, Penev, & Van Cauter, 2007). Research also suggests that sleep plays an important role in memory; particularly the consolidation of memories and their transference into long-term memory (Stickgold, 2005). A large amount of research has shown that sleep aids the offline processing of memories; although REM and NREM sleep may have different roles (Stickgold & Walker, 2007). However, the specific way in which sleep aids memory remains unclear (Maquet, 2001; Stickgold, 2005).

1.3.1 Differing Functions of Sleep

It has been argued that the different stages of sleep provide different functions (Hartmann, 1973). The next section will explore the different functions that have been proposed for REM and NREM sleep. This is important, as depression appears to be associated with changes in the patterns of both REM and NREM sleep, which will be explored in Chapter 2.

Slow-Wave Sleep (SWS - stages 3 & 4)

A common theory is that sleep is necessary for restoration. Evidence suggests that SWS plays an important role in restoration (Neubauer & McHugh, 2003), which is why it is often referred

to as 'deep' sleep. It is also the stage in which it is most difficult to arouse from. Waking from SWS is associated with increased sleep inertia; a transitional state of lowered arousal which occurs immediately upon waking, and produces temporary lowered performance (Tassi & Muzet, 2000). SWS helps to reduce energy consumption and resupply the brain with important resources that become depleted during wakefulness (Neubauer & McHugh, 2003). Therefore, the amount of SWS is related to prior wakefulness and energy expenditure (Youngstedt, O'Connor, & Dishman, 1997). Another restorative quality of SWS is that it is the only stage in which the growth hormone (GH) is released (Sassin et al., 1969). Depletion of GH is associated with less SWS, less overall sleep, greater sleep fragmentation and poorer quality of sleep (Van Cauter et al., 2004).

Another important factor regarding SWS is that an individual's subjective perception of their overall sleep quality may be directly related to amounts of SWS that they receive (Akerstedt, Hume, Minors, & Waterhouse, 1997). Therefore, individuals who receive less SWS may report poorer sleep quality, and those who report poorer sleep quality may be experiencing decreased SWS. This highlights the need to incorporate both subjective and polysomnographic measures of sleep. This is particularly relevant in relation to depression, which is commonly associated with decreased SWS (Tsuno, Besset, & Ritchie, 2005). Factors affecting subjective perceptions of sleep quality are investigated later in this thesis.

REM Sleep

Several theories have been proposed regarding the specific function of REM sleep; many of which revolve around the nature of dreams (Neubauer & McHugh, 2003). REM sleep is considered critical for brain development, due to the fact that foetuses and new-borns spend around 50% of their sleep in the stage (Neubauer & McHugh, 2003). It is also the stage most strongly associated with dreaming (Dement & Kleitman, 1957). However, dreaming and REM are dissociable states, each controlled by different mechanisms (Solms, 2000). Whereas REM sleep is controlled by cholinergic brain stem mechanisms, dreaming is thought to be controlled by dopaminergic forebrain mechanisms (Solms, 2000). Solms (2000) states that dreaming is the subjective information about what the brain does during REM sleep. Dreaming can also occur in NREM sleep but may not be remembered to the same extent (Suzuki et al., 2004).

Research has suggested that REM sleep may serve a mood regulatory function (Cartwright & Lloyd, 1994; Cartwright, Young, Mercer, & Bears, 1998). Cartwright et al. (1998) suggest that early negative dreams, and dreaming about current emotional events may serve to regulate and improve mood over time. Furthermore, Walker and van der Helm (2009) argue that the brain mechanisms involved in REM sleep provide an environment for emotive memory to be re-activated and for the emotional component of the memory to be processed (Walker & van der Helm, 2009). Over time, this process is argued to support mood regulation. The mood-regulatory function of sleep will be explored in more detail within Chapter 2.

1.4 Measuring Sleep

1.4.1 Objective Measures

Polysomnography (PSG)

So far, the focus has been on the physiological nature of sleep and its function. PSG is considered the 'gold standard' method of objectively measuring sleep (Kushida et al., 2005), and is the only method that currently provides detailed information on sleep architecture. Although PSG provides the most comprehensive information about sleep, it is also very time-consuming, expensive, resource-demanding and difficult to use (Van de Water, Holmes, & Hurley, 2011), therefore it may not always be practical.

PSG can be conducted within a sleep laboratory, or ambulatory PSG can be used in the home. Ambulatory PSG may be preferable as it provides a more naturalistic environment, and is also more convenient since an individual does not have to leave their home. Therefore it may be particularly useful within populations where it is difficult to visit a sleep laboratory. One such instance may be new mothers who wish to remain close to their newborn infant. However it may be less suitable for the diagnosis of certain sleep disorders as it may not be possible to include as many measures as in the laboratory, such as video recording.

The first night effect is the finding that individuals undergoing laboratory-based PSG experience significantly worse sleep during their first night of testing (Agnew, Webb, & Williams, 1966), and therefore more than one night of PSG may be necessary. A benefit of ambulatory PSG is that the first night effect appears to be avoided (Lee, Zaffke, & McEnany, 2000; Sharpley, Solomon, & Cowen, 1988).

Actigraphy

Another commonly used objective sleep measure is actigraphy. This is usually a small-watch shaped device that is worn around the wrist or upper arm, and monitors sleep-wake cycles. It is commonly used in research as it is a cheaper alternative to PSG. It is effective for the diagnosis of circadian rhythm disorders, for assessing sleep patterns in insomniacs and for studying the effects of treatment designed to improve sleep (Ancoli-Israel et al., 2003). It may be used in conjunction with sleep diaries and can provide information on total sleep time, sleep efficiency and awakenings (Morgenthaler et al., 2007).

Actigraphy is a useful tool for populations in which PSG may be difficult or inconvenient to administer, such as children or elderly adults with dementia (Ancoli-Israel et al., 2003). Another benefit is that it can be worn continually for several weeks; thus providing more information on night-to-night sleep variability and sleep patterns over time. Participants sleep at home rather than at a sleep laboratory, which may be more convenient and naturalistic.

Although some studies have shown actigraphy to be relatively accurate in measuring total sleep time (e.g. Jean-Louis et al., 1996), overestimation can occur since quiet wakefulness can be misinterpreted for sleep (Kushida et al., 2001). A major limitation of most actigraphy devices is that they do not provide validated information on sleep stages. This makes it unsuitable for the diagnosis of sleep disorders that rely on detection of abnormal sleep architecture, such as narcolepsy in which the patient enters sleep through REM rather than progressing through the stages; or depression, which may involve reduced REM latency and reduced SWS (described further in Chapter 2, Kupfer & Ehlers, 1989; Kupfer, 1984)

More recent technology includes wireless systems that claim to be able to measure sleep stages, such as the 'Zeo' (Zeo, Inc., Newton, MA, USA). One study reported approximately 75% accuracy in detection of sleep stages in the Zeo device, compared to PSG-scored sleep (Shambroom, Fábregas, & Johnstone, 2012). However, it was inaccurate in correctly determining REM latency. While such devices are promising for enabling simpler and easier to administer methods of measuring sleep in the future, they have not been commonly used or validated within the research to date.

Actigraphy has been the preferred measure of objective sleep amongst perinatal women,

which will be discussed in detail in Chapter 3.

1.4.2 Subjective Measures

Several scales have been created to measure subjective perceptions of sleep. Subjective sleep scales are important, since they are often the first point of measurement in the diagnosis of sleep disorders. For example, high scores on the Epworth Sleepiness Scale (Johns, 1991) are often found in individuals with obstructive sleep apnea (Johns, 1993), and the diagnosis of insomnia is typically through self-report measures (Roth, 2007). Within research, the term 'subjective sleep' has been used as somewhat of a sweeping term to categorise the many aspects of subjective sleep. However, subjective sleep measures vary considerably in the type of sleep they are measuring (e.g. sleep disorders, daytime sleepiness or general sleep patterns), the time-frame that the questions refer to (e.g. the previous night versus the previous month) and the type of questions that are asked (e.g. quantitative versus qualitative). The choice of questionnaire is therefore important in terms of the particular aspect of sleep that is being explored. The following sections explore the different types of subjective sleep measures. An overview of these measures is provided in Table 1.

As seen in Table 1, the subjective sleep scales vary considerably according to the different ways in which sleep is measured. These scales can be categorised in several ways. The former scales are focused on general aspects of sleep (Akerstedt et al., 1994; Ellis et al., 1981; Monk et al., 1994; Monk et al., 2003; Parrott & Hindmarch, 1978) whilst the latter scales are specifically focused on diagnosing sleep disorders (Bastien, Vallières, & Morin, 2001; Douglass et al., 1994; Partinen & Gislason, 1995; Roth et al., 2002; Spoormaker, Verbeek, van den Bout, & Klip, 2005). They also differ in the time-frame in which sleep is measured (e.g. prospective versus retrospective measures). Many of the scales ask questions relating to sleep characteristics that have occurred in periods somewhat prior to the time in which the questionnaire is administered (e.g. the Pittsburgh Sleep Quality Index, PSQI). While these scales may provide a general overview of an individual's sleep, retrospective answers may be inaccurate due to false recall or forgetfulness (Babkoff, Weller, & Lavidor, 1996; Lomeli et al., 2008). This is an important consideration when choosing the best measure to use within a research design. Prospective scales that incorporate diaries relating to the previous night's sleep may provide a more detailed overview of a particular night or period of time.

Table 1. Overview of Subjective Sleep Measures

Instrument	Authors	Aspects Evaluated	Period Evaluated
St Mary's Hospital Sleep Questionnaire	Ellis et al. (1981)	14 items covering quantitative (timings, awakenings) and qualitative aspects (depth, satisfaction, clear-headedness)	Previous 24 hours
Sleep Timing Questionnaire	Monk et al. (2003)	Typical sleep and wake times on different days (days off versus work days) and stability of timings.	Referring to a recent 'normal average' week
Leeds Sleep Evaluation Questionnaire (LSEQ)	Parrott & Hindmarch (1978)	Covers 4 aspects: getting to sleep, sleep quality, awakenings and behaviour following waking.	Designed to measure changes in sleep during psychopharmacological investigations
Karolinska Sleep Diary (KSD)	Akerstedt, Hume, Minors, & Waterhouse (1994)	12 items referring to sleep quality, sleep onset latency, ease of waking up, sleep continuity	Evaluation of a single night
Pittsburgh Sleep Diary (PSD)	Monk et al. (1994)	One set of questions completed both at bedtime (referring to daytime behaviour e.g. exercise, caffeine, meals, medications) and upon waking (sleep timing, awakenings, quality, mood & alertness upon waking)	Evaluation of a single night
General Sleep Disturbance Scale (GSDS)	Lee (1992)	21-items with 7 subscales: sleep quality, quantity, sleep maintenance, early morning waking, use of sleep medications & daytime functioning	Refers to sleep over past week
Pittsburgh Sleep Quality Index (PSQI)	Buysse, Reynolds, Monk, Berman, & Kupfer (1989)	19 items with 7 subscales: sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medications & daytime dysfunction	Refers to sleep over past month
Medical Outcomes Study (MOS) Sleep Scale	Stewart & Ware (1992)	12 items relating to 6 dimensions: sleep initiation, quantity, maintenance, respiratory problems, perceived adequacy & somnolence.	Refers to sleep over past month
Insomnia Severity Index (ISI)	Bastien, Vallières, & Morin (2001)	7 questions relating to the nature, severity and impact of current insomnia symptoms	Refers to symptoms within past 2 weeks

Sleep Disorders Questionnaire (SDQ)	Douglass et al. (1994)	175 items relating to sleep disturbances & usual sleep habits. 4 sleep disorder categories: sleep apnea, narcolepsy, periodic limb movements & psychiatric sleep disturbances	Refers to sleep over past 6 months
Basic Nordic Sleep Questionnaire (BNSQ)	Partinen & Gislason (1995)	27 items referring to difficulty initiating / maintaining sleep, sleep quality, sleep medication, daytime sleepiness, napping, snoring, general sleep habits. Focuses on sleep apnea / snoring.	Refers to sleep over past 3 months
Global Sleep Assessment Questionnaire (GSAQ)	Roth et al. (2002)	11 items relating to 7 different sleep disorders.	Refers to sleep over past month
SLEEP-50 Questionnaire	Spoormaker, Verbeek, Van den Bout, & Klip (2005)	Designed to distinguish sleep disorders from subclinical symptoms & factors affecting sleep. 50 items relating to 9 sleep disorders.	Refers to sleep over past month

The scales also vary in the extent to which they have been validated and used within research. For example, the PSQI, a general measure of sleep quality, is widely used and has been validated in 56 languages (University of Pittsburgh: Pittsburgh Sleep Quality Index, 2013). Other scales were primarily created to detect sleep problems in specific populations. For example, the LSEQ was designed to measure sleep during psychopharmacological investigations (Parrott & Hindmarch, 1978).

The way in which sleep is measured is a key theme within this thesis, and will be further explored in later chapters. However, it is particularly important to consider the time-frame in which sleep is measured when examining sleep during a certain period, such as the perinatal period, where sleep patterns are continually changing (Lee, Zaffke, & McEnany, 2000). This is also important when considering the relationship between sleep and depression, as depression is associated with a negative cognitive bias (Beck, 1964), which could further affect the accuracy of retrospective sleep perceptions.

Aside from measuring specific aspects of sleep, we can also examine sleep-related factors that affect wakefulness, such as fatigue and sleepiness. The terms 'sleepy' and 'fatigue' are often

poorly defined and used interchangeably, however these are distinct constructs (Shahid, Shen, & Shapiro, 2010). Sleepiness refers to increased propensity to doze off or fall asleep (Curcio, Casagrande, & Bertini, 2001). Excessive daytime sleepiness is commonly experienced in those with narcolepsy and obstructive sleep apnea (Seneviratne & Puwanendran, 2004; Zeman et al., 2004). In contrast, fatigue reflects a feeling of strain or exhaustion, and can be either physiological (normal fatigue induced by physical activity, and reduced upon rest) or pathological (e.g. chronic fatigue; Shahid et al., 2010). Both sleepiness and fatigue have been related have been associated with depression. As with sleep scales, there are various methods of assessing sleepiness and fatigue. These are described in the following sections.

1.4.3 Fatigue

Fatigue is commonly experienced in both physical and psychiatric conditions (Loge, Ekeberg, & Kaasa, 1998), and is the most commonly reported symptom for physicians (Shahid et al., 2010). It is commonly reported in depression (Baldwin & Papakostas, 2006). However, fatigue itself is poorly defined and lacks an objective measure (Taylor, Jason, & Torres, 2000). One reason for this is because fatigue is a multidimensional construct (Belza, Henke, Yelin, Epstein, & Gilliss, 1993). Therefore, fatigue scales including several subscales relating to fatigue symptoms as well as their impact on daytime functioning are most useful (e.g. the Multidimensional Assessment of Fatigue Scale). Although it may be assumed that fatigue is related to sleep disturbances, this may not always be the case. Studies assessing the relationship between fatigue and both subjective and objective sleep have shown that fatigue is significantly related to subjective but not objective sleep (Fortier-Brochu, Beaulieu-Bonneau, Ivers, & Morin, 2010; Lavidor, Weller, & Babkoff, 2003). Fatigue has also been related to depression (Corwin, Brownstead, Barton, Heckard, & Morin, 2005; Lavidor et al., 2003), which will be explored within the studies of this thesis. It is also important to consider the time-frame in which the scale refers to, such as the past week versus the past month. This may be particularly important during periods in which changes to fatigue changes are common, such as during the perinatal period (Lee & Zaffke, 1999). Table 2 provides a summary of some commonly used fatigue scales.

1.4.4 Sleepiness

Sleepiness can be measured objectively or subjectively. A commonly used objective measure

is the multiple sleep latency test (MSLT). Individuals are fitted with EEG and provided with 4-5 nap opportunities, each around 30 minutes, at two hour intervals (Carskadon & Dement, 1982). The premise behind this test is that sleepiness represents sleep propensity, and therefore sleepy individuals should fall asleep when given the opportunity. Another objective measure is the Maintenance of Wakefulness Test (MWT; Doghramji et al., 1997). Individuals are fitted with EEG and instructed to sit in a dimly lit room for around 30 minutes and to stay awake, being measured on their ability to stay awake. However it has been debated as to whether this reflects sleepiness or strength of the arousal system (Shahid et al., 2010).

Subjective measures of sleepiness can be split into state and trait measures. State measures include the Stanford Sleepiness Scale (SSS; Hoddes, Zarcone, & Dement, 1972) and the Karolinska Sleepiness Scale (KSS; Akerstedt & Gillberg, 1990). The SSS is the most widely used measure of subjective sleepiness (Shahid et al., 2010), and asks subjects to indicate their current levels of vigilance on a 7-point scale. The KSS is a 9-point scale that measures levels of sleepiness at a particular time during the day, and is therefore subject to fluctuation.

A commonly used measure of trait sleepiness is the Epworth Sleepiness Scale (ESS; Johns, 1991). Individuals are provided with eight common daytime scenarios and are asked to rate the likelihood that they would fall asleep in a given situation, on a scale of 0-3. The ESS has been shown to have good reliability and internal consistency (Johns, 1991). It is commonly used as a screening for obstructive sleep apnea in clinical settings (Rosenthal & Dolan, 2008).

Another questionnaire measure of sleepiness is the Sleep-Wake Activity Inventory (Rosenthal, Roehrs, & Roth, 1993), which measures multidimensional components of sleepiness. It includes 59 items in which a subject is asked to rate on a scale of 1-9, the extent to which a particular behaviour has been present over the past 7 days.

Table 2. Overview of Fatigue Measures

Instrument	Authors	Aspects Evaluated	Period Evaluated
Brief Fatigue Scale (BFI)	Mendoza et al. (1999)	Designed for cancer patients to assess fatigue severity and impact on daily functioning. Includes 9 items.	Past week / past 24 hours / current
Fatigue Severity Scale (FSS)	Krupp, LaRocca, Muir-Nash, & Steinberg (1989)	9 items measuring impact of fatigue on functioning and behavioural aspects.	Past week
Checklist Individual Strength (CIS)	Vercoulen et al. (1994)	24 items with 4 subscales: subjective experience, concentration, motivation & physical enjoyment.	Past 2 weeks
Fatigue Assessment Inventory (FAI)	Monk et al. (1994)	29 items referring to both qualitative & quantitative aspects of fatigue. 4 subscales: fatigue severity, situation specificity, psychological consequences, and response to rest / sleep.	Past 2 weeks
Fatigue Impact Scale	Fisk et al. (1994)	40 items measuring impact of fatigue on cognitive, physical & psychosocial functions.	Past 4 weeks
Multidimensional Assessment of Fatigue Scale	Belza, Henke, Yelin, Epstein, & Gilliss (1993)	16 items measuring 4 dimensions: severity, distress, degree of interference in daily activities, timing.	Past week
Visual Analogue Scale – Fatigue (VAS-F)	Lee, Hicks, & Nino-Murcia (1991)	18 item VAS scale to measure fatigue and energy.	Current
Revised Piper Fatigue Scale	Piper et al. (1998)	22 items with 4 subscales: behavioural / severity, affective meaning, sensory, cognitive / mood.	Current
Multidimensional	Smets,	20 items with 5 subscales:	Past few days

Fatigue Inventory (MFI)	Garssen, Bonke & De Haes (1995)	general, physical, mental, motivation & activity.
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1.5 Relationship between Objective and Subjective Measures of Sleep

So far this chapter has described the physiological nature of sleep, the functions of sleep, and the different subjective and objective measures that we can use to assess sleep and fatigue. This section explores the relationship objective and subjective measures of sleep, which is a key theme throughout this thesis. Subjective perceptions of sleep may not provide an accurate reflection of actual sleep, and certain populations such as those with depression may be less accurate in their subjective sleep perceptions (Baker, Maloney, & Driver, 1999; Rotenberg et al., 2000). Despite these findings, few studies use both subjective and objective measures together. Within those that do, the relationship between objective and subjective sleep is often not reported. This is important within the context of this thesis for the following reasons:

- Many studies exploring the relationship between sleep and postpartum depression have relied upon a single measure of sleep, and therefore cannot provide a complete picture of the nature of this relationship.
- Subjective measures are commonly used as a first step in the diagnosis of sleep disorders. Therefore, it is crucial for health professionals to understand the relationship between subjective and objective sleep in relation to diagnosis and treatment options.
- Since subjective sleep reports are the most commonly used measure of sleep, it is important in order to fully understand the underlying factors affecting subjective sleep. For example, which factors affect how an individual perceives and reports their sleep?

The relationship between subjective and objective sleep can be assessed in relation to both qualitative and quantitative aspects. Firstly, we can examine the extent to which individuals are accurate in reporting various quantitative aspects of their sleep in relation to objective

measures. Within this, we can also examine other factors that may affect this relationship, and whether certain groups of individuals are less accurate in reporting their sleep. Second, we can examine the factors relating to subjective sleep quality. For example, which sleep variables are most strongly related to an individual's subjective perception of their overall sleep quality? These two questions are explored further in the following section, and are addressed in the main study of this thesis.

1.5.1 Accuracy of Subjective Sleep

Surprisingly little research has explored the accuracy of subjective sleep perceptions. The largest study to date comparing subjective and objective sleep was taken from the Sleep Heart Health Study (Silva et al., 2007). In this study 2,113 participants over 40 years old underwent one night of home PSG and were asked to complete a morning questionnaire regarding their sleep. The results showed that participants overestimated both their total sleep time and sleep onset latency. After adjusting for demographic factors, subjective total sleep time was significantly greater than PSG-measured total sleep time. Additionally, lower educational level was associated with a greater degree of sleep misperception; suggesting that non-sleep factors may play an important role in the subjective sleep reporting.

Jackowska et al. (2011) compared subjective and objective estimates of sleep efficiency in 199 females using actigraphy. After adjusting for personal income, age, having children, marital status, body mass index and negative affect, the factors associated with underestimation of sleep efficiency included over-commitment, low social support and poor self-reported health. This further suggests that the accuracy of sleep perceptions may be influenced by psychosocial factors.

Accuracy of sleep reporting may also be affected by other health conditions. Research has highlighted a subgroup of insomnia sufferers who report poor sleep despite showing normal sleep when studied objectively (Edinger & Krystal, 2003). This has been classified as 'sleep state misperception' or 'subjective insomnia' (Edinger & Krystal, 2003). Fernandez-Mendoza et al. (2011) found that sleep state misperception in insomnia patients was associated with depressive symptoms, anxious, ruminative personality traits and poor coping style.

Others have also suggested that sleep state misperception may be a feature of depression. Rotenberg et al. (2000) found that the degree of wrong sleep timing estimation was larger in depressed individuals compared to controls. Similarly, Tsuchiyama et al. (2003) assessed sleep perceptions against PSG variables in 23 depressed patients and found that the degree of inaccuracy also correlated with severity of depression. The degree of inaccuracy was also shown to increase in accordance with the degree of objective sleep disturbance (particularly decreased SWS and increased awakening). This suggests that those experiencing sleep disruptions may be less accurate in their subjective sleep perceptions, which may be due to decreased cognitive performance.

Given that the research to date suggests that disparities exist between subjective and objective measures of sleep, both types of measurements should ideally be employed within research. Chapter 6 describes the accuracy of sleep in pregnant and non-pregnant women. Since levels of inaccuracy in sleep perceptions appear to relate to levels of sleep disruption (Tsuchiyama et al., 2003), it is possible that due to disturbed sleep, pregnant women are less accurate at perceiving their sleep. Therefore there is a need to examine the relationship between subjective and objective measures in this group.

1.5.2 Factors Affecting Subjective Sleep Quality

Poor subjective sleep quality appears to be a risk factor for the development of depression. This has been found in a number of populations including pregnant women (Bei, Milgrom, Ericksen, & Trinder, 2010) and young women at high risk of depression (Chen, Burley, & Gotlib, 2012). This relationship is explored in greater depth in Chapter 2. In order to understand the mechanistic pathway linking these factors, it is important to understand the factors that affect subjective sleep quality. Although some research has suggested that subjective sleep quality may be related to sleep architecture, these findings are inconsistent (Akerstedt et al., 1997; O'Donnell et al., 2009; Saletu, 1975). Since SWS is considered the deepest form of sleep, it may be expected that amounts of deep sleep are positively correlated with subjective reports of sleep quality. Although some research has found this to be the case (e.g. Akerstedt et al., 1997; Keklund & Akerstedt, 1997), other research has not (Saletu, 1975). Some have found that subjective sleep quality is positively related to stage 2 sleep (Saletu, 1975) and REM sleep (Mendelson, James, Garnett, Sack, & Rosenthal, 1986; Saletu, 1975). The

inconsistency between these studies may reflect lack of standardisation in study design and choice of measures. It is clear that further research is needed using both subjective and objective measures of sleep to try and resolve the differences in findings that have emerged.

As well as reflecting underlying sleep architecture, subjective sleep may also be related to mental health. Mayers, Grabau, Campbell and Baldwin (2009) found that poor subjective sleep quality was most strongly related to depression, whilst poor sleep quantity was related to anxiety. Individuals with depression reported poorer subjective sleep satisfaction, even though they reported normal amounts of sleep. One possibility is that subjective sleep quality is not necessarily related to sleep quantity in those with depression, and may be more strongly related to overall mood. However, it is also possible that the reason that those with depression reported poorer sleep quality was related to differences in their sleep architecture (such as reduced SWS), but this was not analysed in Mayers et al's study.

Furthermore, in a study of patients with obstructive sleep apnoea, Wells, Day, Carney, Freedland, and Duntley (2004) found that depression was a significant predictor of subjective sleep quality after controlling for polysomnographic measures of sleep (including sleep onset latency, sleep efficiency and arousals). Again, this suggests that individuals with depression report poor subjective sleep quality despite showing healthy objective sleep.

In a study examining relationships between sleep quality, sleep quantity, health and well-being amongst college students, Pilcher, Ginter and Sadowsky (1997) found that subjective perception of sleep quality was not strongly related with subjective sleep quantity. Instead, sleep quality was related to aspects of well-being including depression, fatigue and affect balance, suggesting that an individual's mental health may be a better predictor of subjective sleep *quality* than sleep *quantity*.

Overall, few studies have examined the relationship between sleep quality and other factors, although there is some evidence to suggest that perceptions of sleep quality may be related to sleep architecture. Poor subjective sleep quality is also likely to play an important role in depression. The relationship between depression, sleep quality and sleep quantity is investigated in Chapter 4.

1.5.3 Overview of Subjective and Objective Measures of Sleep

Sleep is complex and can be measured in different ways. Subjective and objective sleep are two categories in which we can define a measure of sleep, but within each of these we can also look at qualitative and quantitative factors. The way in which an individual perceives their sleep may not only be affected by various aspects of their sleep, but also by non-sleep related factors, such as mood.

Throughout this thesis particular emphasis will be placed upon the distinction between objective and subjective measures of sleep. The majority of research looking at the relationship between sleep and postpartum depression has focused primarily on subjective rather than objective measures and has neglected the fact that a significant discrepancy may exist between these measures. This is important in order to assess which sleep factors are most strongly related to postpartum depression. Exploring these different aspects of sleep in more detail will shed light upon the mechanistic pathway in which sleep problems may develop into depression.

1.6 Perinatal Sleep

The major aim of this thesis is to investigate the relationship between sleep and postpartum depression. While a review of this relationship is provided in Chapter 3 it is first important to consider why sleep patterns during this period are unique.

In order to highlight the significance of the sleep changes that occur during this time, it is useful to consider that it is normal for sleep patterns to change throughout the lifespan. Changes occur to the amount of time spent in each stage of sleep and to the overall organisation of sleep, which partly reflect different developmental processes occurring with age. Figure 3 shows how sleep architecture changes throughout the lifespan. The majority of sleep changes occur from early infancy into childhood and adolescence. Amounts of REM sleep, Non-REM sleep and wakefulness decrease significantly from early infancy and childhood into adolescence and adulthood. The perinatal period reflects a time during which women are likely to undergo significant sleep changes in comparison to other periods during their adult life. Despite these changes, and their potential implications for both mother and baby health, research within the field of perinatal sleep is limited.

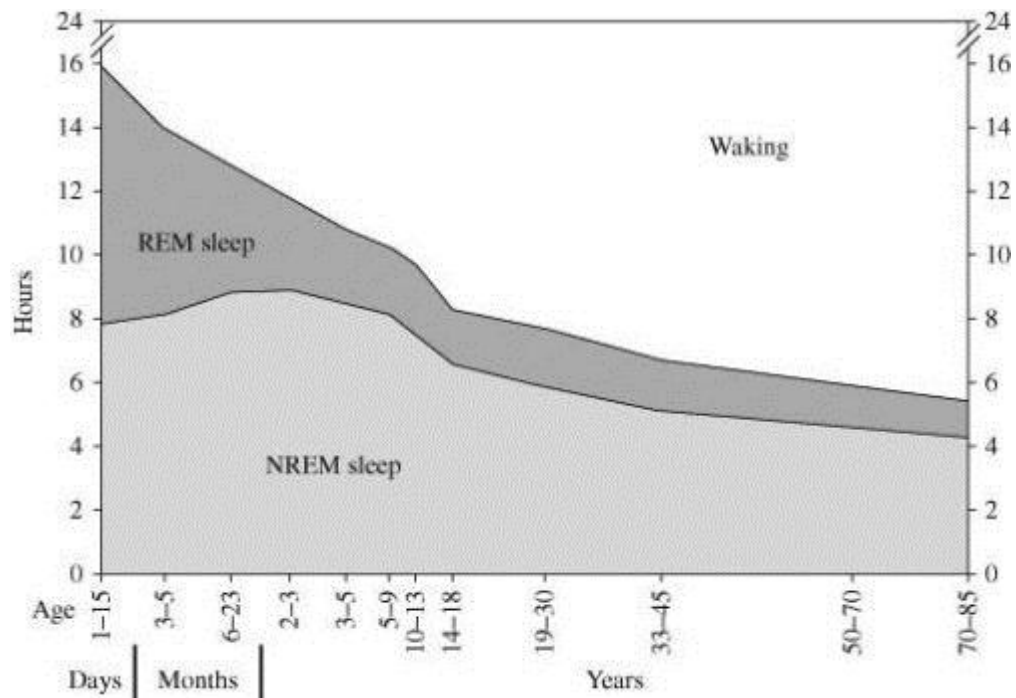


Figure 3. Changes in sleep architecture throughout the lifespan (from Huber & Tonini, 2009)

1.6.1 Sleep during Pregnancy

Pregnancy is a time during which women's sleep is likely to be disturbed. This is due to a combination of physical, hormonal and physiological changes. Facco et al. (2010) reported that nearly 40% of women in the first trimester reported poor overall sleep quality. The percentage of women reporting short sleep duration and poor sleep quality significantly increased in the third trimester. Schweiger (1972) found that 68% of pregnant women reported disturbed sleep, with the most prominent changes occurring in the third trimester. First trimester sleep disturbance was related to nausea, vomiting, backache, and increased need to urinate. Second trimester sleep was disrupted due to foetal movement and heartburn, and in the third trimester women reported increased need for urination, shortness of breath, leg cramps and itching. An increase in the frequency and severity of nightmares has also been reported during pregnancy (Baratte-Beebe & Lee, 1999; Lee & DeJoseph, 1992).

In terms of sleep quantity, several studies have shown that total sleep time increases in the first trimester, may normalise in the second trimester (Lee, Zaffke, & McEnany, 2000; Suzuki,

Dennerstein, Greenwood, Armstrong, & Satohisa, 1994) but then decreases throughout the rest of pregnancy. The majority of studies agree that sleep becomes most disturbed in the third trimester (Baratte-Beebe & Lee, 1999; Hedman, Pohjasvaara, Tolonen, Suhonen-Malm, & Myllylä, 2002; Lee, Zaffke, & McEnany, 2000; Schweiger, 1972).

A small number of studies have explored the effects of parity on perinatal sleep. Using actigraphy, Signal et al. (2007) found that nulliparas (women with no previous children) experienced overall poorer sleep quality throughout pregnancy than multiparas (women who have had two or more births), but did not differ on total sleep time or frequency of napping. Reduced sleep efficiency in nulliparas in the third trimester was also reported by Wilson et al. (2011). In contrast, using PSG, Lee et al. (2000) found that it was multiparas who experienced poorer sleep efficiency during pregnancy, except at one month postpartum when nulliparas' sleep was significantly worse. However, this discrepancy could be due to differences in definitions of awakenings between the studies (Signal et al., 2007), which may have resulted in lower sleep efficiency in the study by Lee et al. (2000). More studies are needed to examine the effects of parity of sleep during this period.

Fatigue

Maternal fatigue is common both during late pregnancy and in the early postpartum period, with rates increasing from 20% prior to conception to 50-64% in the immediate postpartum period (Lee & Zaffke, 1999). Fatigue tends to increase during pregnancy, peaking in the third trimester (Elek, Hudson, & Fleck, 1997). It is common for new mothers to report feeling drained, tired and exhausted (Dennis & Ross, 2005). Fatigue is often viewed as a normal consequence of becoming a new mother, however some research has suggested that it is associated with depression (Dennis & Ross, 2005; Doering Runquist, Morin, & Stetzer, 2009; Lee & Zaffke, 1999). Relationships between fatigue and both major and perinatal depression are investigated in later chapters.

Hormonal Factors

Some of the sleep changes during pregnancy relate to hormonal factors, which have been shown to affect sleep (Wilson et al., 2011). In particular, progesterone is known to have sedative-like properties, and may induce increases in non-REM sleep (Caufriez, Leproult,

L'Hermite-Balériaux, Kerkhofs, & Copinschi, 2011; Friess, Tagaya, Trachsel, Holsboer, & Rupperecht, 1997). However, Wilson et al. (2011) found that during the third trimester of pregnancy, when progesterone levels were highest, women experienced increased awakenings and more time awake during the night. In contrast, increased progesterone in control subjects was related to fewer awakenings. One possible explanation for these contrasting findings amongst pregnant women is that progesterone has also been shown to relax smooth muscle tone in the urinary system. Combined with foetal pressure on the bladder, this may exacerbate the need for urination during pregnancy, which could lead to increased nightly awakenings (Lee, 1998; Swift & Ostergard, 1993); and potentially counteract the sedative effects of progesterone.

It has been well-documented within rat studies that oestrogen, which increases during pregnancy, reduces amounts of REM sleep (Branchey, Branchey, & Nadler, 1971; Colvin, Whitmoyer, Lisk, Walter, & Sawyer, 1968). However, future research is needed to specifically examine relationships between hormonal levels and sleep architecture during human pregnancy (Santiago, Nollado, Kinzler, & Santiago, 2001). The effects of pregnancy on sleep are examined later in this thesis.

1.6.2 Sleep Disorders during Pregnancy

Although this thesis does not specifically examine perinatal sleep disorders, it is important to note that certain sleep disorders are more prevalent during this time.

Sleep-Disordered Breathing

Many women will develop snoring for the first time in pregnancy, partly due to weight gain (Loube, Poceta, Morales, Peacock, & Mitler, 1996). Bourjeily, Raker, Chalhoub and Miller (2010) found that 35% women report snoring by the end of their pregnancy, which was associated with higher incidence of pregnancy-induced hypertension, pre-eclampsia, gestational diabetes and unplanned caesarean.

It has been suggested that pregnancy may precipitate or exacerbate obstructive sleep apnoea (OSA), which involves periodic cessation of breathing and associated arousals. This is usually accompanied by heavy snoring and gasping after periods in which breathing stops. Apneic events are associated with decreases in oxygen saturation. OSA is also accompanied by

reports of excessive daytime sleepiness (Bassiri & Guilleminault, 2000). While some elements of pregnancy increase the risk of sleep-disordered breathing, there are also some protective factors. A major factor increasing the risk of OSA is weight gain (Quan, Budhiraja, & Parthasarathy, 2008), which is characteristic of pregnancy. Pregnancy hormones can also influence the respiratory system. Oestrogen is associated with increased mucosal oedema and vasomotor rhinitis, resulting in a narrowing of the upper airway thus increasing resistance (Venkata & Venkateshiah, 2009). In contrast, progesterone increases respiratory drive and minute ventilation, serving as a protective factor. Additionally, reduced REM sleep in pregnancy that has been reported in some studies (e.g. Hertz et al., 1992; Wilson et al., 2011) also decreases risk of apnoeic events, which are most common during this stage (Venkata & Venkateshiah, 2009). Whilst the prevalence of OSA in pregnancy remains unknown (Kowall, Clark, Nino-Murcia, & Powell, 1989), research suggests that it is associated with adverse pregnancy outcomes (Chen et al., 2012).

Restless Legs Syndrome

Restless legs syndrome (RLS), described as an uncontrollable desire to move the legs due to intense tingling sensations, is more common amongst pregnant women (Manconi et al., 2011). Symptoms of RLS can increase sleep onset and night-time awakenings, thus decreasing sleep efficiency (Allen & Earley, 2001). One study reported a prevalence of 26% in pregnancy, which was strongly associated with the third trimester and mostly disappeared around time of delivery (Manconi et al., 2011). Another reported third trimester prevalence rates of 32% (Neau et al., 2010). RLS appears to be associated with iron levels. Lee, Zaffke and Baratte-Beebe (2001) found that women with RLS during pregnancy had reduced serum folate levels and were also more likely to have a longer sleep-onset latency and poorer mood. Periodic limb movements, commonly associated with RLS, also increase in prevalence during pregnancy (Nikkola, Ekblad, Ekholm, Mikola, & Polo, 1996).

1.6.3 Physiological Changes

Sleep changes during pregnancy may also be related to the fact that pregnancy is primarily an anabolic period associated with increased metabolic rate (Richardson, 1996). The first two thirds of pregnancy are devoted to maternal anabolic activity and tissue development, whilst the last third of pregnancy favours anabolic activity related to growth and maturation of the

foetus (Richardson, 1996). Given this, Driver and Shapiro (1992) state that pregnancy provides 'a natural experiment for testing the theory that sleep, especially slow-wave sleep...has an anabolic and restorative function' (p.452). However, few studies have examined these changes, and findings to date do not appear to support the idea that pregnant women have more slow-wave sleep as a result of the anabolic demands of pregnancy. Other factors, such as the hormonal and physical changes previously described are also likely to influence sleep during this time.

A review by Lee (1998) concluded that, over a period of thirty years, less than twenty studies had examined sleep during pregnancy. Although this research area has received more attention in recent years, studies of PSG during pregnancy are limited. These studies are described in the following section.

1.6.4 Changes in Sleep Architecture during Pregnancy

Sleep in Pregnant Versus Non-Pregnant Women

Only three studies to date appear to have used a control group of non-pregnant women to examine pregnancy-related changes in sleep architecture. Karacan et al. (1968) were the first to examine perinatal sleep using laboratory-based PSG. In this study, the authors examined the sleep of seven pregnant women during the third trimester and at two weeks postpartum, compared to seven non-pregnant controls. Pregnant women took longer to fall asleep, had more awakenings and less SWS, compared to controls. No differences were found in amounts of REM sleep between groups. Following this, Hertz et al. (1992) studied the sleep of 12 third trimester women, compared to 10 controls using laboratory-based PSG. Pregnant women showed poorer sleep efficiency, less REM sleep and more stage 1 sleep, but in contrast to Karacan et al., no significant differences were found in amounts of SWS between groups.

A more recent study by Wilson et al. (2011) compared the sleep of 21 first trimester women, 27 third trimester women, and 24 non-pregnant controls, using laboratory-based PSG. Women in the third trimester had poorer sleep efficiency, more awakenings, less stage 4 sleep, more stage 1 sleep and less REM sleep, compared to controls. Women in the first trimester also experienced reduced stage 4 sleep and more awakenings compared to controls, but to a lesser extent than those in the third trimester.

Compared to non-pregnant women, it seems that women in the third trimester experience significantly poorer sleep quality on a number of dimensions. However, findings regarding differences in sleep stages, particularly REM and SWS, are conflicting. Furthermore, each of these studies used laboratory-based PSG which may lack ecological validity. Clearly, more studies with larger sample sizes are needed in order to better understand the sleep architectural changes associated with pregnancy.

Longitudinal Changes in Sleep throughout Pregnancy

Rather than comparing sleep in pregnant and non-pregnant women, the following studies have examined how women's sleep changes throughout their pregnancy. Branchey and Petre-Quadens (1968) studied the sleep of 17 women at various points throughout their pregnancies, using laboratory-based PSG. REM sleep significantly increased during weeks 33-36 but returned to normal towards the end of pregnancy. No data were reported on other sleep stage variables. Also using laboratory-based PSG, Driver and Shapiro (1992) studied the sleep of 5 women, every 2 months throughout their pregnancies. Amounts of SWS significantly increased in the second and third trimester, compared to the first trimester. Amount of REM sleep was significantly lower in the third compared to first trimester. However, not all women were studied at the same time-point, making comparisons between participants difficult. Additionally, it is unclear why only the first 6 hours of the PSG recording were used, since it is likely that some of the REM sleep common in the latter part of the night would have been missed.

Brunner et al. (1994) studied the sleep of 9 women during each trimester using laboratory-based PSG. Women experienced increased waking in the third trimester, however no significant differences changes were reported in amounts of SWS according to trimester. High variability was found in REM sleep within the sample, with a trend for decreased REM sleep in later pregnancy.

Two additional studies examined women's sleep during pregnancy, using ambulatory PSG, which provides a more naturalistic environment. Coble et al. (1994) studied the sleep of 34 women, 14 of whom had a history of affective disorder. Sleep changes were modest throughout pregnancy, but significantly poorer at one month postpartum. Women with a history of affective disorder slept longer during the third trimester, but significantly shorter

postpartum, compared to women with no history. The REM latency of these women was significantly shorter from the third trimester, and remained lower throughout the postpartum period.

The most extensive study of longitudinal sleep during pregnancy using PSG was carried out by Lee et al. (2000), who examined sleep changes in 30 women from pre-conception, throughout each trimester of pregnancy and at 1 and 3 weeks postpartum. Time spent in REM sleep did not significantly differ according to time point, but SWS was significantly reduced throughout pregnancy compared to baseline and postpartum measures.

Available sleep stage data derived from the above studies has been summarised in Table 3. What is evident from this table is the large discrepancies that exist between studies. Percentages of SWS range from 9-28% in the first trimester, 10-36% in the second trimester, and 8-36% in the third trimester. Percentages of REM sleep are less variable; ranging from 20-27% in the first trimester, 18-24% in the second trimester and 16-23% in the third trimester. Examining the average sleep stage values according to each trimester, it appears that SWS and REM are both reduced in the third trimester, compared to the first and second trimester; but the differences are small.

While the main aim of this thesis is to investigate the effects of sleep on postpartum depression, given the mixed findings to date, this thesis will further investigate the effects of pregnancy on sleep. Chapter 6 will investigate differences in sleep in pregnant and non-pregnant women using polysomnography, and Chapter 7 will investigate longitudinal changes to sleep, fatigue and depression, before finally examining the relationships between sleep and depression in Chapter 8.

Table 3. Overview of PSG data during pregnancy

		1 st Trimester					2 nd Trimester					3 rd Trimester				
	Sample Size	St.1 %	St.2 %	SWS %	REM %	REML (mins)	St.1 %	St.2 %	SWS %	REM %	REML (mins)	St.1 %	St.2 %	SWS %	REM %	REML (mins)
Karacan et al. (1968)	7	-	-	-	-	-	-	-	-	-	-	6	62	10	23	-
Driver & Shapiro (1992)	5	4	34	28	27	66	5	35	36	20	65	5	34	36	23	79
Coble et al. (1994)	34	4	59	16	21	83	4	35	16	20	83	4	61	15	20	81
Brunner et al. (1994)	9	9	55	16	20	-	8	61	16	18	-	9	58	14	19	-
Hertz et al. (1992)	12	-	-	-	-	-	-	-	-	-	-	19	55	12	14	125
Lee et al. (2000)	33	3	54	9	24	74	-	-	10	24	-	4	56	8	21	87
Wilson et al. (2011)	21 (1 st T) / 27 (3 rd T)	8	41	36	16	118	-	-	-	-	-	9	43	32	16	126

Average	6	49	21	22	85	6	44	20	21	74	8	53	18	19	100
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Note. T = trimester

1.7 Postpartum Sleep

Sleep disruptions are inevitable in the early postpartum period, when the mother has to partake in infant care duties and nightly feeds. A new mother also has to adapt to the not yet fully established sleep-wake cycle of her new-born infant. Assessing women's sleep throughout pregnancy and the postpartum period, Lee, Zaffke and McEnany (2000) found that sleep disturbance was greatest at one month postpartum, particularly for first-time mothers. Sleep efficiency was significantly reduced in the postpartum compared to pregnancy, and remained lower than pre-pregnancy values at three months postpartum, despite the majority of sleep disturbances improving by this point. There was a significant increase in SWS from third trimester to one month postpartum, but no changes in amounts of REM sleep. This supports the restorative theory of deep sleep taking precedence over REM sleep in periods of sleep deprivation (Lee et al., 2000). A rebound in SWS in the immediate postpartum period compared to pregnancy ($n=7$) was also reported by Karacan et al. (1968). This was also accompanied by a reduction in REM sleep. In contrast, Nishihara and Horiuchi (1998) found that amounts of SWS and REM remained stable throughout pregnancy and postpartum ($n=8$). However, postpartum sleep was characterised by an increase in waking and reduced stage 2 sleep.

Others have examined postpartum sleep using actigraphy (Gay, Lee, & Lee, 2004; Kang, Matsumoto, Shinkoda, Mishima, & Seo, 2002; Signal et al., 2007) which allows for sleep to patterns to be measured over longer periods of time. Kang et al. (2002) found that in the weeks following delivery, women ($n=10$) experienced less total sleep time, poorer sleep efficiency and increased waking, compared to pregnancy. Similarly, with a larger sample, Signal et al. (2007) found that at one week postpartum, women experienced on average 1.5 hours less total sleep time, three times as many sleep episodes in a 24-hr period, more napping and greater day-to-day variability in sleep, compared to pregnancy.

Examining the sleep of 72 couples using actigraphy throughout pregnancy and the postpartum period, Gay et al. (2004) found that both mothers and fathers experienced more sleep disruption in the postpartum period than during the mother's pregnancy, including less night time sleep and more nightly awakenings.

Insana, Williams and Montgomery-Downs (2013) found that average postpartum nocturnal total sleep time was 7.2 hours, which did not change significantly from 2-16

weeks. The authors proposed that the biggest contributor to postpartum sleep disruption is sleep fragmentation rather than sleep deprivation.

In a recent study, the same authors found that despite improvements in sleep from 2-13 weeks postpartum, neurobehavioural performance worsened over time (Insana et al., 2013). They suggest that this is due to cumulative effects of sleep disturbance over time. Research has shown that the cumulative effect of sleep fragmentation is equivalent to a night of sleep deprivation (Bonnet & Arand, 2003), which results in neurobehavioural impairment equivalent to a 0.10% blood-alcohol concentration (double the prescribed level of alcohol intoxication in many western industrialised countries, Dawson & Reid, 1997). The effects of sleep fragmentation on performance in postpartum women should therefore be given more attention, since research suggests that they are likely to experience significant impairments in everyday tasks, which could include caring for the infant. Furthermore, these impairments last at least several months after childbirth, persisting long after most women will have returned to work (Insana et al., 2013). The period at which these impairments persist remains unknown.

1.8 Sleep in Infants and Children

While this thesis does not specifically examine infant sleep, it is of interest to consider how infant sleep may contribute to postpartum sleep disruption. Newborn babies sleep up to 18 hours per day (Lee-Chiong, 2008). Their sleep is polyphasic, meaning that it occurs across the 24-hour period. The amount of total sleep decreases to around 14-15 hours by the 16th week, remaining at this level for the next 8 months (Parmelee, Schulz, & Disbrow, 1961). Sleep cycles last around 50 minutes in infants, compared to 60-70 minutes in children, and 90-120 minutes in adults (Lee-Chiong, 2008). Infants also differ significantly in their sleep architecture compared to adults, spending around half of their sleep in the stage of REM for the first few months of life (Lee-Chiong, 2008). Therefore, caregivers have to adapt to the sleep cycles of the infant. Interestingly, research has shown that sleep tends to be worse in infants whose mother is at risk for, or currently experiencing symptoms of postpartum depression (Armitage et al., 2009; Field et al., 2007; Karraker & Young, 2007). However, whether this is the 'chicken or the egg' remains unclear.

1.9 Overview of Chapter 1

This chapter has provided an overview of sleep, along with the specific ways in which sleep can be measured. Particular focus has been given to the difference between

subjective and objective sleep; a theme that reappears throughout this thesis. It has also described the extent to which sleep may change throughout the perinatal period, although this is an area which has received limited research attention to date.

Before exploring the relationship between sleep and postpartum depression, it is first important to explore research that has examined the relationship between sleep and major depressive disorder, since the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013) does not recognise PPD as a separate diagnosis. While the utility of this classification is questioned, it is important to understand the way in which sleep and depression appear to be related.

Chapter 2 describes the diagnoses of major depressive disorder and postpartum depression, followed by a brief overview of research that has analysed the relationship between sleep and depression.

CHAPTER 2: SLEEP AND DEPRESSION

2.1 Overview of Chapter

This chapter starts by describing the diagnostic criteria for major depressive disorder and postpartum depression. While the major focus of this thesis is on postpartum depression, this chapter describes how, diagnostically, these conditions are not necessarily viewed as distinct. Furthermore, more research to date has examined the relationship between sleep and major depressive disorder than postpartum depression. Given the diagnostic overlap between conditions, this chapter follows by briefly describing research that has examined the relationship between sleep and depression. This chapter sets the scene for Chapter 3, which provides a systematic review of the relationship between sleep and postpartum depression. Chapter 1 focused on the different ways in which sleep can be measured. Therefore, throughout this chapter, particular emphasis is given to specific aspects of sleep that appear to be most relevant to depression. This informed decisions regarding the measures of sleep that were appropriate to use in the studies described within this thesis.

2.2 Major Depressive Disorder (MDD)

2.2.1 Diagnosis

Depression is a common mental illness, with an approximate lifetime prevalence of 15% in high-income countries and 11% in low-middle income countries (Bromet et al., 2011). The average age of onset is around 25 years of age, with a female-male ratio of 2:1 (Bromet et al., 2011). The following criteria are listed in the DSM-5 for the diagnosis of a major depressive episode (American Psychiatric Association, 2013):

Five or more of the following symptoms must be present during the same two-week period, and must be a change from previous functioning. At least one of the symptoms must be depressed mood or loss of interest / pleasure. The symptoms must be accompanied with significant distress or impairment in social / occupational or other important areas of life. It must not be due to a medical condition or substance (e.g. drugs / alcohol).

- 1) *Depressed mood for most of the day, nearly every day.*
- 2) *Marked diminished interest or pleasure in most activities, most days.*

- 3) *Significant weight loss when no dieting or weight gain (a change of > 5% body weight in past month) or decrease / increase in appetite most days.*
- 4) *Insomnia / hypersomnia most days.*
- 5) *Psychomotor agitation or retardation, most days (observable by others, not just self-reports of restlessness / feeling slowed down).*
- 6) *Fatigue or loss of energy most days.*
- 7) *Feeling worthless / excessive or inappropriate guilt, most days (may be delusional; observable by others).*
- 8) *Decreased ability to think or concentrate, or indecisiveness, most days.*
- 9) *Recurrent thoughts of death (beyond fear of dying), recurrent suicidal ideation with / without current plan or attempted suicide.*

2.2.2 Aetiology

It is beyond the scope of this thesis to fully explore the aetiology of depression; however some of the main theoretical accounts are described briefly below. They indicate the different levels of explanation from genetic and biological through to socio-cultural and cognitive.

Genetic Factors

Twin studies have provided evidence for heritability of depression. For example, McGuffin, Katz, Watkins and Rutherford (1996) found that monozygotic twins had a depression concordance rate of 46% whilst the rate in dizygotic twins was 20%. Adoption studies have also shown a higher concordance of depression in adoptees and biological parents compared to non-biological parents (Shih, Belmonte, & Zandi, 2004). The specific genes involved in depression are thought to be those involved in the transmission of serotonin at the synapse (Surtees et al., 2006). Although Caspi et al. (2003) found that genetic variation in the serotonin transporter gene (5-HTTLPR) in interaction with stressful life events plays a role in predisposition to depression, a recent meta-analysis of studies in this area did not support this view (Risch et al., 2009).

Biological Explanations

The monoamine hypothesis of depression states that depression is due to a depletion in levels of serotonin, norepinephrine and dopamine (Delgado, 2000). Support for the theory comes from the effectiveness of many antidepressants, such as selective serotonin reuptake inhibitors (SSRIs), which work by increasing levels of serotonin at the synapse (Schildkraut, 1995). However, the theory fails to address the effectiveness of antidepressants in other disorders, or why other drugs that similarly enhance serotonergic transmission are not effective for depression (Hirschfeld, 2000). Despite this, the theory has been of great importance in the development of pharmacological treatment of depression, and over the years has been adapted to account for some of its limitations (Hirschfeld, 2000).

Socio-Cultural Factors

Prevalence of depression is particularly high among groups of individuals with low socio-economic status, ethnic minorities, those who have experienced traumatic life events, and those with poor social support (Grav, Hellzèn, Romild, & Stordal, 2012; Kessler, 1997; Lorant et al., 2003). This suggests that these factors play a role in the aetiology of depression.

Cognitive Factors

Several cognitive theories have been proposed to account for the development of depression. The theory of learned helplessness suggests that depression is a result of internal, stable and global beliefs (e.g. It's my fault, it will always go wrong, this is typical of my life; Abramson, Seligman, & Teasdale, 1976). A major cognitive theory, that has underpinned modern day cognitive therapy is that of Beck (1967). Beck argued that depression is a result of inaccurate cognitive responses to events, caused by automatic negative thinking. These depressonogenic thinking errors include overgeneralisation (drawing a general negative conclusion based on a single incident), personalisation (interpreting events as being caused by personal rather than external factors) and absolutistic thinking (thinking in 'all or nothing' terms).

2.2.3 Treatments

Current treatments in the United Kingdom predominately focus on cognitive and biological explanations of depression. The United Kingdom National Institute of Clinical

Excellence (NICE) guidelines specify a stepped-care approach to the treatment of depression (NICE, 2004). Sub-threshold and mild-moderate depression should be treated with guided self-help based on individual cognitive behavioural therapy (CBT), computerised or group CBT, or structured group physical activity programs. Antidepressant medication is recommended for those with severe / persistent depression, or with a previous history of depression.

2.3 Postpartum Depression (PPD)

Postpartum depression affects between 10-15% of women (Gale & Harlow, 2003). According to both the DSM-5 (American Psychiatric Association, 2013) and International Statistical Classification of Diseases and Related Health Problems (ICD-10; World Health Organisation, 2010), PPD is not considered a separate diagnosis to MDD. To be classified as PPD, symptoms must commence within 4 weeks postpartum according to the DSM-5, and within 6 weeks according to the ICD-10. This has received much criticism, with some arguing that this should be extended up to at least 6 months postpartum (Jones & Cantwell, 2010). For example, in a large Danish study of over 630,000 postpartum women, a three-fold risk of admission due to depression was found between 31-60 days postpartum, somewhat beyond the specified cut-off (Munk-Olsen, Laursen, Pedersen, Mors, & Mortensen, 2006). Others have found that increased risk for psychiatric illness persists until at least year one postpartum (Kendell, Wainwright, Hailey, & Shannon, 1976). Despite expert recommendations to increase the postpartum onset period in the latest DSM-5 (Jones & Cantwell, 2010) no major changes have been made regarding the diagnosis of PPD, except that the 'postpartum onset' specifier has been changed to 'peripartum onset,' given that many women who develop PPD will exhibit symptoms during pregnancy (American Psychiatric Association, 2013). Current diagnosis of PPD is therefore problematic given research findings which suggest that increased risk persists much longer than the diagnostic guidelines.

Although the diagnostic criteria for PPD are the same as MDD, some typical depressive symptoms, such as changes in weight and sleep loss, are characteristic of this period. Therefore, scales that have been adapted specifically for the postpartum period are useful diagnostic tools (e.g. the Edinburgh Postnatal Depression Scale, EPDS; Cox, Holden, & Sagovsky, 1987). Bernstein et al. (2008) found that while both non-postpartum and postpartum depressed women experienced low energy and restlessness, some differences emerged in other symptoms between groups. For example, whereas non-postpartum

women reported more sadness, reduced interest and suicidal ideation, postpartum women reported more restlessness, agitation and impaired concentration / decision-making.

Whether or not PPD is viewed as a separate diagnosis to MDD affects the way in which treatment options are viewed. If it is viewed as a unique condition specifically related to the birth of a child then there would be no reason to believe that standard treatment options would be appropriate (Whiffen, 1992). However, as PPD is not currently recognised as a separate diagnosis, treatment guidelines are similar to those of MDD (NICE, 2007).

Special guidance needs to be provided to pregnant and postpartum women regarding the use of antidepressant medications, as some pose more risk than others in being transferred to the unborn neonate or through breast milk (NICE, 2007). In a recent review, Field (2008) found that current knowledge regarding the effects of antidepressants on breast-milk is limited to uncontrolled studies with small samples. Due to this uncertainty, fewer mothers with postpartum depression breastfeed their infants compared to non-depressed mothers. However it remains unknown whether antidepressants or untreated depression have the most significant impact on the infant.

It is also important to distinguish PPD from other perinatal mood-related disorders. Between 35-80% women experience the postpartum blues, which typically diminishes after the first week of childbirth (O'Hara, 1987; Rollins, 1996) and is thought to be largely hormonal in its aetiology (O'Hara, Schlechte, Lewis, & Wright, 1991). Despite this, postpartum blues has been shown to significantly predict later postpartum depression (Fossey, Papiernik, & Bydlowski, 1997). In contrast, postpartum psychosis is considered a more severe mental illness affecting around one in 1000 women and may include symptoms of mania, severe depression or psychosis (Brockington, 2004). Despite vast differences in symptomatology, duration, aetiology and severity between the disorders, both postpartum psychosis and postpartum blues are often misclassified under the label of PPD (Jones & Cantwell, 2010).

A number of risk factors have been identified for PPD. Unlike the postpartum blues there is little research to suggest that PPD has a hormonal cause, suggesting that non-biological risk factors play an important role (Whiffen, 2004). The findings from a large meta-analysis conducted by Robertson, Grace, Wallington and Stewart (2004) identified

the following as strong predictors of PPD: depression or anxiety during pregnancy or a previous history of depression, experiencing stressful life events during pregnancy or the early postpartum and low levels of social support. However these risk factors are inadequate in predicting which women will develop PPD or recur during this time (Okun, Hanusa, Hall, & Wisner, 2009).

The extent to which poor sleep relates to the development of PPD is reviewed in Chapter 3. Before examining the research that has investigated sleep and postpartum depression, it is first important to examine research that has looked at the relationship between sleep and more general (e.g. major) depression. This is followed by a description of theories that have attempted to account for the relationship.

2.4 Sleep and Depression Research

The relationship between sleep disturbance and depression is well established. Not only does poor sleep present a risk factor for the development of depression (Chang, Ford, Mead, Cooper-Patrick, & Klag, 1997; Ford & Kamerow, 1989; Germain & Kupfer, 2008; Riemann & Voderholzer, 2003; Szklo-Coxe, Young, Peppard, Finn, & Benca, 2010), poor sleep is a common symptom of depression (Tsuno et al., 2005). It has therefore been argued that the relationship between sleep and depression is bi-directional (Riemann, Berger, & Voderholzer, 2001).

2.4.1 Sleep Disturbances as a Symptom of Depression

Between 50-90% of individuals with depression report some form of sleep disturbance (Tsuno et al., 2005). Chapter 1 emphasised the complexity of sleep and the different ways that it can be measured. Therefore, when considering the relationship between sleep and depression it is important to question which aspects of sleep are most disturbed. This section describes studies that have examined the relationship between depression and different types of sleep disturbance; including sleep duration, insomnia symptoms, subjective sleep quality and objective sleep disturbances.

Sleep Duration

The relationship between sleep duration and depression is not straight-forward. In a large epidemiological study of 24,000 Japanese citizens, Kaneita et al. (2006) found that those reporting an average of less than 6 hours or more than 8 hours of sleep were more likely to be depressed than those reporting between 6-8 hours. This suggests that a 'U-shaped'

relationship exists between sleep and depression. This has also been found to reflect the nature of the relationship between sleep duration and mortality, with both short and long sleep associated with poorer outcomes (Kripke, Garfinkel, Wingard, Klauber, & Marler, 2002; Kripke, Simons, Garfinkel, & Hammond, 1979; Tamakoshi & Ohno, 2004).

Subjective Sleep Quality and Insomnia

Poor subjective sleep quality is commonly reported amongst those with depression (Almeida & Pfaff, 2005; Mayers et al., 2009; Mayers, van Hooff, & Baldwin, 2003).

Subjective sleep complaints are important, since they are the primary way in which sleep problems are detected. Subjective reports of insomnia or hypersomnia symptoms are used in the diagnosis of depression (according to the DSM-5; American Psychiatric Association, 2013).

One of the earliest epidemiological studies exploring the relationship between sleep disturbance and psychiatric disorders was carried out by Ford and Kamerow (1989).

Healthy individuals, as well as those with insomnia and hypersomnia were interviewed at two time points between 1981 and 1985. Compared to 24.9% of the general population; 57.4% of those with insomnia and 64% of those with hypersomnia had a psychiatric illness. Insomnia was most common amongst those with depressive symptoms.

Hypersomnia (excessive daytime sleepiness) is also a commonly reported symptom in those with depression (Dauvilliers, Lopez, Ohayon, & Bayard, 2013; Detre et al., 1972).

Almeida and Pfaff (2005) found that 63% of older adults ($n = 1029$) visiting the general practice reported sleep complaints (difficulty falling asleep, restlessness / wakefulness during the night, early morning awakening). These individuals were 3.7 times more likely to be depressed than those without sleep complaints, after controlling for a number of additional risk factors.

However, an issue with exploring the relationship between subjective sleep quality and depression is that no single definition of 'subjective sleep quality' exists. A popular tool for measuring subjective sleep quality is the PSQI (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989), which has been used in a number of studies to explore the relationship between subjective sleep quality and depression (e.g. Franzen, Buysse, Rabinovitz, Pollock, & Lotrich, 2010; Huang, Carter, & Guo, 2004; Isaac & Greenwood, 2011; Kamysheva, Wertheim, Skouteris, Paxton, & Milgrom, 2009). However, the PSQI involves a number of subscales (such as duration of sleeping problems, sleep disturbances, sleep onset latency,

daytime dysfunction, sleep efficiency, sleep quality and use of sleep medication) which are combined to give a total score. While this is a well-validated measure, it does not tell us much about which aspects of sleep are most relevant to depression.

Mayers et al. (2003) found that individuals with depression reported poorer perceptions of sleep quality than non-depressed individuals, despite estimates of sleep disturbance being similar between groups. In a later study, individuals with depression were more likely to report poor subjective sleep satisfaction, rather than issues surrounding sleep quantity (e.g. total sleep time, Mayers et al., 2009). The opposite pattern was found in those with anxiety. This suggests that sleep disturbances in depression reflect general rather than specific concerns. Given this, it is possible that some of the sleep problems commonly reported in depression are a symptom of the negative thinking style that is characteristic of depression (Beck, 1967), rather than actual deficits. This highlights the need to consider the tools used to measure sleep in relation to depression.

2.4.2 Sleep Disturbance as a Predictor of Depression

As well as being a symptom of depression, there is strong evidence suggesting that certain aspects of sleep are associated with increased risk of developing depression (Chang et al., 1997; Szklo-Coxe et al., 2010). For example, in the study by Chang et al. (1997), insomnia symptoms in young men were found to be a significant predictor of depression status over 30 years later, when controlling for other risk factors. A review by Baglioni, Spiegelhalder, Lombardo and Riemann (2010) found that, out of twenty one longitudinal studies examining sleep, depression and anxiety; only two studies failed to demonstrate that symptoms of insomnia predicted an increased risk of future depression.

The findings of these studies are important, as they suggest that treating sleep disturbances could reduce the likelihood of an individual developing later depression. Despite this, to date, no large-scale studies of this nature have been carried out. Recent research has, however, found that treating insomnia in those with co-morbid depression and insomnia subsequently leads to improved depressive symptoms (Manber et al., 2008).

2.4.3 Objective Sleep and Depression

As highlighted in Chapter 1, sleep can be measured both subjectively and objectively. Although subjective sleep appears to be important to depression, studies using polysomnography (PSG) have explored whether those with depression also experience differences in their underlying sleep architecture.

Using PSG, the following objective sleep characteristics have been reported in depression: increased sleep onset latency, increased wake after sleep onset, poorer sleep efficiency, less slow-wave sleep, reduced REM latency and increased REM activity (Kupfer, 1984; Tsuno et al., 2005). A meta-analysis by Benca, Obermeyer, Thisted and Gillin (1992) concluded that although no single sleep variable shows absolute specificity for a particular psychiatric disorder, those with affective disorders differ most significantly and often from non-depressed individuals. The authors concluded that depression is likely to be accompanied by a combination of different sleep markers rather than one specific marker.

Assessing whether sleep disturbances in depression represent state or trait variables is important in elucidating the underlying pathophysiology of the disorder (Kupfer, 1984). If sleep disturbances are state variables it would suggest that they accompany an acute episode of depression and may provide a useful marker for diagnosing depression. On the other hand, if sleep disturbances are trait variables they may provide important vulnerability markers for those at increased risk of developing the disorder.

Kupfer and Ehlers (1989) proposed that trait variables ('type 1' variables) include reduced REM latency, decreased slow-wave sleep and decreased delta sleep ratio. On the other hand, state variables ('type 2' variables) include reduced REM latency, increased REM density² and reduced sleep efficiency. Notably, REM latency was argued to be both trait and state-like; as although it may shorten during a severe acute episode, it often does not normalise after remission. However more often than not, reduced REM latency is considered to be a trait marker of depression (Kupfer & Ehlers, 1989).

In support of this theory, Thase (1998) examined depressed patients before and after 16 weeks of cognitive behavioural therapy (CBT) in order to evaluate the extent to which sleep abnormalities would reverse after treatment or remain stable. After treatment, abnormal type 1 variables (reduced REM latency; decreased delta sleep ratio and reduced slow-wave sleep percentage) remained stable, whereas type 2 variables (increased REM density, reduced sleep efficiency) improved, though some experienced persistent sleep abnormalities into remission. The authors suggested that patients whose sleep normalises

² REM density refers to the relative number of rapid eye movements that are present during this stage of sleep

after an episode of depression (in terms of state variables) are less likely to relapse than those who continue to exhibit disturbed state sleep abnormalities.

Reduced REM latency and amounts of slow-wave sleep were also reported to remain stable into remission in a study of elderly depressed patients (Lee et al., 1993). However, in a meta-analysis of 56 studies exploring EEG markers and depression, Pillai, Kalmbach and Ciesla (2011) concluded only REM density and slow-wave abnormalities are likely to persist into remission.

A problem with classifying sleep disturbances as state/trait-like according to their stability or recovery after remission is that it runs the risk of mistaking a variable as a trait when in fact it may represent a biological 'scar' from the depressive episode itself. One way of clarifying the relationship is to use longitudinal and familial studies, which allow for the investigation of whether trait-like vulnerability markers exist prior to depression or in high-risk probands. In line with this, Giles, Etzel and Biggs (1990) found similar sleep abnormalities in first-degree relatives of depressed patients with reduced REM latency, but who had no personal history of depression. These patients also showed similar negative cognitions relating beliefs and attributional style. Reduced REM latency in probands was associated with a two-fold increase in the probability of lifetime sleep abnormalities.

In a review, Tsuno et al. (2005) concluded that reduced REM latency is the most significant sleep disturbance found in depressed patients. Furthermore, depressed patients who exhibit reduced REM latency have an increased chance of relapse and more rapid relapse than those with normal REM latency (Giles, Jarrett, Roffwarg, & Rush, 1987). Given the research surrounding REM sleep and depression, it is suggested that REM abnormalities (particularly REM latency) represent an endophenotype (a bridge between underlying genes and overt behavioural abnormalities) for depression (Modell & Lauer, 2007). Individuals displaying REM sleep abnormalities may be at greater risk of developing depression.

It is also possible that REM sleep disturbances in depression may serve a mood regulatory function (Cartwright & Lloyd, 1994; Walker & van der Helm, 2009). According to Cartwright and Lloyd (1994), REM sleep changes associated with depression are adaptive, rather than dysfunctional. Several studies have found a number of favourable outcomes to be associated with reduced REM latency in depressed patients. For example, in one study,

exhibiting this marker predicted a better response to antidepressant medication (Rush et al., 1989), while another study found that untreated depressed patients with reduced REM latency had a higher recovery rate after one year than untreated patients with normal REM latency (Cartwright, Kravitz, Eastman, & Wood, 1991).

Cartwright and Lloyd (1994) found that reduced REM latency in depression was associated with greater intensity of the first dream compared to later dreams, and that individuals who exhibited this marker, also showed greatest reduction in depressive scores one year later. The authors proposed that reduced REM latency, accompanied by intense first dreams, represents a compensatory mood regulation mechanism that may occur in response to negative affect. In contrast, Walker and Van der Helm (2009) propose that it is the process of REM sleep itself, rather than dreaming (which they consider a functionless by-product of REM) that is important in emotional regulation. In their 'sleep to remember, sleep to forget' hypothesis, the authors state that over time we remember the content of memories, but the emotional, affective tone diminishes. They argue that REM sleep provides the optimal brain state in which this emotional regulation can occur. Activation of the amygdala and hippocampus reactivate previously acquired memories, whilst the theta oscillations offer large scale network cooperation allows for the integration and 'working through' of emotional memories (Walker & van der Helm, 2009).

Overall, there appears to be some evidence that REM sleep and dreaming relate to emotional regulation, which may account for the poor emotional regulation and abnormal REM sleep characteristic of depression. While it is beyond the scope of this thesis to test particular theoretical underpinnings of the relationship between sleep and depression, this research highlights the importance of measuring sleep architecture when examining the relationship between sleep and depression. The only way that this is possible is to examine sleep using polysomnography, yet little research to date has used this method when examining the relationship between sleep and postpartum depression. A review of the research is provided in Chapter 3.

2.5 Overview of Chapter 2

In summary, it is evident that there is a strong bi-directional relationship between sleep disturbance and depression. However, the nature of research design (e.g. longitudinal versus cross-sectional) is clearly an important factor in depicting the nature of this relationship. Recognising how patients with depression describe their sleep is important for identifying a possible depressive episode. On the other hand, the fact that sleep

disturbances present a risk factor for the development of depression suggests that managing sleep problems could potentially reduce the risk of depression.

There are many different ways in which we can assess sleep, and more research needs to look at how these factors are inter-related. Depression appears to be associated with a number of characteristic disturbances in sleep architecture including reduced REM latency and decreased slow-wave sleep. Reduced REM latency appears to be a key marker of depression, which may predict future risk of developing depression. However, in everyday practice, undergoing PSG is often not an option, as it is costly and time-consuming. Therefore subjective reports of sleep are also important. As described in Chapter 1, more research is needed to examine whether subjective perceptions tell us anything about the individual's underlying sleep architecture. This question will be addressed later in the thesis. Having examined the relationship between sleep and major depressive disorder, the next chapter provides a systematic review of the research that has specifically examined the relationship between sleep and postpartum depression.

CHAPTER 3: SLEEP AND POSTPARTUM DEPRESSION: A SYSTEMATIC REVIEW

3.1 Overview of Chapter

The previous chapter emphasised that the relationship between poor sleep and depression is bi-directional (e.g. Sivertsen et al., 2012). Poor sleep is a common symptom of depression, with up to 90% patients reporting sleep complaints (Tsuno et al., 2005). Furthermore, it is a well-established finding that sleep disturbances may increase the risk of developing depression (Baglioni & Riemann, 2012; Chang et al., 1997; Ford & Kamerow, 1989; Szklo-Coxe et al., 2010). However, much less is known about how sleep problems are related to the development of postpartum depression (PPD). This is despite the fact that both sleep problems and depression are common during the perinatal period (Gavin et al., 2005; Signal et al., 2007). This chapter provides a systematic review of the literature to date that has examined the relationship between sleep and postpartum depression.

3.2 Introduction

Karacan, Williams, Hirsch, McCaully and Heine (1969) were the first study to use electroencephalography (EEG) to report that women who were vulnerable to PPD differed in their sleep architecture. Since then, several studies have confirmed a relationship between poor sleep and PPD (e.g. Bei, Milgrom, Ericksen, & Trinder, 2010; Dorheim, Bondevik, Eberhard-Gran, & Bjorvatn, 2009; Swain, Ohara, Starr, & Gorman, 1997). However, the exact nature of this relationship remains unclear. This is in part due to methodological issues with the research that has been carried out, including over-reliance of cross-sectional designs, varying measures of sleep and depression, and failing to control for depression at baseline. As a result, studies to date have not been able to successfully delineate which sleep variables are most predictive of PPD. Each of these issues are examined within this review.

The Directional Nature of the Relationship between Sleep Patterns and PPD

One key issue that remains unclear is whether the relationship between poor sleep and PPD is also bi-directional; do sleep disturbances precede the onset of PPD, and possibly present a significant risk factor in its development; or are they just a symptom of PPD? Whilst some research has attempted to address these issues (e.g. Marques et al., 2011;

Wolfson, Crowley, Anwer, & Bassett, 2003) there is no clear consensus between studies as to the exact way that sleep and PPD are related. Cross-sectional studies are useful in helping us to understand how sleep is reported in women experiencing PPD, so that we can improve detection. For example, women experiencing PPD may be reluctant to talk openly about their feelings due to the stigma surrounding perinatal mental illness (Bilszta, Ericksen, Buist, & Milgrom, 2010) and discussing sleep problems may provide an entry point to talk about underlying mood. However, it is important to carry out longitudinal research to tease out the precise nature and direction of the relationship between sleep disturbance and the development of PPD, so that we can identify and provide support to 'at risk' women.

Which Aspect of Sleep is Most Relevant to PPD?

A second key issue that remains unclear is which specific aspects of sleep are most strongly related to PPD. Research within this area has tended to focus on subjective measures of sleep (e.g. Dorheim, Bondevik, Eberhard-Gran, & Bjorvatn, 2009; Tsai & Thomas, 2012; Wilkie & Shapiro, 1992; Wolfson, Crowley, Anwer, & Bassett, 2003), while the majority of those using an objective measure have used actigraphy rather than polysomnography (PSG) (Bei, Milgrom, Ericksen, & Trinder, 2010; Dorheim, Bondevik, Eberhard-Gran, & Bjorvatn, 2009; Goyal, Gay, & Lee, 2009; Lee & Kimble, 2009; Posmontier, 2008). PSG is important in order to understand how women's sleep architecture (e.g. amount of slow-wave sleep) relates to PPD. As described in Chapter 1, the subjective / objective distinction is important, given that previous research has suggested that they may not necessarily be measuring the same constructs (e.g. Rotenberg, Indursky, Kayumov, Sirota, & Melamed, 2000).

3.3 The Current Review

This review critically evaluates the research that has addressed the relationship between sleep and PPD, with a focus on how the two questions described above have been addressed. Additionally, it carefully examines how the nature of the experimental designs and measures used in the research to date affects our understanding of this relationship.

3.3.1 Excluded Literature

While some studies have examined the relationship between sleep and antepartum depression (e.g. Kamysheva, Skouteris, Wertheim, Paxton, & Milgrom, 2010), the focus is specifically related to postpartum depression. It is also acknowledged that infant sleep

may play a role in PPD (Gress et al., 2010; Hiscock & Wake, 2001; Karraker & Young, 2007; Orhon, Ulukol, & Soykan, 1992). Research in this area suggests that poor infant sleep could lead to chronic maternal sleep deprivation, which could trigger depression (Ross, Murray, & Steiner, 2005). However, infant sleep is not specifically examined within this thesis, since it is difficult to separate the ‘chicken from the egg’ (Ross et al., 2005). Ross et al. (2005) describe several pathways in which poor infant sleep and PPD may be related. For example:

- Children of depressed mothers may be more prone to sleep problems as a response to a lack of maternal responsiveness (Field, 2010).
- PPD is related to unfavourable parenting practices that may result in poorer infant sleep (e.g. not adopting the preferred infant sleep position, Zajicek-Farber, 2008).
- Children of depressed mothers may be showing early symptoms of genetically transmitted psychiatric or sleep problems.
- The relationship may be indirect; poor infant sleep may also affect the sleep of other family members which could disrupt maternal sleep.

Although specific infant sleep factors are not examined in this thesis, it is important to bear in mind that a vicious cycle is likely to exist between poor sleep and PPD. Even if infant sleep (or a lack thereof) is not the cause of PPD, common symptoms of PPD, including reduced maternal sensitivity and poor maternal sleep (Field, 2010; Goyal, Gay, & Lee, 2007), may serve to exacerbate infant sleep problems and subsequently increase maternal depressive symptoms (Paulson, Dauber, & Leiferman, 2006).

Furthermore, this review did not include studies solely focusing on underlying mechanisms of the relationship between sleep and PPD (e.g. circadian markers). However, research of this nature will be discussed later in the thesis.

3.3.2 Method

To identify relevant articles the terms ‘sleep’ and ‘depress*³’ and ‘postpartum’ or ‘postnatal’ were entered into a series of relevant databases (see Figure 4 for an overview

³ This is a truncated term

of the search strategy). Studies had to report original empirical data on the relationship between maternal sleep and postpartum depressive symptoms. At least one measure of maternal sleep had to be included in the study, and this had to be analysed in relation to depression or mood, so that the relationship between sleep and PPD could be examined. A summary of the abbreviations used throughout this review is presented in Table 4

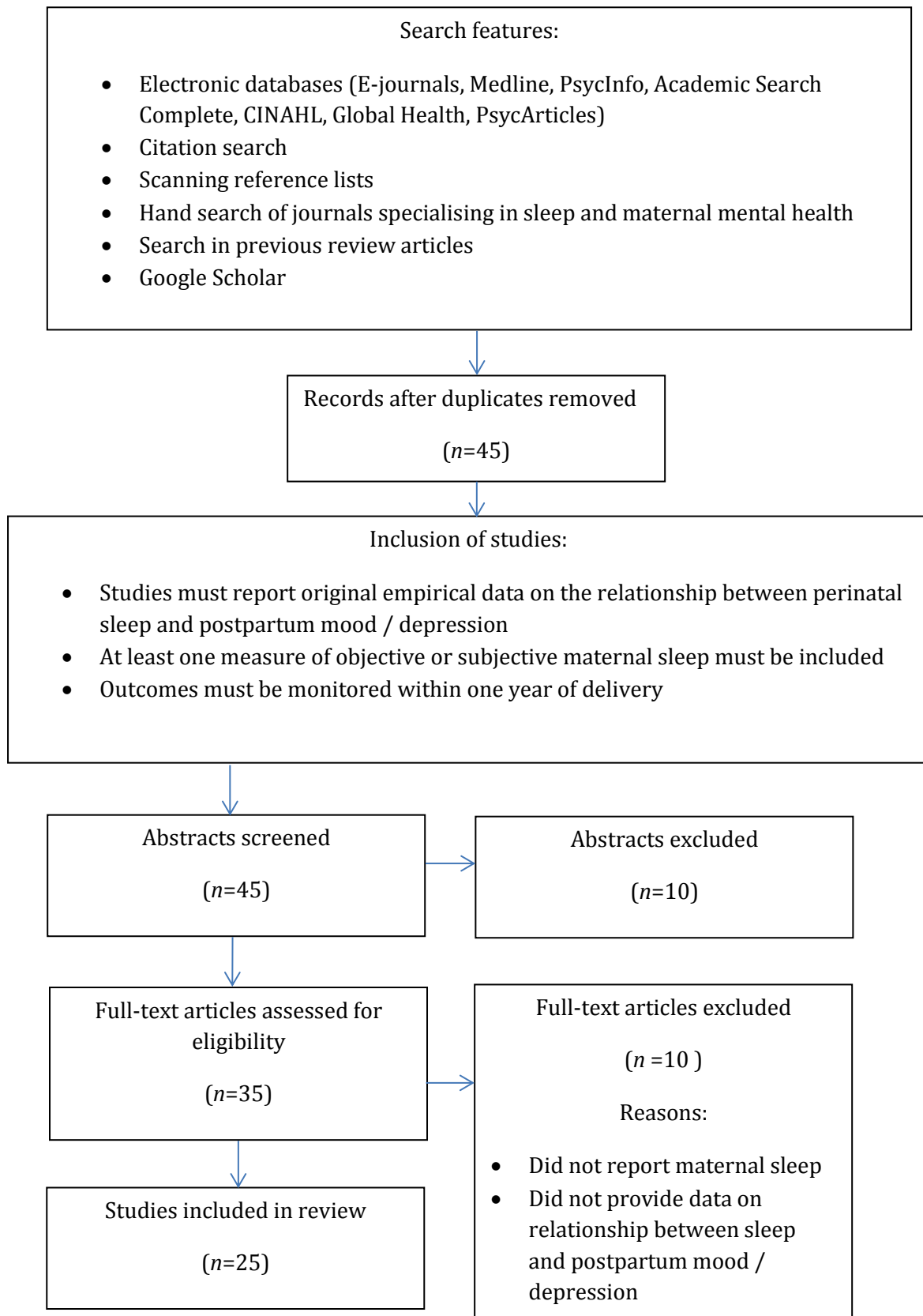


Figure 4. Flow diagram for the search strategy and inclusion of studies

Table 4. Description of Abbreviations for Sleep Terminology, Sleep Scales and Depression Scales

Sleep terminology	Description
NREM	Non rapid-eye movement sleep; stages 1, 2, 3 & 4
REM	Rapid eye movement sleep
REML	REM latency; time from first epoch of sleep to the first period of REM sleep
SE	Sleep efficiency – total time spent asleep (TST) / total time spent in bed
SoL	Sleep onset latency; time from lights out until first stage of sleep
SQ	Sleep quality
SWS	Slow-wave sleep / deep sleep. Stages 3 & 4 (N3).
TST	Total sleep time
WASO	Wake after sleep onset; amount of time spent awake after sleep onset.

Sleep scales	Author	Full name
ESS	Johns (1991)	Epworth Sleepiness Scale
GSDS	Lee (1992)	General Sleep Disturbance Scale
ISI	Bastien et al. (2001)	Insomnia Severity Index
PSQI	Buysse et al. (1989)	Pittsburgh Sleep Quality Index

Depression scales	Author	Full name
BDI	Beck et al. (1961)	Beck Depression Inventory
CES-D	Radloff (1977)	Centre for Epidemiological Studies Depression Scale
DASS	Lovibond & Lovibond (1995)	Depression Anxiety Stress Scale
EPDS	Cox et al. (1987)	Edinburgh Postnatal Depression Scale
HRDS	Hamilton (1960)	Hamilton Rating for Depression Scale
MINI	Sheehan et al. (1998)	Mini International Neuropsychiatric Interview
PDSS	Beck & Gable (2002)	Postpartum Depression Screening Scale
PSS	Cohen et al. (1983)	Perceived Stress Scale
PANAS	Watson et al. (1988)	Positive and Negative Affect Schedule
POMS	McNair et al. (1992)	Profile of Mood States
RDC	Spitzer et al. (1978)	Research Diagnostic Criteria

3.4 Comment on Literature Search and Structure of the Review

From Figure 4 it is apparent how few studies have examined the relationship between sleep and PPD. This is somewhat surprising given the richness of the data available regarding the relationship between sleep and major depression. This may be due to the fact that PPD is not considered a distinct diagnosis to major depression in the DSM-5 (American Psychiatric Association, 2013). Additionally, it may be more difficult to recruit perinatal women for sleep research due to the commitment that this often entails and the other commitments that these women may have (indeed, this will be discussed later in the thesis in relation to the researcher's own difficulties with recruiting this sample).

This review is structured to highlight the key issues surrounding the direction of the relationship between poor sleep and PPD, and the way in which sleep is measured. Therefore the subsequent sections are divided into cross-sectional and longitudinal studies and are further partitioned according to whether they used an objective versus subjective measure of sleep. Each of the tables presented in this chapter are formatted in chronological order, to show how research in this area has developed over time.

3.5 Cross-Sectional Designs

The use of cross-sectional designs is common within this research area. The perinatal period can be a busy and stressful time for women; particularly during late pregnancy and the early postpartum period. Since longitudinal studies typically involve more commitment from participants, cross-sectional designs may be favoured. Although these studies cannot provide information on cause and effect, they have provided some insight into the nature of the relationship between sleep and PPD. They also provide useful information regarding the recognition of PPD symptoms.

3.5.1 Subjective Sleep Studies (Cross-Sectional)

Table 5 provides an overview of all of the subjective cross-sectional studies that have examined sleep and PPD ($n = 8$). One thing that is particularly apparent from this table is that the study designs vary significantly. Amongst these studies, time-points vary from 13 days postpartum to 12 months postpartum, and various measures are used to measure both sleep and PPD. Three different measures of depression are used, and cut-off scores vary. Only four studies used a validated measure of PPD (Da Costa, Dritsa, Rippen, Lowensteyn, & Khalifé, 2006; Dorheim et al., 2009; Hiscock & Wake, 2001; Swanson, Pickett, Flynn, & Armitage, 2011). A more detailed

discussion of PPD measures will be discussed later in the review. Remarkably though, despite the varied methodology, all of the studies reported a relationship between sleep and PPD.

A common theme from these studies is that subjective sleep *quality* appears to be particularly important in relation to PPD, perhaps more so than sleep *quantity* (Da Costa, Dritsa, Rippen, Lowensteyn, & Khalifé, 2006; Dorheim et al., 2009; Groer & Morgan, 2007; Hiscock & Wake, 2001; Huang, Carter, & Guo, 2004; Swanson, Pickett, Flynn, & Armitage, 2011). A particularly interesting finding by Hiscock and Wake (2001) was that mothers reporting good subjective sleep quality, despite reporting poor infant sleep, were less likely to be depressed than those reporting poor sleep quality. We may expect that poor infant sleep would result in the mother also reporting poor sleep, however this finding emphasises the importance of the way in which the mother perceives her sleep.

Table 5. Cross-sectional Studies Assessing Subjective Sleep and PPD

Authors, Publication year	Sample / time-points	Measure of sleep	Measure of depression	Outcome
Hiscock & Wake (2001)	738 women between 6-12 months PP	Survey on infant sleep over past 2 weeks and whether mother considered it a problem & severity scale. One question on maternal sleep quality.	EPDS (analysed using various cut-offs)	Maternal report of infant sleep problem was a significant predictor of depression when controlling for other factors. Mothers reporting good sleep despite infant sleep problems not more likely to report depression.
Huang, Carter, & Guo (2004)	163 first-time mothers between 13-20 days PP	PSQI (analysed each subscale)	CES-D cut-off ≥ 16	Depressed mothers reported poor sleep quality, reduced sleep efficiency, more sleep disturbances and more daytime dysfunction than non-depressed mothers.
Da Costa, Dritsa, Rippen, Lowensteyn & Khalifé (2006)	78 women between 4-28 weeks PP	PSQI (global score only)	EPDS cut-off ≥ 10 Medical Outcomes Study Short Form-36 HRQoL	Poor sleep was associated with worse mental health status after controlling for depression severity.
Groer & Morgan (2007)	25 women between 4-6 weeks PP	ESS	POMS cut-off ≥ 21	Depressed mothers showed more daytime sleepiness and were twice as fatigued as non-depressed mothers.
Dorheim, Bondevik, Eberhard-Gran, & Bjorvatn, (2009)	2380 women between 6-20 weeks PP	PSQI (analysed each subscale)	EPDS cut-off ≥ 10	Poor sleep was associated with PPD when controlling for other risk factors. Poor subjective sleep quality was the factor most strongly related to PPD.

Swanson, Pickett, Flynn, & Armitage (2011)	114 pregnant and 148 PP women (up to 6 months)- all receiving outpatient psychiatric treatment	ISI	EPDS cut-off ≥ 12	Correlation between EPDS and ISI in PP women. Difficulty falling asleep was the only regression variable to predict EPDS and PSWQ.
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Note. PP=postpartum. See Table 4 for list of abbreviations.

It is important to consider the factors that may differentiate between the mothers reporting good versus poor subjective sleep quality, when both are reporting poor infant sleep. This could relate to the mother's expectation regarding infant sleep and what she considers 'normal' or manageable. Another possibility is that women with PPD have a tendency to report poorer perceptions of sleep quality. Research investigating major depression has suggested that depression is related to poorer subjective sleep quality, even when the perceived amount of sleep is satisfactory (Mayers et al., 2009). It is possible that women who were depressed reported poorer sleep quality due to the general negative bias that often accompanies depression (Beck, 1964); or because they felt less able to cope with the insufficient sleep caused by the infant.

Alternatively, women reporting poor subjective sleep quality may have been receiving less restorative sleep, such as less slow-wave sleep which may lead them to perceive their sleep quality as poor. Research described in Chapter 2 suggests that this may indeed be the case. Akerstedt et al. (1997) found that amount of slow-wave sleep related to subjective perceptions of sleep quality. However, this could not be examined in the aforementioned subjective perinatal sleep studies as none of these studies included PSG to objectively measure sleep architecture. This highlights the need to examine the relationship between objective and subjective measures of sleep, in order to understand the factors that affect how women perceive their sleep quality.

The most commonly used subjective measure of sleep within these studies is the PSQI (Buysse et al., 1989). Groer and Morgan (2007) found that depressed mothers were more likely to report daytime sleepiness (measured through the Epworth Sleepiness Scale, ESS), and were twice as likely to feel fatigued as non-depressed mothers. However, as described in Chapter 1, feelings of fatigue and sleepiness do not necessarily relate to quantity of sleep (Lavidor et al., 2003; Pilcher et al., 1997), and therefore benefit from being examined as independent factors. The relationship between sleep and fatigue is described later in the chapter.

Swanson, Pickett, Flynn and Armitage (2011) focused on reports of insomnia in pregnant and postpartum women seeking outpatient psychiatric treatment. Correlations were found between insomnia and depression scores. However only one item - assessing difficulty falling asleep - was found to be a significant predictor of depression scores. This suggests that symptoms of insomnia are apparent among pregnant women and may increase the likelihood of developing PPD. However, it could be argued that since the Insomnia Severity Index (ISI) has not been validated for use in a pregnant sample, it may lack validity since symptoms of insomnia may

overlap with general aspects of early motherhood (e.g. waking in the night to feed the infant, waking early in the morning).

Only two of the studies using the PSQI analysed each of the subscales separately (Dorheim et al., 2009; Huang et al., 2004). Although the PSQI is a highly validated measure (Buysse et al., 1989), as described in Chapter 1, it provides a global score that reflects many aspects of sleep quality (including sleep quality, sleep latency, sleep duration, sleep efficiency, use of sleep medication and daytime functioning). While a global score can be useful in order to gain a comprehensive understanding of the relationship between sleep and PPD, we first need to examine individual aspects of sleep, given that it may only be specific aspects of sleep that are able to predict PPD.

One study that did explore the individual aspects of the PSQI was carried out by Dorheim, Bondevik, Eberhard-Gran, and Bjorvatn (2009). Questionnaires, including the PSQI, were sent to 2380 women (a very large sample size for research in this field) at seven weeks postpartum, to examine the prevalence of sleep disturbances and depression. Poor sleep was associated with depression when controlling for other significant risk factors (including psychosocial factors and individual disposition). The PSQI subscales most strongly associated with depression were sleep disturbances and subjective sleep quality. This suggests that other aspects of poor sleep (such as sleep efficiency and daytime dysfunction) are not poorer in women with PPD, highlighting the need to tease apart specific sleep factors in relation to PPD. In contrast, Huang, Carter, and Guo (2004) found that women classified as depressed between the 13th and 20th postpartum day reported significantly poorer sleep on a number of PSQI subscales, including sleep quality, sleep efficiency, sleep disturbances and daytime dysfunction compared to non-depressed women. Therefore, there appears to be conflicting evidence as to which aspects of sleep are most relevant to PPD.

While analysing individual subscales of the PSQI is preferable, even some of the individual subscale scores are aggregate measures of several forms of sleep disturbance. For example, the 'sleep disturbance' subscale covers disturbances including difficulty falling asleep, middle of the night / early morning waking, having to get up to use the bathroom, coughing / snoring loudly, feeling too hot / cold, having bad dreams, having pain and breathing uncomfortably. These factors cover a range of sleep-related disturbances; some of which may be common in this period (such as waking up early to feed the baby, and pain from delivery), whilst others may suggest the presence of a sleep-disorder, such as sleep apnoea or insomnia. A further limitation of the PSQI is that it refers to sleep over the past month. Since women's sleep is continually changing throughout pregnancy and in the early postpartum period (as described in

Chapter 1), looking at specific time-points is useful. Furthermore, as previously noted, women may have been reporting poor sleep due to the negative thinking associated with depression (Beck, 1964). Again, this highlights the need for objective measures of sleep, to assess the extent to which poor sleep in women with PPD is largely subjective, or whether their sleep quality or quantity actually differ in a particular way to women without PPD. This is explored in the next section.

Despite the varied methodologies used in these cross-sectional studies, each of them reported an association between poor sleep and PPD. However, a limitation of these studies is that none of them measured sleep prospectively (e.g. using a sleep diary), assessing the specific factors of sleep that are most relevant to depression. Additionally, due to the nature of cross-sectional research, they do not provide information about whether sleep disturbances were a cause or consequence of PPD.

3.5.2 Objective Sleep Studies (Cross-Sectional)

The objective sleep studies are presented in Table 6. A benefit of using objective measures is that they can provide a more accurate representation of sleep. Objective sleep can refer to the use of actigraphy or PSG, both of which were discussed in Chapter 1. While both measures provide information on quantitative aspects of sleep, such as total sleep time, sleep efficiency and number of awakenings, PSG also provides information on sleep architecture. This is useful in order to assess whether abnormal sleep architecture (such as decreased SWS) accompanies subjective reports of poor sleep in women with PPD, or whether only one aspect is relevant.

A benefit of actigraphy, however, is that it is cheaper to use and requires less technical expertise than PSG. Furthermore, actigraphy can provide more information about typical sleep patterns over time, since it is generally worn for a period of at least 7 days (Morgenthaler et al., 2007). In contrast, PSG is normally worn for 1-3 nights, depending on whether it is ambulatory or carried out within a laboratory (Agnew, Webb, & Williams, 1966; Kushida et al., 2005; Sharpley, Solomon, & Cowen, 1988). Therefore, PSG provides rich, in-depth information relating to sleep over a short period, whilst actigraphy may be better for assessing more general sleep patterns. While both of these methods are useful for studying sleep in perinatal women, the majority of studies looking at objective sleep and PPD have used actigraphy.

Table 6. Cross-sectional Studies Assessing Objective Sleep and PPD

Authors, Publication year	Sample / time-points	Measure of sleep	Measure of depression	Outcome
Godfroid, Hubain, Dramaix, & Linkowski (1997)	28 women (8 with PPD up to 6 months PP; 8 with depression & history of PPD, 8 with depression and no history of PPD)	3 nights of PSG in sleep laboratory (one night used for analysis)	RDC	Those with PPD had shorter stage 1 sleep and longer stage 4. No other significant between group differences.
Posmontier (2008)	23 women with PPD 23 women without PPD Between 6-26 weeks PP	7 nights of actigraphy and daily activity log	MINI & PDSS	Women with PPD had more WASO, lower sleep efficiency (SE) and longer SoL. Sleep quality score (formed of WASO, SoL & SE) predicted PPD severity.
Lee & Kimble (2009)	20 first-time mothers with low birth weight hospitalised infants 2 weeks PP	2 days of actigraphy & sleep diary GSDS	EPDS Medical outcomes short form HRQoL	No significant relationship between night-time TST and depressive symptoms.
Dorheim, Bondevik, Eberhard-Gran & Bjorvatn (2009)	21 women with PPD 21 women without PPD Around 9 weeks PP	PSQI Daily Sleep diary 2 weeks of actigraphy	EPDS cut-off ≥ 10	Those with depression had significantly higher global PSQI score & on 4 sub-scales (subjective sleep quality, daytime dysfunction, SoL, TST). No associations between PPD and sleep diary / actigraphy, except more daytime dysfunction and lower day / night activity ratio

Tsai & Thomas (2012)	22 first-time mothers Up to 3 weeks PP	7 days of actigraphy followed by GDSD	EPDS cut-off ≥ 13	No women met PPD criteria. Night-to-night sleep variability (actigraphy) and early awakening (GDSD) significantly related to PPD scores. No other significant correlations.
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Note. PP=postpartum. See Table 4 for list of abbreviations.

Only one study has used PSG to examine sleep in women currently presented with PPD (Godfroid et al., 1997). Godfroid et al. (1997) compared the sleep of 8 women currently experiencing PPD, to 8 women with depression and a history of PPD, and 8 women with depression and no history of PPD. All women spent three nights in a sleep laboratory undergoing PSG. Surprisingly, results showed that those with PPD has significantly less stage 1 sleep (light sleep) and increased stage 4 sleep (deep sleep); indicative of better sleep among the mothers with PPD. However, it is difficult to draw any firm conclusions from this study because it is unlikely that it provided an accurate representation of typical sleep. Since postpartum women experience significant sleep disturbances (Lee, Zaffke, & McEnany, 2000), when being given the opportunity to have a period of undisturbed sleep, away from their infant, it is likely that their sleep may be of better quality. Indeed, it is a well-established finding that then when sleep deprived individuals are given a night of 'recovery' sleep, they experience a rebound of deep sleep (Borbély & Wirz-Justice, 1982; De Gennaro et al., 2010). Furthermore, in this study all groups were experiencing depression; there was no control group of postpartum women without PPD, and the sample was small (8 women in each group).

The remaining objective cross-sectional studies used actigraphy as a measure of sleep. One of these studies was carried out on mothers with low birth weight infants whose infants were not sleeping at home (Lee & Kimble, 2009). Although this study is interesting as it was able to investigate the relationship between sleep and PPD without infant sleep as a confounding variable, these findings cannot be generalised to the majority of new mothers. In this case, no significant relationship was found between sleep and depression scores, raising the possibility that infant sleep is an important mediator in the relationship between sleep and PPD. However, actigraphy was only used for 2 days, which is less than the recommend period (Kushida et al., 2005).

Despite similarities in experimental design, two further studies using actigraphy reported somewhat conflicting findings (Dorheim et al., 2009; Posmontier, 2008). While Posmontier (2008) found that women with PPD had more waking after sleep onset, lower sleep efficiency and longer sleep onset latency than women without PPD, these differences were not found by Dorheim et al. (2009). However, Dorheim et al. also included subjective measures of sleep (PSQI and sleep diaries). While no significant differences were found between women with and without PPD using actigraphy or sleep diaries (both prospective measures), a significant difference was found when using the PSQI (a retrospective measure). This is an important finding, as it highlights the need to include various measures of sleep, as not all measures are

similarly related to PPD. These findings are similar to Wolfson et al. (2003), who found that postpartum sleep diary responses were not related to PPD symptoms.

There are several possible explanations for the different findings between sleep measures. Dorheim et al. (2009) noted that there was a time-lag between completing both types of measures; leaving the possibility that women's sleep patterns changed during this time. Alternatively, the findings may suggest that women were not very accurate in their perception of sleep when compared to actigraphy. However, the authors did not comment on the specific relationship between these measures. Another point to highlight is that the PSQI refers to sleep over the past month. Following from an earlier point, it is possible that women with PPD had a more negative global outlook on their sleep over the past month, due to the negative distortions which are common in depression (Beck, 1964).

3.5.3 Overview of Cross-Sectional Studies

The majority of cross-sectional studies within this sample used a subjective measure of sleep. The small number of objective studies varied considerably in their methodology, making it difficult to compare and summate findings. An interesting observation is that whilst all of the *subjective* sleep studies reported a relationship between poor sleep and PPD, only one study found poorer *objective* sleep in women with PPD. This raises the following questions:

- Are subjective and objective measures of sleep tapping into the same constructs?
- Is poor subjective sleep in women with PPD simply an index of their depressive mood?

Furthermore, the findings from Dorheim et al.'s (2009) study showed that differences exist even within subjective measures of sleep. Taken together, it is important that specific subjective and objective measures are combined in order to fully investigate the relationship between sleep and PPD.

3.6 Longitudinal Designs

Longitudinal studies are necessary for understanding the prospective relationship between sleep and PPD. These types of studies have suggested that poor sleep presents a risk factor for the development of major depression (Baglioni & Riemann, 2012; Chang et al., 1997; Ford & Kamerow, 1989; Szklo-Coxe et al., 2010) but less is known about whether similar relationships exist with PPD. As described in Chapter 1, although diagnostically PPD is not considered a separate condition, it is argued that distinct features of the postpartum period (such as hormonal changes and infant care) warrant its investigation as a separate entity. The

following sections describe longitudinal research that has used either subjective or objective sleep measures.

3.6.1 Subjective Sleep Studies (Longitudinal)

Table 7 presents the studies that have used a longitudinal design to measure the relationship between subjective sleep and PPD. Out of these nine studies, only six provided information on the prospective relationship between sleep prior to birth and the development of PPD (Flesher, 2009; Marques et al., 2011; Okun et al., 2011; Okun, Hanusa, Hall, & Wisner, 2009; Wilkie & Shapiro, 1992; Wolfson et al., 2003). The remaining studies looked at the relationship between sleep and PPD at several time-points in the postpartum period, but carried out cross-sectional rather than longitudinal analyses at each time-point, or summated scores over time (Dennis & Ross, 2005; Goyal, Gay, & Lee, 2007; Swain et al., 1997). Each of studies reported a significant relationship between disturbed night-time sleep and PPD symptoms, both during late pregnancy (Goyal et al., 2007) and into the postpartum period (Dennis & Ross, 2005; Goyal, Gay, & Lee, 2007; Swain et al., 1997).

The first study to examine the prospective relationship between subjective sleep and PPD was carried out by Wilkie and Shapiro (1992), who found that greater sleep disturbance in the third trimester of pregnancy (characterised by difficulty getting to sleep, broken sleep and early morning waking) related to increased symptoms of postpartum blues. This suggested that sleep in late pregnancy may be important in determining postpartum mood. However, whilst postpartum blues is related to the onset of PPD (Beck, 2001), depression in the later postpartum period was not examined. Another limitation of this study is that it did not control for depression at baseline. Therefore it is possible that poor sleep in women during pregnancy may have been a symptom of pre-existing depression, rather than a causal factor.

Table 7. Longitudinal Studies Assessing the Relationship between Subjective Sleep and PPD

Authors, Publication year	Sample / time-points	Measure of sleep	Measure of depression	Outcome
Wilkie & Shapiro (1992)	63 married women from 36 weeks -10 days after birth	Rating of sleep in late pregnancy, and asked about sleep in late pregnancy compared to pre-conception. 10-day postpartum sleep diary.	Stein Questionnaire VAS scale mood state	Higher postpartum blues score between 3-5 days for those who had night-time labour. After birth, only subjective sleep quality related to mood; but weakest at time when blues highest. Greater sleep disturbance in 3 rd T related to higher blues ratings in PP
Swain, Ohara, Starr, & Gorman (1997)	30 first-time mothers in first 3 weeks PP 28 non-PP mothers	PSQI	VAS daily mood scale for 21 days	Loss of night-time sleep related to higher levels of negative mood in 1 st week PP.
Wolfson, Crowley, Anwer, & Bassett (2003)	38 first-time mothers followed from 3 rd T through to 12-15mths PP Screened for depression at baseline.	Mother's sleep-wake diary 24 hour sleep-wake chart Completed for 7 days at 4 time-points from 3 rd T-PP	CES-D cut-off ≥ 16	No difference between sleep in depressed and non-depressed mothers at 2-4 weeks. PPD at 2-4 weeks related to later rise times, longer naps and longer TST in 3 rd T.
Dennis & Ross (2005)	505 women 4 weeks PP Screened for depression at baseline.	8 questions relating to infant sleep and impact on mother; at 4 & 8 weeks PP	EPDS cut-off >12	Women with PPD significantly more likely to: report baby cried often, be woken more often, have <6 hours sleep and report poorer sleep, than women without PPD.

Goyal, Gay, & Lee (2007)	124 first-time mothers followed from 3 rd T through to 3mths PP	GSDS	CES-D cut-off ≥ 16	Sleep disturbances related to PPD at 3 rd T and 3mths PP.
Okun, Hanusa, Hall, & Wisner (2009)	51 women with a history of PPD followed by 3 rd T through to 20 weeks PP Screened for depression at baseline.	PSQI	21_HRDS Clinical diagnosis if scored ≥ 5 on 2+ occasions	PSQI scores did not predict recurrence but predicted timing of recurrence. Early recurrers (<4 weeks PP) had better sleep quality in 3 rd T; late recurrers had poorer SQ in 3 rd T.
Flesher (2009)	38 women at 3 rd T and 1mth PP	PSQI ESS	CES-D Perceived Stress Scale Zung Anxiety Scale	Women with high PPD scores had significantly worse PSQI and daytime sleepiness. Daytime somnolence in 3 rd T and PP related to PPD
Marques et al. (2011)	581 women at 3 rd T and 3mths PP Screened for depression at baseline.	Questions relating to history of insomnia and current symptoms.	Diagnostic Interview for Genetic Studies POMS BDI-II	No association between insomnia and PPD symptoms when controlling for negative / positive affect and history of insomnia
Okun et al. (2011)	56 women with history of PPD at 8 time-points in first 17 weeks PP. Screened for depression at baseline	PSQI	21_HRDS	PSQI scores during first 17 weeks related to significantly increased risk of PPD recurrence.

Note. PP=postpartum, T = trimester. See Table 4 for list of abbreviations.

In order to examine whether sleep disturbances precede the onset of PPD, it is therefore important that studies control for depression at baseline. However, three prospective studies that did control for depression reported conflicting findings (Marques et al., 2011; Okun, Hanusa, et al., 2009; Wolfson et al., 2003). The first of these was carried out by Wolfson et al. (2003), who found that women who showed symptoms of PPD did not differ in their sleep at 2-4 weeks postpartum compared to those without PPD, but their third trimester was characterised by longer naps, later rise times and more total sleep time. Contrary to what may be expected, these results are suggestive of better sleep during late pregnancy in women at risk of PPD. However, it could be that these women were less prepared for what was to come, and may have experienced more drastic changes in their sleep from pre- to postpartum. This would be similar to the findings of Lee, McEnany and Zaffke (2000) who found that women with negative postpartum affect showed longer sleep during the third trimester but significantly shorter sleep postpartum compared to women with positive postpartum affect, which was suggestive of a more drastic change in sleep quantity preceding the development of poor postpartum mood.

Another prospective study was carried out by Okun et al. (2009), who examined the relationship between sleep and PPD in women with a history of PPD. While sleep quality in late pregnancy was not related to the likelihood of PPD recurrence, it was related to timing of recurrence. Better sleep quality in late pregnancy (according to the PSQI) related to early recurrence (within 4 weeks), whereas poor sleep quality related to later recurrence. Although the explanation for these results is unclear, this study highlights the need to measure PPD at several time-points in the postpartum period.

A recent study examined the relationship between insomnia in late pregnancy and PPD in a large sample of women (n=581; Marques et al., 2011). Women were asked a series of (non-validated) questions about their history and current insomnia symptoms. These were focused on difficulty falling asleep, nightly awakenings, early morning awakenings and daytime consequences of sleep disturbances (over the past month). The authors found that insomnia in late pregnancy predicted PPD symptomatology (measured using the BDI-II) but not diagnosis (measured via a face-to-face clinical interview). This is an interesting finding as it highlights the importance of the way in which PPD is measured. However, this relationship was no longer significant when controlling for postpartum affect and history of depression. The authors suggested that negative affect may be an important mediator between insomnia during pregnancy and PPD. However, since a number of women in the study had a lifetime history of depression, the cause and effect relationship between

insomnia and PPD could not be addressed. Furthermore, the authors did not specifically examine which sleep-related disturbances were most strongly related to PPD.

Overall, the longitudinal subjective sleep studies suggest that women's sleep during late pregnancy is related to the development of PPD symptoms. However, the majority of studies focus on a specific aspect of sleep and vary considerably in their definition of 'subjective' sleep. Not all studies controlled for depression at baseline, therefore it is unclear whether poor sleep was simply a symptom of pre-existing depression. Future research should tease apart which specific aspects of subjective sleep appear to be most relevant to postpartum depression, while controlling for depressive symptoms during pregnancy.

3.6.2 Objective Sleep Studies (Longitudinal)

The studies that have assessed the longitudinal relationship between sleep and PPD using objective sleep (PSG or actigraphy) are presented in Table 8. Given the limitations of subjective sleep measures and cross-sectional designs, these studies are considered the most informative.

Out of these studies, only two used PSG as part of their longitudinal design (Coble et al., 1994; Lee et al., 2000). Coble et al. (1994) carried out PSG on 34 healthy women (14 of whom had a history of affective disorder) during pregnancy and the postpartum period. However, rather than specifically investigating the relationship between poor sleep and PPD, this study focused on how sleep differed in women with and without a history of affective disorder. Compared to women with no history of affective disorder, those with a positive history showed longer total sleep time during pregnancy but less total sleep time in the postpartum. Although both groups showed similar trajectories over time, the magnitude of changes were stronger for the women with a history of affective disorder. REM latency also differed between groups; while values were similar for both groups at 12 and 24 weeks of pregnancy, by 36 weeks, women with a positive history experienced a marked decrease in REM latency. This remained reduced for the remainder study period, supporting previous findings that reduced REM latency is associated with increased vulnerability to depression (Giles et al., 1990). In contrast, women with no history of affective disorder experienced an increase in REM latency at 36 weeks, which dropped slightly after birth. However, since only one woman with a history of depression recurred, the authors suggested that although sleep disturbances may be an important marker of those 'at risk' of PPD, other factors must also be involved.

Lee et al. (2000) conducted PSG on 31 healthy women with no history of psychiatric disorders. Women were studied in each trimester of pregnancy as well as postpartum. Women were divided into negative and positive postpartum affect groups, according to those whose depression scores

increased or decreased by more than 30% from the third trimester. Those in the negative affect group had shorter REM latency and less REM sleep in the postpartum compared to the third trimester, whilst the positive affect group also had shorter REM latency and more REM sleep. The positive affect group had stable sleep times through the third trimester and postpartum, but the negative affect group slept 20 minutes longer than the positive group in the third trimester but 80 minutes less one month postpartum. This is similar to the finding by Wolfson et al. (2003) that women who sleep longer during late pregnancy are at greater risk of developing depression, perhaps because they experience more drastic changes in their sleep patterns from pregnancy to postpartum.

Even though both of the above studies varied in their methodology and samples, both suggest that women 'at risk' (whether due to a history of affective disorder, or experiencing negative affect) show reduced REM latency and a more dramatic decrease in sleep from 3rd trimester to postpartum. A limitation of these studies is that neither used a validated measure of PPD.

The remaining studies that assessed the longitudinal relationship between objective sleep and PPD used actigraphy. Goyal et al. (2009) assessed sleep during the first three weeks postpartum but did not include any antenatal sleep measures, and the data was not analysed prospectively. As measured by actigraphy, mothers who slept less than four hours between 12-6am and who napped less than 60 minutes per day had a significantly increased risk of PPD. Sleeping through the night and napping in the day were associated with a decreased risk. However, even after controlling for other risk factors including objective sleep, mothers who felt that they were not obtaining adequate sleep (either difficulty falling asleep or excessive daytime sleepiness) had significantly increased risk of PPD. This suggests that while daytime napping and sleeping throughout the night are important in protecting against PPD, a mother's overall subjective perception of her sleep is most important. This is similar to the findings of Hiscock and Wake (2001), who found that mothers who reported good subjective sleep quality despite poor infant sleep were less likely to be depressed than those who reported poor sleep quality.

Table 8. Longitudinal Studies Assessing the Relationship between Objective Sleep and PPD

Authors, Publication year	Sample / time-points	Measure of sleep	Measure of depression	Outcome
Coble et al. (1994)	34 women examined from 12 wks pregnancy through to 8 mths PP. 14 had a history of affective disorder.	PSG 2 weeks of sleep diaries Subjective sleep information taken from HRDS	Research Diagnostic Criteria (to diagnose history)	Those with history of affective disorder had: reduced REM latency from 3 rd T – 8mth PP; greater and earlier onset of sleep disturbances and showed disturbed subjective sleep; more dramatic decrease in TST from 3 rd T to 1mth PP. Only 1 woman became depressed.
Lee et al. (2000)	31 pregnant women followed from 1 st T through to 12 weeks PP. Screened for depression at baseline.	PSG (home) – 2 nights at each point 7 day sleep/activity diary	CES-D (classified as PA / NA according to whether scores changed by $\geq 30\%$ from pregnancy to PP), POMS	Those with NA had shorter REM latency and less REM compared to 3 rd T whilst those with PA had shorter REM latency but more REM. Those with PA had stable TST from 3 rd T to 3-4wks PP; those with NA slept 20mins longer 3 rd T and 80mins less at 3-4wks PP
Goyal et al. (2009)	112 first-time mothers followed from 3 rd T through to 3 weeks PP	GSDS Actigraphy for 48 hours at each time-point	CES-D cut-off ≥ 16	At 3mths PP: sleeping at least 1hr during the day was associated with lower PPD scores and spending two or more hours awake between 12 AM and 6 AM was associated with higher PPD (actigraphy). Regardless of other factors, including objective sleep, mothers who felt they were not obtaining adequate sleep, (either in the GSDS subscale for difficulty falling asleep or for excessive daytime sleepiness) had higher PPD scores.

Bei, Milgrom, Ericksen, & Trinder (2010)	44 women followed from 3 rd T through to 1wk PP. Screened for depression at baseline	7 days actigraphy at each time-point PSQI	HADS PANAS DASS	Subjective sleep quality during 3 rd T and PP associated with PP HADS depression. TST (actigraphy) in 3 rd T associated with PP positive affect (not other depression scales). Strongest relationships were between PP subjective sleep and PPD.
Coo Calcagni, Bei, Milgrom, & Trinder (2012)	72 women followed from 3 rd T to 1wk PP	Actigraphy worn for 7 days during 3 rd T and into 1 st week PP. PSQI	HADS DASS	Neither objective nor subjective sleep during 3 rd T was significantly related to PPD. Subjective sleep in the PP was the only factor related to PPD; accounted for 35% in HADS depression scores.

Note. PP=postpartum, T = trimester. See Table 4 for list of abbreviations. PA = positive affect, NA = negative affect.

The importance of subjective sleep quality was also highlighted by Bei et al. (2010), who found that subjective night-time sleep, sleep-related daytime dysfunction and daytime napping both during the 3rd trimester and early postpartum period, were the variables most strongly associated with symptoms of PPD at one week postpartum. Poor objective sleep (measured using actigraphy) in the third trimester did not contribute to PPD symptoms measured via the HADS or DASS. The authors concluded that the perception of poor sleep and the conscious awareness of its impact during the day may share a stronger relationship with the occurrence of immediate postpartum mood than objective sleep quality and quantity.

An extension of this study was carried out by Coe, Calcagni, Bei, Milgrom and Trinder (2012), who additionally investigated whether parity affected the relationship between sleep and PPD. In contrast to Bei et al. (2010), neither objective (actigraphy) or subjective third trimester sleep related to postpartum mood (at one week postpartum). Postpartum subjective sleep quality was, however, a significant predictor of postpartum mood and depressive symptoms. Apart from a stronger relationship between subjective sleep and stress in first-time mothers, no other differences emerged when assessing parity.

Both of these studies concluded that subjective sleep appears to be more relevant to PPD than objective sleep. However, since these studies relied upon actigraphy as the objective measure, it is possible that differences may have emerged in relation to sleep architecture, which could only be revealed if using PSG. Although the findings of Coble et al. (1994) and Lee et al. (2000) are limited, both of these studies found that PSG revealed differences in the sleep architecture of women at risk of PPD. This warrants the need for future studies to include PSG as a measure of objective sleep. Another limitation of these studies is that depressive symptoms were only measured at one week postpartum, which likely reflects postpartum blues rather than depression (O'Hara, Schlechte, Lewis, & Wright, 1991).

Another point to note is that although none of these studies specifically compared subjective and objective sleep, the fact that they do not change in parallel, and that subjective sleep appears to be more strongly associated with PPD, suggests that they are measuring different concepts. However, as these findings directly relate to sleep measured using actigraphy, this further highlights the need for studies to include PSG.

3.6.3 Overview of Longitudinal Studies

Longitudinal studies investigating the relationship between sleep and PPD are considerably lacking in this population. Each of the studies described within this section examined sleep and

PPD over a number of time-points, yet not all studies investigated the relationship between sleep and PPD in a prospective manner. Furthermore, some did not control for depression at baseline, so it is possible that poor sleep could have been a symptom of pre-existing depression, rather than a causal factor in its development. A major limitation of the objective longitudinal studies is that the majority only measured early symptoms of PPD. Only two measured symptoms after three weeks postpartum. Some women would have been likely to develop symptoms after this period, and it is important to consider the difference between the blues and PPD (Jones & Cantwell, 2010).

Those that have assessed sleep during the third trimester of pregnancy have suggested that this is relevant to PPD. However, the specific nature of this relationship remains unclear. Some research suggests that women 'at risk' appear to sleep well during late pregnancy, but may experience a more dramatic change in sleep parameters from pre- to postpartum, which could place them at greater risk of developing PPD. In contrast, others have found that poor subjective sleep quality in late pregnancy is associated with an increased risk of PPD. However, definitions of sleep quality are diverse, and few studies have specifically teased apart which factors of sleep are most relevant. Furthermore, future research is needed to establish what factors affect subjective perceptions of sleep quality. The findings relating to PSG are limited and more research needs to be done in order to clarify whether abnormal sleep architecture may place women at risk of PPD. This will be investigated later in the thesis.

3.7 Fatigue and Postpartum Depression

As described in Chapter 1, the relationship between sleep and fatigue is not straightforward. Individuals reporting fatigue do not necessarily show impaired sleep (Lavidor et al., 2003). However, it is important to recognise fatigue as another factor that may relate to PPD, since it is commonly experienced among perinatal women (Lee & Zaffke, 1999)

As well as being a common symptom of major depressive disorder, fatigue has also been shown to significantly predict its onset. Addington, Gallo, Ford and Eaton (2001) found that individuals without current depression, but who reported fatigue, were 28.4 times more likely to develop depression thirteen years later than those who did not have fatigue at baseline. It has been suggested that both excessive sleepiness and fatigue may represent

prodromal symptoms of major depressive disorder (Fava, Grandi, Canestrari, & Molnar, 1990).

Similarly, some research has also suggested that fatigue may be predictive of PPD (Bozoky & Corwin, 2002; Corwin et al., 2005). Fairbrother, Hutton, Stoll, Hall and Kluka (2008) found that fatigue scores measured by the Multidimensional Assessment of Fatigue Scale (Belza et al., 1993) were significantly correlated with measures of depression and sleep disturbances during pregnancy and postpartum. The correlation remained strong even after removing items on the BDI that overlapped with fatigue. This suggests that the relationship between postpartum fatigue and low mood is not simply a function of overlapping item content. Fatigue will therefore be included in this thesis as a separate factor that will be investigated in relation to PPD.

3.8 Measuring Postpartum Depression

This review has specifically focused upon the different ways that sleep has been measured in relation to PPD, however it is also important to note the variety of PPD measures that have been used. Within the studies described, the following measures of PPD were used: the EPDS, CES-D, POMS, Research Diagnostic Criteria, PDSS, Stein Questionnaire, VAS mood scales, HRDS, Perceived Stress Scale, Zung Depression Scale, BDI-II, PANAS and DASS (see Table 4 for full names and authors).

While the DSM-5 categorises PPD as a sub-type of major depression occurring within four weeks postpartum (American Psychiatric Association, 2013); it has been argued that this time-frame does not allow for adequate detection of symptoms (Gale & Harlow, 2003). Furthermore, many aspects of PPD make it distinct from other episodes of major depression (Gale & Harlow, 2003). Therefore, specific measures of PPD are useful. Specific PPD scales include the EPDS (Cox et al., 1987) and the PDSS (Beck & Gable, 2002). These will be described in more detail in the thesis.

The EPDS is the most commonly used scale within the studies reviewed. It is the most frequently researched method to identify PPD and has been translated and validated in multiple different languages (Hewitt, Gilbody, Mann, & Brealey, 2010). A review of eight different instruments for the screening of PPD, including the BDI, EPDS, Postpartum Depression Screening Scale and the Zung self-rating Depression Scale, found that the EPDS was the most widely studied measure with moderate psychometric soundness (Boyd, Le,

& Somberg, 2005). However, the EPDS has low to moderate correlations with state anxiety and therefore may not be a pure measure of PPD (Brouwers, van Baar, & Pop, 2001).

It has been suggested that general depression screening instruments can be used as time after delivery increases, but a more specific scale, such as the EPDS, should be used in the early postpartum period to account for the somatic changes associated with this time (Boyd et al., 2005). Although screening scales are useful in screening women for PPD, they are not intended to be the primary mode of diagnosis. While majority of studies within this review did not include a clinical diagnosis, they do provide a strong indication of women at risk of PPD.

3.9 Summary and Limitations of the Research to Date

The current literature examining the relationship between sleep and PPD is limited, and the varied methodology that exists between studies makes it difficult to infer the precise nature of this relationship. However, some preliminary themes can be drawn.

Cross-sectional studies have indicated that women experiencing symptoms of PPD are likely to report poorer sleep; particularly poor subjective sleep quality. However, definitions of poor sleep quality remain vague. In contrast to the subjective studies, the majority of objective studies did not find poorer sleep in women with PPD, perhaps suggesting that subjective sleep is more relevant to PPD. Due to the bi-directional relationship between sleep and depression, cross-sectional studies do not provide information regarding whether sleep disturbances or depression arose first.

The importance of subjective sleep was also highlighted within the longitudinal research. Subjective sleep in late pregnancy appears to be related to PPD, although the exact nature of this relationship is unclear. Some research found that subjective reports of sleeping longer in late pregnancy are associated with increased risk of PPD, while others found that poor subjective sleep quality during this time places women at greater risk. However, the discrepancy between these findings may be due to fact that the studies were measuring different aspects of subjective sleep (qualitative versus quantitative).

The difference between objective and subjective sleep measures has been highlighted throughout this review. Studies combining both types of measures can provide more intricate detail about the nature of this relationship. A 'gold standard' study would therefore include both subjective and objective measures into a longitudinal design. Only

one such study to date has adopted this design (Bei, Milgrom, Ericksen, & Trinder, 2010). This study concluded that subjective sleep appears to be more relevant to PPD than objective sleep. However, as it relied upon actigraphy as the objective measure (rather than using PSG), important information may have been missed about women's sleep architecture. Indeed, some aspects of sleep architecture described previously in this thesis (such as reduced REM latency) are predictive of those at risk of major depression (Giles, Kupfer, Rush, & Roffwarg, 1998), therefore it is not unreasonable to expect that similar relationships may exist with PPD. Only two studies examined within this review investigated the prospective relationship between sleep architecture and PPD using PSG (Coble et al., 1994; Lee, McEnany, et al., 2000). The findings of these studies were limited due to non-validated definitions of PPD and a lack of concurrent subjective measures. What these studies did suggest, however, is that some aspects of sleep architecture, particularly REM sleep, appear to be relevant to PPD.

By providing details about sleep architecture, PSG would provide intricate information about women's sleep that may be missed by actigraphy or solely relying on subjective measures of sleep. Furthermore, it may have been that the participants in Bei et al.'s (2010) study reported poor subjective sleep quality due to decreased amounts of SWS, as found in previous research (Keklund & Akerstedt, 1997). None of the studies in this review reported on relationships between subjective and objective measures, which is important if we are to fully the relationship between sleep and PPD.

Another point to highlight is that many of the measures of both sleep and depression used within the studies described were not specific to the perinatal period. Although some aspects of sleep and depression are likely to be similar among perinatal and non-perinatal women, certain factors specific to perinatal women should be considered. For example, typical symptoms of insomnia (such as nightly awakenings) may overlap with general infant care duties. Furthermore, the PSQI, which was the most commonly used measure of sleep, refers to sleep perceptions over the past month. Since women's sleep appears to change significantly during this time, it may not be the best measure to use; particularly if we are to understand the specific periods in which sleep is most important.

3.10 Recommendations for future research

Based on the limitations discussed in relation to previous research, the following suggestions are proposed for future studies within this area.

Future studies should:

- 1) Use longitudinal designs and measure the relationship between sleep and PPD in a prospective manner.
- 2) Screen for depression at baseline.
- 3) Assess sleep during pregnancy as well as in the postpartum period.
- 4) Use an appropriate measure of sleep, paying particular consideration to the time-frame in which the measure refers to, and whether it is relevant to perinatal women. Sleep diaries may provide more specific information on sleep at a particular moment in time, and may be less susceptible to memory biases.
- 5) Tease apart the specific aspects of sleep that appear to be most relevant to PPD.
- 6) Use a validated measure of PPD.
- 7) Incorporate PSG alongside measures of subjective sleep, and examine specific relationships between these measures.
- 8) Distinguish between measures of sleep quality and sleep quantity.

3.11 Aims of the Thesis

The aims of the thesis are described below.

1) To examine which factors of subjective sleep are most predictive of depression in a general sample

The review of sleep and PPD suggests that subjective sleep appears to be related to the development of PPD. However, there are different ways in which subjective sleep quality can be measured, and the literature review found that different types of subjective sleep measurements appear to share different relationships with PPD (e.g. Dorheim, Bondevik, Eberhard-Gran, & Bjorvatn, 2009). Therefore, the first aim of the thesis was to explore the

relationship between different aspects of subjective sleep and depression in a general sample, in order to delineate which aspects of subjective sleep were most relevant to depression. This would inform the rationale and choice of measures for the main study of perinatal women. Based on previous research (e.g. Mayers, Grabau, Campbell, & Baldwin, 2009; Pilcher & Ott, 1998), it was anticipated that depression would be more strongly related to sleep quality and fatigue than to sleep quantity.

2) To compare how sleep differs in pregnant and non-pregnant women

Despite the potential adverse outcomes of poor sleep, there remains much to be known about sleep changes during pregnancy (this was described in Chapter 1). Recent years have seen an increase in the number of studies within this area (e.g. Jomeen & Martin, 2007; Ko, Chang, & Chen, 2010; Signal et al., 2007; Wilson et al., 2011). However, fewer than ten studies have used examined women's sleep during pregnancy using polysomnography (Brunner et al., 1994; Coble et al., 1994; Driver & Shapiro, 1992; Hertz et al., 1992; Karacan et al., 1968; Lee et al., 2000; Wilson et al., 2011). Of these, only three have recruited a control group of non-pregnant women (Hertz et al., 1992; Karacan et al., 1968; Wilson et al., 2011). The small sample sizes and varied methodologies have resulted in conflicting findings regarding the effect of pregnancy upon sleep architecture. Therefore, the second aim was to further explore the nature of the sleep differences that exist between pregnant and non-pregnant women, using polysomnography. It was anticipated that pregnant women would experience poorer sleep than non-pregnant women on a number of measures.

3) To investigate the relationship between subjective and objective sleep in pregnant and non-pregnant women; both in terms of accuracy, and factors affecting perceptions of sleep quality.

Chapter 1 described the relationship (and potential differences) between subjective and objective measures of sleep, both in terms of the extent to which an individual is accurate in their sleep perceptions and the factors of sleep that most affect subjective perceptions of sleep quality. The relationship between subjective and objective sleep in perinatal women has not previously been examined in the literature. This is an important first step in understanding the differences that appear to exist between subjective and objective sleep in relation to postpartum depression (e.g. Bei, Milgrom, Ericksen, & Trinder, 2010;

Dorheim et al., 2009). Given previous research suggesting that those with more disturbed sleep are likely to be less accurate in their sleep perceptions (Tsuchiyama et al., 2003), it was anticipated that due to poorer sleep, pregnant women would be less accurate in their sleep perceptions compared to non-pregnant women.

4) To examine longitudinal changes in sleep, fatigue and depression throughout the perinatal period.

As well as comparing specific aspects of sleep between pregnant and non-pregnant women, it is also important to consider changes to women's sleep over the course of the perinatal period. No studies to date appear to have concurrently examined longitudinal changes to sleep, fatigue and depression over the perinatal period. Therefore, the fourth aim was to investigate how women's sleep changed from late pregnancy into the postpartum period, and whether these sleep changes were mirrored by changes in fatigue and depression.

5) To investigate the association between subjective sleep, objective sleep, fatigue and depressive symptoms during late pregnancy and the postpartum period.

Given the conclusions of the systematic review, more research is needed to examine the relationships between subjective sleep, objective sleep, fatigue and depression throughout the perinatal period. Based on previous research, the relationship between these measures was examined at three time-points: during pregnancy and at 1 and 12 weeks postpartum. It was anticipated that significant relationships would exist between these measures, and that week 12 EPDS scores would be most strongly related to sleep and fatigue during pregnancy.

6) To investigate which subjective and objective sleep variables most strongly predicted depression scores at 1 and 12 weeks postpartum.

Given the lack of clarity in relation to the specific aspects of sleep that are most relevant to PPD (described in Chapter 3), a series of regression analyses were carried out to address this question. Based on previous research investigating sleep, depression and postpartum depression (e.g. Bei et al., 2010; Chen, Burley, & Gotlib, 2012; Dorheim, Bondevik, Eberhard-Gran, & Bjorvatn, 2009; Giles, Jarrett, Roffwarg, & Rush, 1987; Kupfer, 1984), it

was anticipated that the strongest predictors of EPDS would be third trimester subjective sleep quality and REM latency.

Having reviewed the relevant research in this area, the remainder of the thesis will describe the studies that were carried out in order to address these aims. Although the main focus of this thesis is to investigate the relationship between sleep and PPD, the studies reviewed within this chapter emphasised that sleep is multidimensional and can be measured in a number of ways. Furthermore, in the literature reviewed, these measures were differentially related to PPD.

Before describing the main study of this thesis, the next chapter describes a preliminary study that was carried out in order to capture the essence of the nature of the relationship between subjective sleep and depression, by delineating which aspects of subjective sleep (qualitative versus quantitative), as well as fatigue, are most relevant to depression.

CHAPTER 4: RELATIONSHIPS AMONG SLEEP QUALITY, QUANTITY, FATIGUE AND DEPRESSION IN A GENERAL SAMPLE.

The aim of this study was to gain a better understanding of the specific types of sleep disturbances that are most relevant to depression. Ninety-one participants completed a week of St Mary's Hospital Sleep Questionnaire followed by the Beck Depression Inventory-II and The Multidimensional Assessment of Fatigue Scale. Those who were screened as depressed reported significantly longer sleep onset latency, more awakenings, poorer sleep quality and more fatigue, but did not differ in relation to sleep quantity. This supports the theory that depression is more strongly related to sleep quality than sleep quantity.

4.1 Background

The research examined so far in this thesis has highlighted that those with depression experience poorer sleep. Studies using polysomnography (PSG) have examined a number of markers relating to abnormal sleep architecture in depression, including increased sleep onset latency, increased wake after sleep onset, poorer sleep efficiency, less slow-wave sleep, reduced REM latency and increased REM activity (Kupfer, 1984; Tsuno et al., 2005). While these factors are important in identifying potential biological markers of depression, most individuals do not undergo PSG, and instead clinicians rely on subjective reports of sleep. Therefore, this initial study examined the relationship between different components of subjective sleep and depression.

Previous research has demonstrated that individuals with depression tend to report poorer subjective sleep satisfaction than non-depressed individuals, in the absence of complaints surrounding sleep quantity (Mayers et al., 2009). This suggests that the way in which depressed individuals perceive their sleep is not necessarily related to the amount of sleep that they receive, and could be related to cognitive factors, such as negative thinking. Discrepancies between sleep quality and quantity in relation to depression were also reported by Bower, Bylsma, Morris and Rottenberg (2010), who found that poor subjective sleep quality and daytime dysfunction were the only Pittsburgh Sleep Quality Index (PSQI) components that significantly predicted negative and positive affect in healthy and mood-disordered individuals. As described in Chapter 3, similar relationships have been reported in women with postpartum depression (PPD). Two studies found that women with PPD reported significantly poorer sleep quality than non-depressed

postpartum women (according to the PSQI), but they did not differ according to sleep quantity (Dorheim, Bondevik, Eberhard-Gran, & Bjorvatn, 2009; Huang, Carter, & Guo, 2004).

A limitation of previous research is that the measures used to investigate subjective sleep are often retrospective and relate to general rather than specific sleep factors. As previously described, the PSQI, a commonly used measure of sleep in those with depression, contains seven subscales that are summated to provide a global score. Some studies of PPD have relied upon this global score (e.g. Da Costa, Dritsa, Rippen, Lowensteyn, & Khalifé, 2006) and therefore are unable to provide specific information about which aspects of sleep are most relevant. While referring to specific PSQI subscales is more informative, individual subscales can still cover a wide range of factors. For example, the 'sleep disturbance' subscale covers disturbances including difficulty falling asleep, middle of the night / early morning waking, having to get up to use the bathroom, coughing / snoring loudly, feeling too hot / cold, having bad dreams, having pain and breathing uncomfortably. Therefore, there is a need for studies to measure individual aspects of subjective sleep in order to work out which factors are most relevant to depression.

Another important consideration is that differences have been shown to exist between retrospective and prospective measures of sleep (Babkoff et al., 1996; Habte-Gabr et al., 1991). The PSQI is a retrospective measure, referring to sleep in the past month. Since depression is characterised by negative thinking style, a cognitive bias towards negative information and negative autobiographical memory (Beck, 1964; Gotlib, 1983; Hamilton & Gotlib, 2008), depressed individuals may be more likely to recall their sleep as poor. In light of this, Dorheim, Bondevik, Eberhard-Gran and Bjorvatn (2009) found that women with PPD differed to non-depressed women in relation to retrospective measures (PSQI) but not prospective measures (actigraphy and sleep diaries). Therefore, using prospective measures may be a means of providing a more accurate representation of sleep in those with depression, and one that is less likely to be affected by memory bias.

The current study used the St Mary's Hospital Sleep Questionnaires (SMHSQ; Ellis et al., 1981), because it is a validated prospective measure of sleep. It also contains questions relating to both sleep quantity and quality, allowing for exploration of specific types of subjective sleep factors most relevant to depression. Furthermore, the SMHSQ has

previously been used to compare PSG and subjective sleep perceptions in depressed patients (Argyropoulos et al., 2003).

Fatigue is another factor that has been related to both major depression and PPD (Baldwin & Papakostas, 2006; Bozoky & Corwin, 2002; Dennis & Ross, 2005; Lee & DeJoseph, 1992). However, as previously described in this thesis, whether sleep and fatigue are closely related, and therefore show similar relationships with depression, remains unclear (Lavidor et al., 2003). Previous research found that fatigue is more strongly related to subjective sleep quality rather than quantity (Lavidor et al., 2003), which again highlights the need to examine individual aspects of sleep. This study therefore also sought to clarify the relationships between fatigue, subjective sleep, and depression.

4.2 The Current Study

The main aim of this study was to investigate which specific factors of subjective sleep were most relevant to depression. Although a great deal of research has been undertaken to examine the general relationship between sleep and depression, it has not dealt with the specific aspects of subjective sleep that are most relevant. This relationship was investigated in a general population sample, before examining these relationships within perinatal sample. A second aim was to examine the relationships between sleep and fatigue, in order to identify which aspects of sleep are most strongly related to fatigue. The final aim was to identify which aspects of sleep appeared to be most strongly related to subjective perceptions of sleep quality, given that that sleep quality appears to be related to depression.

The relationship between sleep and depression was measured using a prospective sleep diary measure, detailing several different aspects of subjective sleep. Using a prospective measure meant that recall would be less likely to be affected by memory bias, and would therefore provide a more accurate representation of sleep. Additionally, a measure of fatigue was included in order to investigate its relationship with subjective sleep quality, quantity and depression. The factors measured in this study were as follows:

- 1) Sleep quantity (total sleep time)
- 2) Ease of falling asleep and sleep continuity (sleep onset latency and number of awakenings)

- 3) Sleep quality (single score derived from depth of sleep, how 'well' slept, satisfaction)
- 4) Fatigue (global score)
- 5) Depression (global score)

It was predicted that those with depression would report poorer sleep quality and more fatigue than non-depressed individuals, but that they would not differ in relation to sleep quantity. It was anticipated that fatigue and sleep quality would significantly predict depression score. Based on previous research (e.g. Lavidor et al., 2003), it was also anticipated that fatigue would be more strongly related to sleep quality than sleep quantity.

4.3 Method

4.3.1 Design

This was a cross-sectional study whereby associations were measured between sleep, fatigue and depression at a single time-point. Between-group analyses were carried out between individuals with high depression scores versus those with low depression scores, according to a score of ≥ 17 on the Beck Depression Inventory II (Beck, Steer, & Brown, 1996).

4.3.2 Participants

One hundred and twenty questionnaires were sent out to a wide range of individuals, both in the community and at the university. University students were provided with course credit for taking part in the study. No other incentives were given. Of the questionnaires sent out, 91 of these were returned and fully completed. Table 9 shows that the majority of participants were female and university students, with most of the remainder being in full- or part-time employment. According to self-disclosed diagnosis of depression, only three participants had a current diagnosis, although five reported taking antidepressant medication. This is considerably less than estimated prevalence in western countries (Bromet et al., 2011). However, not all individuals experiencing depressive symptoms will have received a formal diagnosis, which may account for the low prevalence rate in this sample. Self-reported depression scores will be reported in the results section. Despite the

low prevalence of currently diagnosed depression, nearly a quarter of participants had a history of depression, and over a third reported that a close family member had previously been depressed. Only three participants *reported* a diagnosed sleep disorder, with two participants reporting insomnia and one participant reporting restless legs syndrome. Again, this is less than would be expected in the general population (Ohayon, 2007), but may be due to a relatively young mean age of participants and a lack of clinical diagnoses.

Table 9. Socio-demographic characteristics of participants

Age (years; mean \pm SD)	26.9(\pm 13.0)
Gender (no. of cases)	
Female	65
Male	26
Occupation (%)	
Full-time education	72.5
Full-time employment	13.2
Part-time employment	7.7
Homemaker	1.1
Unemployed	2.2
Retired	3.3
Currently diagnosed as depressed (no. of cases)	3
Taking antidepressant (no. of cases)	5
History of depression (%)	
Personal history	22.0
Family history	37.4
Currently diagnosed with sleep disorder (no. of cases)	
Insomnia	2
Restless legs syndrome	1

4.3.3 Ethics

Ethical approval study was obtained from the Psychology Research Group at Bournemouth University.

4.3.4 Measures

Sleep: St. Mary's Hospital Sleep Questionnaire (SMHSQ)

The St Mary's Hospital Sleep Questionnaire (SMHSQ; Ellis et al., 1981) consists of 14 items covering: the timing of sleep over the last 24-hour period (time of settling down for the night, falling asleep, waking up, getting out of bed), amount of night-time and daytime sleep, quality of sleep (depth, how 'well' slept, satisfaction), number of awakenings, morning clear-headedness, presence of early morning waking and not being able to return to sleep, difficulty falling asleep and sleep onset latency (see Appendix 1). Each question is scored individually, and respondents are asked to tick the box that most corresponds with their sleep (e.g. 'How well did you sleep last night?' Participants ticked a box ranging from very badly (1) to very well (6)). In this study, a 'sleep quality' was measured by combining the scores of the three items that specifically tap into the individual's perception of their sleep quality (depth, how 'well' slept, satisfaction), as done by in a previous study of sleep and depression (Argyropoulos et al., 2003). High scores indicate good subjective sleep quality. Kendall's tau, a test-retest reliability measure for the questionnaire items, has ranged from 0.70-0.96 (Ellis et al., 1981).

This questionnaire was specifically chosen because it contains questions relating to both sleep quantity and quality, which may differ in relation to depression (Mayers et al., 2009). The SMHSQ has previously been used to compare objective and subjective sleep in depressed patients (Argyropoulos et al., 2003).

Fatigue: Multidimensional Assessment of Fatigue Scale

Fatigue was measured using the Multidimensional Assessment of Fatigue Scale (MAF; Belza, Henke, Yelin, Epstein & Gilliss, 1993). This is a 16-item scale measuring four dimensions of fatigue: severity, distress, degree of interference in activities of daily living and timing. Two items require multi-choice responses and fourteen items contain numerical rating scales. Participants respond based on feelings of fatigue during the past week, and can omit items that do not apply. The scale produces a Global Fatigue Index score (GFI) ranging from 1 (no fatigue) to 50 (severe fatigue). The MAF has been shown to

have high convergent validity (Cronbach's $\alpha = .93$) and concurrent validity with the fatigue subscale of the Profile of Mood States ($r = .84$; Belza, 1995). This measure was specifically used as it refers to fatigue symptoms over the past week, avoiding the possible biases discussed in relation to retrospective measures relating to longer periods of time. This measure was completed at the end of the week so that it would relate to the sleep of the previous week, in order to examine relationships between the measures. Although this study was not examining perinatal women, it has been specifically validated for use within this population (Fairbrother et al., 2008). Therefore, another benefit of including it in this preliminary study was to assess whether it was a useful measure in assessing the relationship between fatigue, sleep and depression, before applying it to the later study involving pregnant women.

Depression: Beck Depression Inventory (BDI-II)

The BDI-II (Beck, Steer, & Brown, 1996) was used to measure depressive symptoms and severity. It includes 21 questions, each measured on a 4-point scale (0 = absence of symptoms, 3 = severe symptoms). Respondents were asked to choose the statement that best reflects how they have been feeling over the past two weeks; including today. Overall scores range from 0 to 63; with higher scores indicating more severe depressive symptoms. In this study a cut-off score of ≥ 17 was used to identify depression, as described in the BDI-II manual (Beck et al., 1996). In the study described by Beck et al. (1996), using this score yielded a 93% true-positive rate and an 18% false-positive rate. Additionally, Carney, Ulmer, Edinger, Krystal and Knauss (2009) found 81% sensitivity and 79% specificity for depression using this cut-off amongst individuals with insomnia. The same method of classification was adopted in this study in order to compare sleep items between groups. Questions cover both cognitive and somatic symptoms of depression. For the purpose of this study, the sleep item (question 16) was excluded to avoid confounding depressive symptoms and sleep ratings. The BDI-II has good internal consistency (Cronbach's $\alpha = .93$) and a test-retest reliability of .93 (Beck et al., 1996).

The BDI-II was chosen because it is a well validated measure that has been used in previous studies to assess the relationship between sleep and depression (e.g. Isaac & Greenwood, 2011; Swanson, Flynn, Wilburn, Marcus, & Armitage, 2010). Given that it contains 21 questions relating to both cognitive and somatic symptoms, it is more thorough than other self-report scales such as the Hospital Anxiety and Depression Scale which only contains seven questions related to depression (Zigmond & Snaith, 1983).

4.3.5 Procedure

Participants were given a pack containing one of each of the above questionnaires, and seven copies of the SMHSQ. They were asked to complete the SMHSQ each morning for seven consecutive days, and to complete the MAF and the BDI-II at the end of the week. The questionnaire pack also contained an information sheet and a demographics questionnaire to obtain psychosocial information from participants. The information derived from the demographics questionnaire is presented in Table 9.

4.4 Results

4.4.1 Overview of Statistical Analyses

Statistical analyses were performed using SPSS, version 19.0 for Windows (SPSS, Inc., Chicago, IL). All data were checked for normality via Kolmogorov-Smirnov tests and skewness and kurtosis. The quantitative and qualitative sleep factors were averaged over the seven days. Mean scores were used in all subsequent analyses. Sleep variables were normally distributed across depression groups. Independent *t*-tests were carried out to examine differences in sleep variables between groups. The sleep factors that were moderately correlated with depression were then entered into a stepwise multiple linear regression model, to assess which variables most strongly predicted depression score. Data were checked for linearity, multicollinearity and independent errors. The stepwise method was chosen to ascertain the relative contribution of each predictor variable after accounting for the other factors.

4.4.2 Sleep and Fatigue According to Depression Status

Table 10 shows the mean sleep variable scores in those with and without depressive symptoms. Interestingly, although only three participants reported a current clinical diagnosis, a third scored ≥ 17 according to the BDI-II. As predicted, the majority of sleep variables were poorer among those with depression. Individuals with depression also reported significantly higher fatigue scores, compared to the non-depressed group. However, total sleep time did not differ significantly between groups.

Table 10. Mean Sleep Disturbances Scores According to Depressive Symptoms

	Non depressed (n=62)	Depressed (n=29)	p-value
Total sleep time (minutes)	464.21 (57.94)	457.08 (72.89)	.616
Sleep onset latency (minutes)	32.53 (23.10)	48.39 (31.48)	.001
Number of awakenings	1.29 (0.76)	1.79 (0.91)	.001
Sleep Quality	13.18 (1.91)	11.46 (2.12)	.001
Fatigue	18.10 (10.57)	28.99 (9.98)	.001

In order to examine the sleep factors that were most strongly related to depression, a series of correlations were carried out. Since BDI-II score was not normally distributed, transformed BDI scores were used. Table 11 shows correlations between depression scores and each of the different sleep-related variables. The overall pattern of correlations suggests that sleep quality, fatigue and sleep onset latency are moderately correlated with BDI score. However, the association between BDI score and total sleep time was weak and non-significant. This suggests that fatigue, sleep quality, number of awakenings and sleep onset latency each contribute to depression score.

Table 11. Pearson's Correlations between the Sleep Variables and Depression Scores

	Fatigue	Sleep Quality	Total sleep	No. of awakenings	Sleep onset latency
BDI	.434**	-.426*	-.076	.283**	.398 **
Fatigue		-.343**	-.209	.103	.271*
Sleep quality			.215*	-.500**	-.352**
TST				-.021	-.264*
No. of awakenings					.114

**p<.01; *p<.05

4.4.3 The Relationship between Sleep and Fatigue

As predicted, the factor most strongly associated with fatigue was sleep quality. Poor subjective sleep quality was moderately associated with increased levels of fatigue, which

was significant. In contrast, the relationships between fatigue and total sleep time and number of awakenings were weak and were not significant. Higher levels of fatigue were also moderately related to increased sleep onset latency, which was significant.

4.4.4 Factors associated with Subjective Sleep Quality

The factor that was most strongly associated with sleep quality was number of awakenings. Fewer awakenings were strongly associated with better subjective sleep quality. Sleep quality was weak-moderately correlated with sleep onset latency and total sleep time.

4.4.5 Regression Analysis to Predict Depression Score

A multiple linear regression analysis was carried out to examine which sleep variables best predicted depression. Total sleep time was not included in the model as it was not significantly correlated with BDI score. The following variables were entered into the regression model, using the stepwise method: sleep quality, sleep onset latency, number of awakenings and fatigue score. Results are reported in Table 12.

Table 12. Stepwise Regression Analysis with BDI-II score as the Outcome Measure

Predictor variable	R ²	Adjusted R ²	R ² change	F	Gradient	t	p
Model	.310	.285		21.22**			
Fatigue			.188**		.261	2.98	.004
Sleep quality			.081**		-1.70	-2.41	.018
Sleep onset latency			.041*		.083	2.23	.029

**p<.01; *p<.05

The result of the regression analysis showed that fatigue, sleep quality and sleep onset latency significantly predicted depression, accounting for 31% of the sample outcome variance (adjusted R²=.285). Fatigue accounted for the greatest variance in depression scores. After accounting for fatigue, sleep quality independently accounted for an additional 8.1% of the variance, and sleep onset latency independently accounted for a further 4.1% of the variance. Number of nightly awakenings was excluded as it did not

significantly contribute to the overall model. This supported the hypothesis that depression would be better predicted by sleep quality and fatigue, than by quantity of sleep. Increased sleep onset latency also appears to contribute to depression scores.

4.5 Discussion

Overall, the findings support the hypothesis that depression is associated with poorer sleep. When participants were categorised as depressed / non-depressed, those with depression reported significantly poorer sleep. In terms of quantitative aspects of sleep, those with depression had longer sleep onset latency and more awakenings but did not differ in terms of total amount of sleep. This supports the hypothesis that depression is more strongly associated with sleep quality rather than sleep quantity, and is in line with previous research (Bower et al., 2010; Mayers et al., 2009)

As predicted, those with depression also experienced more fatigue. This supports previous research highlighting the relationship between fatigue and depression (Ferentinos et al., 2009). Furthermore, fatigue shared stronger associations with sleep quality compared to the quantitative sleep variables. Although fatigue was significantly correlated with sleep onset latency, it was not significantly related to total sleep time or number of awakenings. This supports findings by Lavidor et al. (2003) that fatigue is more strongly related to qualitative, rather than quantitative aspects of sleep.

When the sleep factors correlated with BDI score were entered into regression analysis, fatigue, sleep quality and sleep onset latency were significant predictors of depression score. Number of awakenings was not a significant predictor. However, given that this was strongly related to subjective sleep quality, it may be that number of awakenings was overshadowed by sleep quality. Interestingly, it was fatigue that was the strongest predictor of depression, over and above sleep quality. Since fatigue is a multidimensional construct, and was moderately correlated with both sleep quality and depression, this may reflect some overlap between these concepts. However, the fact that sleep quality independently accounted for additional variance in depression scores after accounting for fatigue suggests that, at least to a certain extent, they reflect separate constructs (Lavidor et al., 2003).

Both subjective sleep quality and fatigue require an element of subjective evaluation, and therefore may be more subject to cognitive biases. This could explain why these factors were more pronounced in those with depression, as depression is characterised by

negative thinking style, a cognitive bias towards negative information and negative autobiographical memory (Beck, 1964; Gotlib, 1983; Hamilton & Gotlib, 2008). In contrast, it could be argued that asking a participant to estimate how much sleep they obtained is more specific, therefore requiring less subjective evaluation. This leads to the question of whether individuals with depression show 'real' sleep deficits, or if they simply perceive their sleep to be worse because they are depressed.

However, poor sleep quality was related to more awakenings and longer sleep onset latency, suggesting that poor sleep among those with depression is not solely due to negative thinking. Increased sleep onset latency may be due to the ruminative thought patterns that are common in depression (Cribb, Moulds, & Carter, 2012), which are known to contribute to difficulties initiating sleep in those with insomnia (Harvey, 2002). Indeed, previous research has found associations between rumination, negative mood and poor sleep quality in a general population sample (Thomsen, Yung Mehlsen, Christensen, & Zachariae, 2003).

In contrast to previous research (e.g. Da Costa et al., 2006; Dorheim et al., 2009), a benefit of this study was that it adopted a prospective measure of sleep, requiring participants to evaluate their previous night's sleep, rather than retrospectively evaluating their sleep habits over the past month. It could be argued that the sleep measures obtained in this study were less subject to memory bias, and may have provided a more accurate representation of the individual's sleep.

A limitation of this study, however, is that it relied upon subjective sleep perceptions, which may have been inaccurate if compared to an objective measure. Furthermore, previous research has suggested that individuals with depression are likely to be less accurate in their sleep perceptions (Armitage, Trivedi, Hoffmann, & Rush, 1997; Rotenberg et al., 2000). Even though quantitative sleep estimates may be less influenced by cognitions than subjective sleep quality, they still involve a perceptual element. Also, this study did not examine sleep architecture. It could be that individuals with depression reported poorer sleep and were more fatigued due to factors relating to sleep stages. It is a well-established finding that depressed individuals often show disturbances in their sleep architecture, including less slow-wave sleep and reduced REM latency (Benca et al., 1992; Kupfer, 1984), and these factors may affect subjective perceptions of sleep quality (Åkerstedt, Hume, Minors, & Waterhouse, 1994; Kerklund & Åkerstedt, 1997).

As with all cross-sectional studies, the findings from this study cannot infer causal relationships between sleep and depression. Since depression is associated with low mood and negative thoughts, this likely reflected the way in which sleep was described amongst depressed individuals. While it is important to understand the way in which sleep problems are reported in those currently depressed, more studies are needed to address the longitudinal relationship between different aspects of sleep and depression.

Overall the findings of this study support previous research that depression is more strongly associated with sleep quality than sleep quantity (Bower et al., 2010; Mayers et al., 2009). Similar relationships have also been reported in relation to postpartum depression (Da Costa et al., 2006; Dorheim et al., 2009). This study highlights the need to tease apart specific aspects of subjective sleep quality when exploring their relation to depression. It also demonstrates that fatigue is an important predictor of depression, and should therefore be examined as a separate factor.

4.6 Overview of Chapter 4

The aim of this preliminary study was to examine whether different aspects of subjective sleep share different relationships with depression. Indeed, the findings of the study showed that sleep quality, fatigue and sleep onset latency are better predictors of depression in a general sample, than total amounts of sleep. The remainder of the thesis describes the main study, which investigated the relationship between sleep and postpartum depression. Building on this preliminary study, the main study included polysomnography to measure objective sleep, and adopted a longitudinal design. The methods used in this study are described in the following chapter.

CHAPTER 5: METHODS

This chapter describes the methods used in the main study investigating the relationship between sleep and postpartum depression (PPD). This chapter will explore the study design, justification for the choice of measures, sample size and selection, procedures, statistical analyses and ethical considerations.

5.1 Choice of Study Design

In order to address the aims and objectives of the research (see sections 3.11), a quantitative design was used. Five dimensions were considered in order to choose the most appropriate quantitative study design (Polit & Beck, 2009). These are: the time frame of the study, the measurement of the variables, control over variables, type of comparison and the degree of structure. These are described below.

Time frame: As described in Chapter 3, the majority of research on sleep and PPD has relied upon cross-sectional designs. However, these studies cannot provide information about whether poor sleep is a causal factor or a symptom of PPD. For this reason a longitudinal design was employed in this study. The chosen time-points are detailed below.

Time-Point 1: Third Trimester of Pregnancy

The literature reviewed in Chapter 1 suggested that the most significant sleep changes during pregnancy occur in the third trimester (Signal et al., 2007). There is evidence that sleep during this time is related to the development of PPD (Bei et al., 2010; Wolfson et al., 2003). In order to better understand this prospective relationship, it was important that sleep was measured prior to the postpartum period (i.e. during pregnancy), so that it would not be confounded with potential symptoms of PPD. Sleep was measured subjectively, through questionnaires, and objectively, via polysomnography (PSG). Given that pregnancy was identified in the literature as a particularly important time, and that new mothers may have been more reluctant to take part once their new-born had arrived, it was decided that PSG would be carried out during the third trimester of pregnancy. Previous research has also shown that specific markers of sleep, such as reduced REM latency, are predictive of those 'at risk' of depression (Lee et al., 1993), further supporting the decision to measure sleep before symptoms of PPD could arise.

Time-point 2: The Week Following Birth

In order to build upon previous studies and to capture changes in the development of PPD symptoms, it was decided that two postpartum time-points would be measured. The second overall time-point of the study was in the early postpartum period, specifically, the week following birth, similar to previous studies (Bei et al., 2010; Coo Calcagni et al., 2012). However, it is acknowledged that depressive symptoms during this period may reflect postpartum blues rather than depression (which is experienced by up to 80% of women in the first two weeks following birth; O'Hara, 1987). Therefore, a second time-point was included to measure depressive symptoms later in the postpartum period.

Time-point 3: Twelve Weeks Following Birth

As described in Chapter 1, the time-frame in which PPD can arise is a topic of considerable debate (Jones & Cantwell, 2010). Although the DSM-5 limits the diagnosis of PPD to the first four weeks following birth (American Psychiatric Association, 2013), others have argued that symptoms can arise up to one year postpartum (Gaynes et al., 2005). Research has suggested that the first three postpartum months are associated with the highest risk of PPD symptomatology (Cox, Murray, & Chapman, 1993; O'Hara, Neunaber, & Zekoski, 1984). With this in mind, it was decided that the third time-point would occur twelve weeks following birth. This would allow time for potential symptoms of PPD to develop.

Measurement of independent and dependent variables:

Prospective Versus Retrospective Measures of Sleep

It was important to consider whether variables would be measured retrospectively or prospectively. To reflect the prospective nature of the study, the study incorporated prospective measures of sleep, fatigue and PPD. The difference between prospective and retrospective measures has already been described in detail in the previous chapters, particularly in relation to sleep. To recap, since the perinatal period is a time during which women's sleep changes considerably over time due to hormonal and physical differences, it is important to use prospective measures. This ensures that women's sleep is a reflection of the specific time-point in which the measures were administered, rather than reflecting a general period of time. In addition, since retrospective measures require more evaluation and recall, women who develop symptoms of PPD may have a tendency to

report poorer sleep due to the negative thinking that is characteristic of depression (Beck, 1967).

Equally, it was important that the measure used to assess depressive symptomatology was prospective and time-specific, since the aim was to measure depression that specifically arose in the postpartum period. For example, if administered in the first week postpartum, a scale measuring symptoms over the past month would not provide an accurate representation of PPD symptoms.

Objective versus Subjective Measures of Sleep

Another objective of this thesis was to explore the relationship between objective sleep measures, subjective sleep measures and PPD. Therefore, sleep was measured both objectively, using PSG, and subjectively, using sleep diaries.

Type of comparison: This study used a combination of within and between group comparisons. Firstly, between-group analyses were carried out in order to investigate the difference in sleep between pregnant and non-pregnant women. A within-groups design was used in order to address the main research question assessing the prospective relationship between sleep and PPD throughout pregnancy and the postpartum period.

5.2 Measures

An overview of the different methods of measuring sleep and fatigue was provided in Chapter 1, followed by a short discussion of postpartum depression measures in Chapter 3. This section examines the measures that were used in this study. In order to address the study aims, it was necessary to use structured, validated measures for collecting complex data relating to subjective sleep, objective sleep, fatigue and PPD.

5.2.1 Sleep Measures

5.2.1.1 Objective Sleep: Polysomnography

Polysomnography was used in this study to address the following research questions:

- 1) How does sleep architecture differ in pregnant and non-pregnant women?
- 2) How accurate are pregnant / non-pregnant women in their subjective perceptions of sleep?
- 3) Do women's subjective perceptions of sleep quality relate to their sleep architecture?

4) Is there a relationship between sleep architecture during pregnancy and the development of postpartum depression?

As described in Chapter 1, there are two main methods by which sleep can be measured objectively: actigraphy and polysomnography. While actigraphy has been used in previous studies of sleep during pregnancy (e.g. Dorheim, Bondevik, Eberhard-Gran, & Bjorvatn, 2009; Dorheim, Bondevik, Eberhard-Gran, & Bjorvatn, 2009; Matsumoto, Kang, & Seo, 2003; Sharkey, Pearlstein, & Carskadon, 2013), it does not typically provide information of sleep architecture (referring to the proportion of time spent in each stage of sleep, and the overall distribution of sleep variables, particularly REM onset latency). This was of key importance in order to address the research questions in this study. Furthermore, actigraphy calculates sleep and wake cycles based on movement, and thus quiet wakefulness can be misinterpreted as sleep (Kushida et al., 2001). Therefore, in order to build upon the literature, it was decided that PSG (the 'gold standard' measure of sleep, Kushida et al., 2005) was necessary.

Overnight PSG was conducted at the participant's home in order to provide a naturalistic environment. Previous research has shown that first night effects (the tendency to experience poorer sleep on the first night of recording) are not apparent through home recording (Lee, Zaffke, & McEnany, 2000; Sharpley, Solomon, & Cowen, 1988). Spending a night away from home in the laboratory may also provide an opportunity for perinatal women to recover from cumulative sleep debt, which could result in better sleep quality than normal (Lee, 1998).

PSG was performed using the Embla Titanium device along with its associated software (REM logic, version 3.1). Signal measures included electroencephalography (EEG), electrooculography (EOG) and electromyography (EMG), as described in Chapter 1 (section 1.1). The device was supported by a holdall and Velcro strap that could be placed over the shoulder or around the chest (see Figure 5 for a photograph of a participant before the sleep study). Electrode placement was in accordance with the American Association of Sleep Medicine (AASM) Manual 2007 (Iber et al., 2007) and the 10-20 international electrode placement system (Klem, Lüders, Jasper, & Elger, 1999; see Figure 6). Two electrodes were placed on occipital, central and frontal locations, along with two ground electrodes. A reference electrode was placed on each mastoid. Disposable EOG electrodes were placed beside each eye (on one eye this was placed 1cm below the outer canthus, and on the other it was placed 1cm above the outer canthus). Following the

guidelines of the AASM manual, three EMG electrodes were positioned on the chin. Although not all of the channels used are needed for the scoring of sleep, using more electrodes increases the likelihood of obtaining good quality data. This was particularly important in this study since participants were sleeping in an uncontrolled environment and displaced electrodes could not be reattached.

Before attaching each electrode, the area of the scalp in which the electrode would be placed was carefully cleaned using an abrasive gel and acetone. A conductive gel was used to stick the electrode onto the scalp. This was followed by 'Collodian' glue and a small piece of gauze which was placed over the electrode and secured using 'Blenderm' tape. This process is the same as that carried out in two hospital-based sleep clinics that the researcher visited (St Thomas' Hospital, London and Queen Alexandra Hospital, Portsmouth, UK), and ensured that the data gathered in the study was of high quality. Once the electrodes were fitted, a bio-calibration was carried out to ensure that the signals were of good quality. The device was programmed so that it would start recording in the evening, at least half an hour before the participant stated was the very earliest time they would anticipate going to bed. It was programmed to stop at least half an hour after the participant anticipated waking up. These precautions were carried out to ensure that sleep onset and total sleep time could be accurately determined. Participants were given anonymous random data codes and sleep recordings were scored blind with respect to pregnancy status.



Figure 5. PSG study with a pregnant participant using the Embla Titanium (image provided with participant consent)

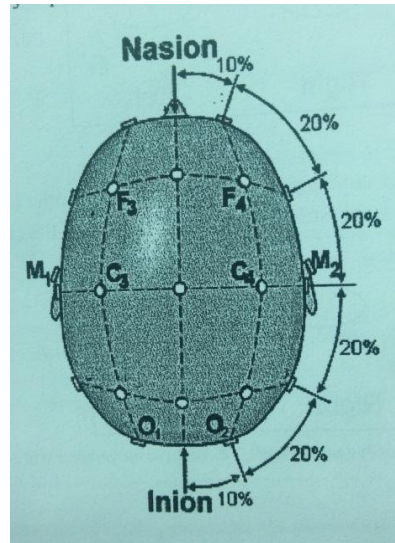


Figure 6. International 10-20 system of electrode placement (from Iber et al., 2007)

PSG recordings were scored in thirty second epochs, with a stage of sleep manually assigned to each epoch. The classification of each stage of sleep followed the well-established criteria outlined by AASM (Iber et al., 2007). These criteria are outlined below. An image of recording corresponding to data from each stage of sleep is provided.

Stage 'Wake' (see Figure 7)

An epoch was classified as 'wake' in the following conditions:

- More than 50% of the epoch had alpha rhythm (8-13 hertz) over the occipital region.
- If no alpha was present the epoch was scored as wake if any of the following were present:
 - a) Eye blinks at a frequency of 0.5 – 2 hertz.
 - b) Reading eye movements.
 - c) Irregular conjugate rapid eye movements associated with normal or high chin muscle tone.

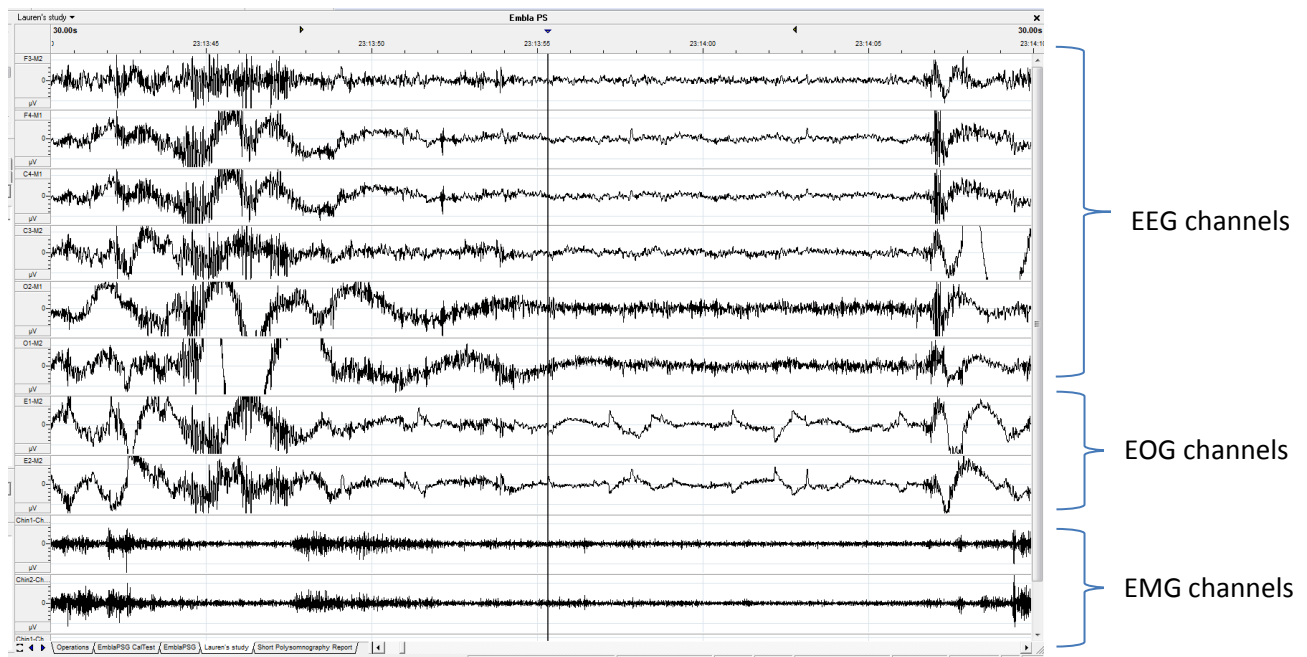


Figure 7. An example of an epoch scored as 'wake'

Stage 1 (see Figure 8)

- In participants who generated alpha rhythm, stage 1 was scored when alpha reduced and became replaced with low amplitude, mixed frequency activity for more than 50% of the epoch.
- In subjects who did not show alpha, stage 1 was scored in the presence of the following:
 - a) Activity in the range of 4-7 hertz with slowing of background frequencies by ≥ 1 hertz from stage 'wake'.
 - b) Vertex sharp waves.
 - c) Slow rolling eye movements, as measured by electrooculography (EOG)

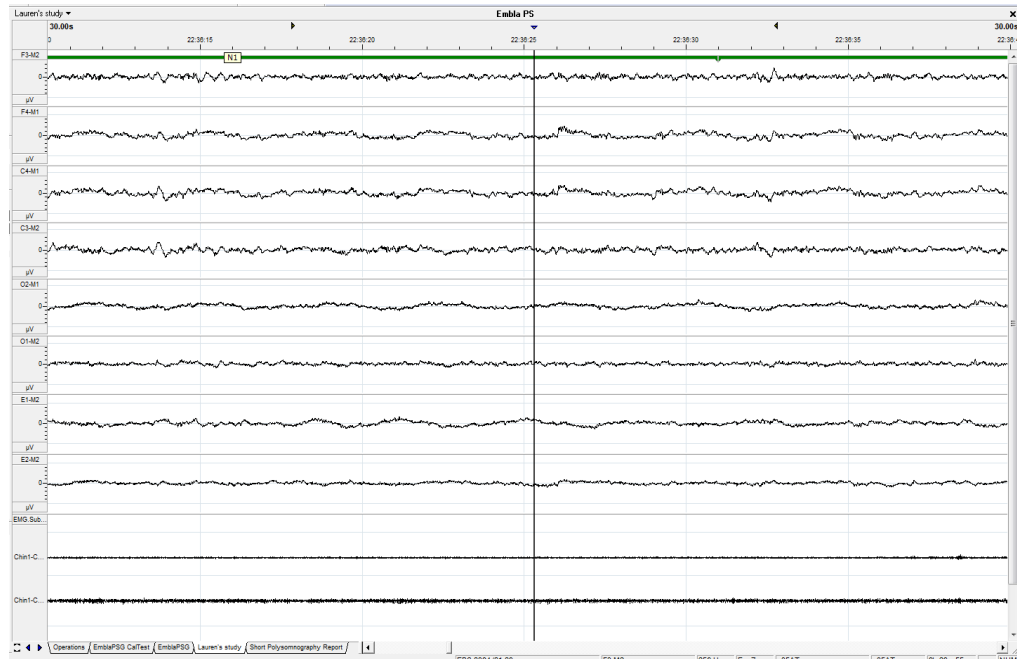


Figure 8. An example of an epoch scored as Stage 1

Stage 2 (see Figure 9)

- A period of stage 2 commenced:
 - a) If one or both of the following occurred during the first half of the epoch or the last half of the previous epoch:
 - One or more K complexes (a negative sharp wave immediately followed by a positive component; see figure 9b) not associated with arousals.
 - One or more trains of sleep spindles (a distinct train of waves from 11-16 hertz; see Figure 9a).
- Stage 2 ended under the following circumstances:
 - a) Transition to wake.
 - b) An arousal.
 - c) A major body movement followed by slow eye movements and low amplitude mixed frequency EEG without non-arousal associated K complexes or sleep spindles.
 - d) Transition to stage 3 or REM.

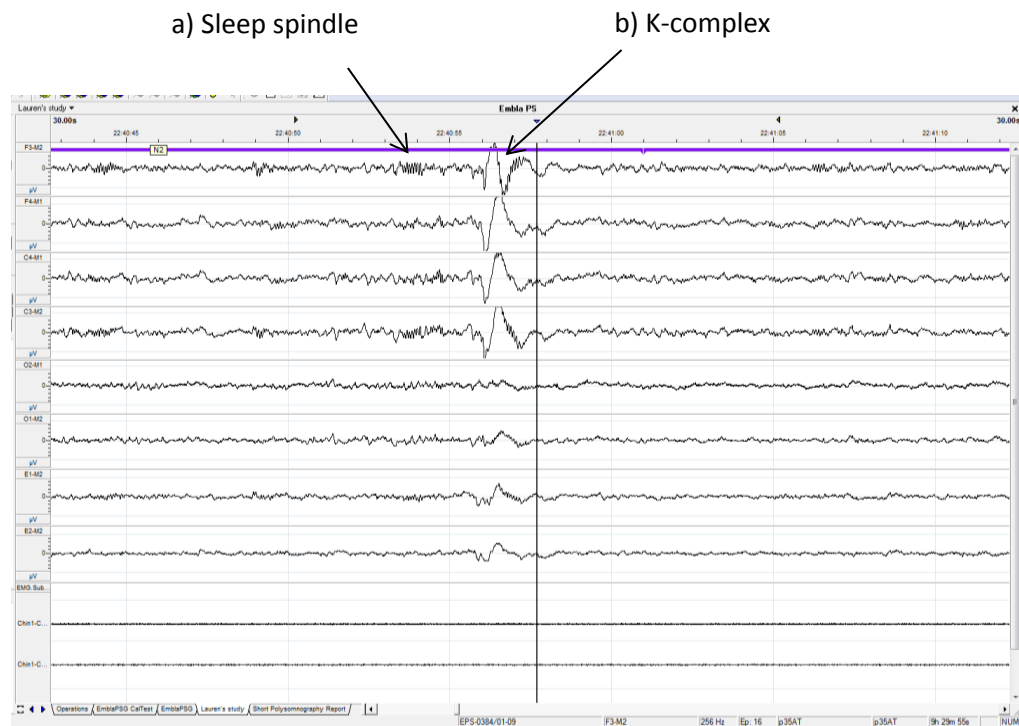


Figure 9. An example of an epoch scored as Stage 2

Stage 3 (see Figure 10)

Stage 3 was scored when at least 20% of the epoch consisted of slow wave activity (0.5-2 hertz with an amplitude of >75 microvolts, measured over frontal regions)

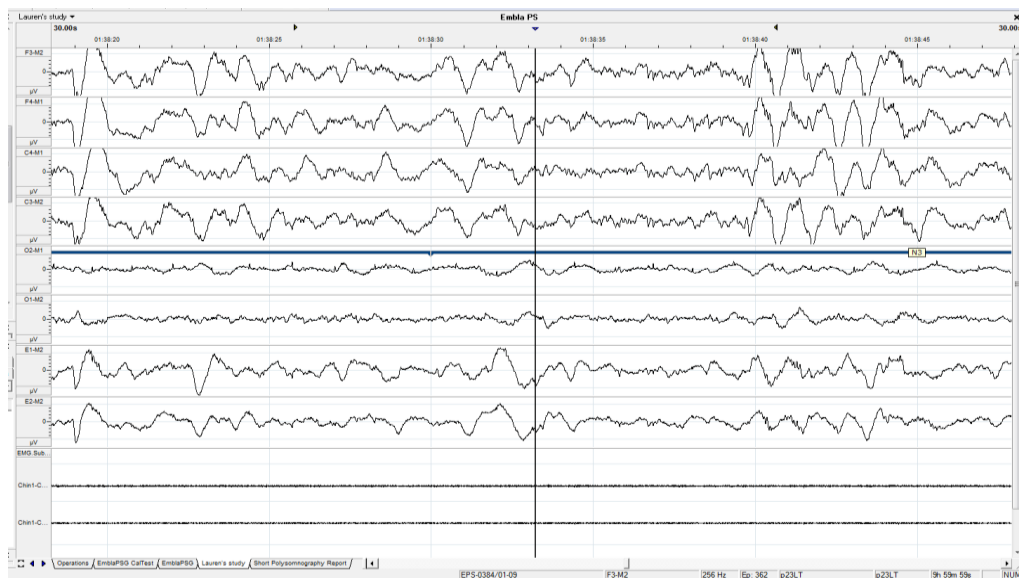


Figure 10. An example of an epoch scored as Stage 3

REM (see Figure 11)

- Stage REM was scored when the following criteria were met:
 - a) Low amplitude, mixed frequency EEG.
 - b) Low chin EMG tone.
 - c) Rapid eye movements.
- Stage REM was ended under the following circumstances:
 - a) Transition to wake or stage 3.
 - b) An increase in chin EMG tone and meeting criteria for stage 1.
 - c) An arousal occurred, which was followed by low amplitude, mixed frequency EEG.
 - d) A major body movement followed by slow rolling eye movements and low amplitude mixed EEG without K-complexes or spindles.
 - e) One or more non-arousal associated K-complexes or sleep spindles were present in the first half of the epoch without rapid eye movements, even if the chin EMG remained low.

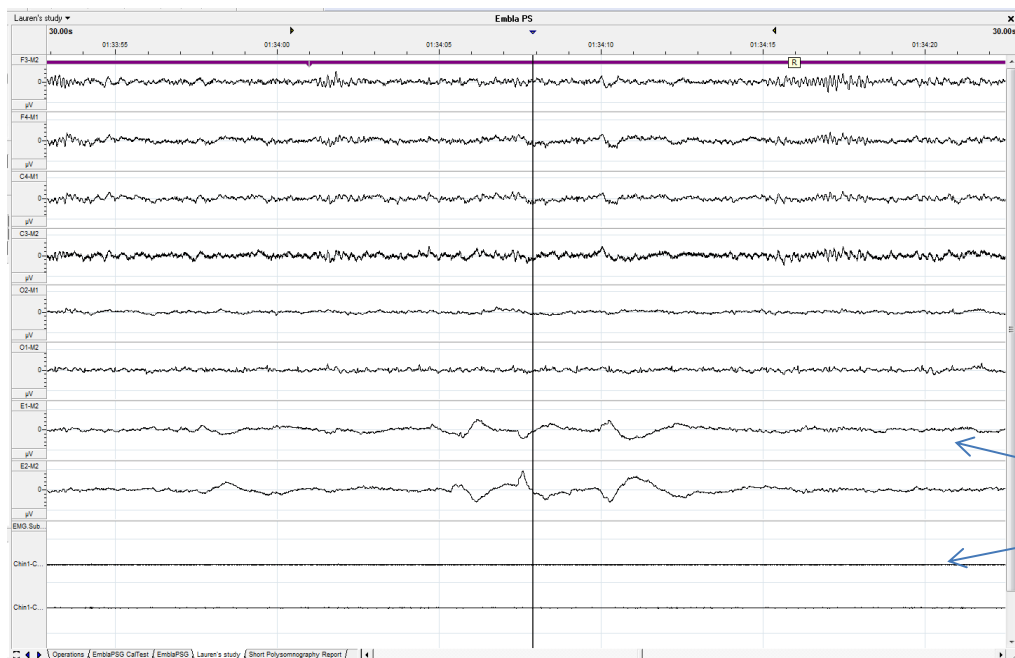


Figure 11. An example of an epoch scored as REM

Collating the PSG data

After scoring was complete, the REMlogic software produced a sleep report which included the following details: total recording time, wake after sleep onset (minutes), sleep onset (minutes), number of awakenings, sleep latency to each of the sleep stages (minutes), sleep period (lights off until lights on, minutes), total sleep time (minutes), sleep efficiency (total sleep time / time in bed, %), percentage and minutes spent in each stage of sleep (wake, 1, 2, 3, REM). Sleep onset latency (the time taken to fall asleep) was calculated from the time at which the participant reported 'lights out' to the first stage of stage 1 sleep. Arousals were scored using standardised criteria. The respiratory montage was not used because the study was interested specifically in EEG variables rather than detecting possible sleep disorders. After discussions with potential participants as well as several sleep experts, it was decided that the increased demands on participants of wearing the full montage outweighed the benefits. The occurrence of sleep disorders was assessed through a screening questionnaire, which is discussed later in the chapter.

The sleep report also included a hypnogram which provided a visual representation of sleep cycles throughout the night (see Figure 12). However, this was not specifically used in the data analysis.

Summary Graph

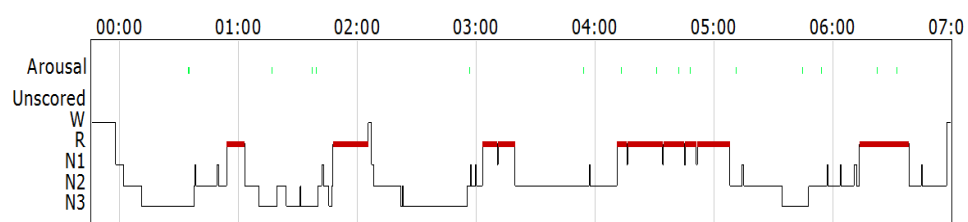


Figure 12. An example of a hypnogram showing the participant's sleep cycles. The stage of sleep is shown on the y-axis (W= wake, R= REM, N1, N2, N3 = stages 1, 2, 3) with time along the x-axis.

In the example hypnogram (Figure 12) the individual takes about 10 minutes to fall asleep, has a REM onset latency of about one hour, spends 4% of her sleep in stage 1 sleep, 43% in stage 2 sleep, 24% in stage 3 sleep and 29% in REM sleep. They only wake up once, and have a healthy sleep efficiency of 96%.

The specific objective and subjective sleep factors analysed in the study are detailed in Table 13. Previous studies have varied in whether they report sleep stage data in

percentage or minutes (e.g. Lee et al., 2000 versus Wilson et al., 2011). It was felt that expressing the data in percentage form was more meaningful and provided a standardised measure in which to compare between groups in the analyses (for the purpose of comparing sleep between pregnant and non-pregnant women). However, in order to address the specific sleep architectural factors that affected women's perceptions of their overall sleep quality, both minutes and percentage of time spent in each stage were included in this particular analysis to assess whether any differences emerged between these two forms of classification.

5.2.1.2 Subjective Sleep: St Mary's Hospital Sleep Questionnaire (SMHSQ; Ellis et al., 1981)

This study used the same subjective sleep questionnaire as that used in Chapter 4, therefore justification for this measure has been described previously (section 4.3.4). In the preliminary study, the analysis of specific sleep measures derived from this questionnaire was found to be effective at delineating which sleep aspects were most relevant to depression. Therefore, it was decided that this measure would be used in assessing the relationship between subjective sleep and PPD.

To recap, the SMHSQ consists of 14 items covering: the timing of sleep over the last 24-hour period (time of settling down for the night, falling asleep, waking up, getting out of bed), amount of night-time and daytime sleep, quality of sleep (depth, how 'well' slept, satisfaction), number of awakenings, morning clear-headedness, presence of early morning waking and not being able to return to sleep, difficulty falling asleep and sleep onset latency. Each question is scored individually, and respondents are asked to tick the box that most corresponds with their sleep. As in the previous study, 'sleep quality' was measured by combining the scores of the three items that specifically tap into the individual's perception of their sleep quality (depth, how 'well' slept, satisfaction). To provide further justification for this in the current study, the correlation between these measures was consistently high throughout the different time-points; ranging from $r=0.67$ - 0.92 , $p<.01$.

The importance of using a prospective, rather than retrospective questionnaire was discussed in section 5.1. An additional purpose of using a prospective measure of sleep is that it refers specifically to the previous night, and could therefore be used to address the relationship between subjective and objective measures, as well as the factors that affect

subjective sleep quality on a given night. The sleep factors derived from this questionnaire and used in subsequent analyses are described in Table 13. In addition to the sleep factors analysed in the preliminary study, the amount of time spent napping during the daytime was included in this study, since napping is common among perinatal women, and may account for a significant percentage of their total sleep time (Hunter, Rychnovsky, & Yount, 2009). It was also of interest to assess the relationship between daytime napping, sleep and depression.

Table 13. Subjective and Objective Sleep Measures Used in the Study

	Subjective Measures (SMHSQ)	Objective Measures (PSG)
Comparable subjective / objective sleep measures	Total sleep time Sleep onset latency Number of awakenings	Total sleep time Sleep onset latency Number of awakenings
Additional sleep measures	Sleep quality Daytime naps	Sleep efficiency % time spent in each stage (1, 2, 3, REM) REM latency

5.2.2 Fatigue: Multidimensional Assessment of Fatigue Scale (MAF; (Belza et al., 1993)

Following the strong relationship found between fatigue and depression in the preliminary study, the MAF was also used as a measure of fatigue in the main study. Details of this measure are provided in Chapter 4 (section 4.3.4). This scale has been validated for use in pregnant and postpartum women, with a high level of internal consistency and good convergent validity with measures of sleep quality and depression (Fairbrother et al., 2008). In addition, this scale was chosen over others as it specifically relates to feelings of fatigue during the previous week, corresponding with the time-frame of the subjective sleep measures.

5.2.3 Depression: Edinburgh Postnatal Depression Scale (Cox et al., 1987)

Various different tools exist for measuring depression within the general public. These include the Beck Depression Inventory II (used in the preliminary study; BDI-II, Beck, Steer, & Brown, 1996), the Hospital Anxiety and Depression Scale (HADS, Zigmond & Snaith, 1983), the Hamilton Rating Scale for Depression (HAMD, Hamilton, 1960), the Centre for Epidemiological Studies Depression Scale (Radloff, 1977) and the Patient Health Questionnaire (PHQ-9, Kroenke, Spitzer, & Williams, 2001). Although these measures have been used in some studies examining the relationship between sleep and PPD (e.g. Goyal, Gay, & Lee, 2007; Huang, Carter, & Guo, 2004), Chapter 1 described the features of PPD that make it distinctive from major depressive disorder. This has led to the formation of specific PPD scales, including the Postpartum Depression Screening Scale (PDSS, Beck & Gable, 2000) and the Brisbane Postnatal Depression Index (Webster, Pritchard, Creedy, & East, 2003).

The PDSS is a 35-item Likert response scale which contains seven dimensions, including sleeping/eating disturbances, anxiety/insecurity, emotional lability, cognitive impairment, loss of sleep, guilt/shame and contemplating harming oneself. The mother is also asked to rate her degree of disagreement or agreement with statements relating to how she has been feeling over the past two weeks. In contrast, the Brisbane Postnatal Depression Index is a prospective measure, identifying 'at risk' women, rather than those who are currently depressed. The most well-recognised of measure of PPD, however, is the Edinburgh Postnatal Depression Scale (Cox et al., 1987).

The EPDS is a screening tool used to detect PPD through a series of 10 statements relating to how the mother has felt in the past seven days. The symptoms of depression measured in the scale include not being able to see the funny side of things, not looking forward to things with enjoyment, unnecessary self-blame, being anxious or worried for no good reason, feeling scared or panicky for no good reason, feeling that things have got 'on top of me,' being so unhappy that sleep has been difficult, feeling sad or miserable, crying, and thoughts of self-harm. The EPDS specifically focuses on emotional and cognitive rather than somatic symptoms, since the latter could be misleading in new mothers given that they may overlap with normal processes associated with motherhood (e.g. changes in weight, Cox et al., 1987). Reactions to the statements are measured on a four-point scale (ranging from 0 to 3), with a total possible score of 30. The EPDS is the most frequently researched method to identify PPD and has been translated and validated

in multiple different languages (Hewitt, Gilbody, Mann, & Brealey, 2010). It has also been validated and used as a measure of antenatal depression (Adouard, Glangaud-Freudenthal, & Golse, 2005; Figueiredo, Pacheco, & Costa, 2007; Ryan, Milis, & Misri, 2005).

An early study comparing the use of the BDI-II and the EDPS to identify depression in 147 postpartum mothers reported sensitivity and specificity of 95% and 93% in the EPDS, and 68% and 88% in the BDI-II, respectively (Harris, Huckle, Thomas, Johns, & Fung, 1989). The authors concluded that the EPDS is substantially superior to the BDI-II in detecting PPD. A further review of eight different instruments for the screening of PPD, including the BDI, EPDS, PDSS and the Zung self-rating Depression Scale, found that the EPDS was the most widely studied measure with moderate psychometric soundness (Boyd et al., 2005). A problem with measuring PPD using depression scales that are not specific to the postpartum period is that some of the items measured in these scales, such as changes in weight, fatigue and sleeping problems, are likely to confound with normal postpartum adjustment (Beck & Gable, 2001).

In a study comparing the PDSS, EPDS and BDI-II, Beck and Gable (2001) asked 150 new mothers to complete each of the scales, followed by a DSM-IV diagnostic interview. The authors found that the PDSS had the highest sensitivity and specificity, identifying 94% of the women diagnosed with major depression, while the EPDS identified 78% and the BDI-II identified 58%. A further study of 60 New Zealand women similarly reported that the PDSS provided a better representation of PPD when compared to the EPDS. However, the authors commented that although the PDSS may be preferable, it is not freely available like the EPDS, and may therefore be more difficult to use in practice. They stated that the EPDS remains a valid and reliable tool that can continue to be used with confidence.

Given the previous comparisons between measures, it was decided that a measure specific to the postpartum period was necessary. The three postpartum scales were the EPDS, the PDSS and the Brisbane Postnatal Depression Index. Since the Brisbane index is a prospective measure, used to predict PPD rather than measure current symptoms, this left the PDSS and the EPDS. It was felt that while the PDSS provides a well-validated measure of PPD, the amount of time that would be required to complete this questionnaire, alongside the sleep and fatigue measures, would be too demanding for participants. The ease of obtaining the EPDS along with its extensive use in previous research and low

demands on participants (10 questions compared to 35 on the PDSS) led to this being chosen as the measure of PPD.

Various cut-off scores have been proposed for the EPDS. Cox et al. (1987) found that a cut-off of 12/13 identified 21 out of 84 women who had received a clinical diagnosis of major depressive disorder, with a sensitivity of 86% and specificity of 78%. Similarly, a review article exploring EPDS cut-off points concluded that a score of ≥ 13 is optimal for postpartum depression while a score of ≥ 15 is optimum for antenatal depression (Matthey, Henshaw, Elliot, & Barnett, 2006). Others have found that a score of ≥ 10 has sensitivity of 84% to 100% and specificity of 76% to 88%, when compared to a clinical diagnosis of minor and major depression (Harris et al., 1989; Murray & Carothers, 1990; Zerkowitz & Milet, 1995). In addition, Hannah, Adams, Lee, Glover and Sandler (1992) found that, of 25 women who had EPDS scores ≥ 13 at 6 weeks postpartum, 17 of these women had scores ≥ 10 in the first postpartum week. A score of ≥ 10 has also been used in prior studies investigating the relationship between sleep and PPD (Da Costa, Dritsa, Rippen, Lowensteyn, & Khalifé, 2006; Dorheim et al., 2009; Dorheim et al., 2009).

A systematic review by the UK National Screening Committee (Hewitt et al., 2009) suggested that an optimal cut-off point of 12 should be used for major depression, and 10 to encapsulate both major and minor depression. However, in order to maximise sensitivity from a clinical perspective, they recommended a cut-off score of 7 for major depression and 8 for major and minor depression. The decision about an appropriate cut-off should therefore be dependent upon several factors, including the type of study, the culture, the severity of symptoms and whether or not it is being used in clinical or research settings.

While it is acknowledged that the EPDS is a primarily a screening method and does not provide a clinical diagnosis, the purpose of this study was to identify risk factors for PPD, rather than to diagnose and treat women with PPD. Furthermore, the EPDS is commonly used to detect symptoms of PPD within UK healthcare settings (Hewitt et al., 2009) and is therefore a valid measure for healthcare practitioners. Given the nature of the study, and in line with previous research investigating sleep and PPD (Da Costa, Dritsa, Rippen, Lowensteyn, & Khalifé, 2006; Dorheim et al., 2009; Dorheim et al., 2009), the current study used a cut-off score of ≥ 10 to classify PPD.

5.2.4 Additional Questionnaires

Alongside the main measures, participants were also required to complete two demographic questionnaires and a screening questionnaire. These are described in the subsequent sections.

5.2.4.1 Socio-Demographic Questionnaires

Participants were asked to complete a socio-demographic questionnaire at the start of the study, and once their baby had arrived (see Sections 5.11.2-3). The purpose of these questionnaires was to explore the sample characteristics, and to investigate additional risk factors for PPD. The first of these was carried out during time-point 1, in the woman's third trimester of pregnancy. This questionnaire included items relating to age, parity, gestational weeks, marital status, level of education, marital status, relationship satisfaction and family history of depression. Participants were asked to complete a second questionnaire at time-point 2, during the first postpartum week. This questionnaire included questionnaires relating to type of birth, gender of baby, type of feeding, infant sleeping location and habits, level of social support and use of antidepressant medication. The factors measured in these questionnaires are a combination of those that should be considered when examining perinatal sleep (Hunter et al., 2009), and those that have been previously established as risk factors for PPD (Gale & Harlow, 2003).

5.3 Sample Selection: Exclusion Criteria

Pregnant participants were excluded if they met any of the following criteria:

- 1) Currently experiencing a current episode of major depression or any other psychiatric disorder.
- 2) Currently diagnosed with a sleep disorder (e.g. insomnia, sleep apnoea).
- 3) Currently taking sleep medication.
- 4) Currently taking antidepressant medication.
- 5) Experiencing pregnancy complications.
- 6) Not English language proficient (oral, read, write).
- 7) Unable to give informed consent.

Rationale for Exclusion Criteria

In order to investigate whether sleep disturbances preceded the development of PPD symptomatology, it was important that women were not experiencing a current episode of depression at baseline. Failing to control for depression at baseline was a major limitation of some of the studies described within Chapter 3. In addition, participants were not eligible to participate if they had any current or historical diagnosis of psychiatric illness, other than major depressive disorder. Although participants were excluded if they were experiencing a current depressive episode, they were not excluded if they reported experiencing a prior history of depression. Prior history of depression increases the risk of PPD (Robertson et al., 2004), therefore increasing the likelihood that some of the sample would develop postpartum depressive symptoms. Participants were, however, excluded if they were taking current antidepressant medication, since these may have implications for both subjective and objective sleep (Mayers & Baldwin, 2005). They were also excluded from taking part in the study if they were currently diagnosed with a sleep disorder, as this would also be likely to interfere with their sleep, or if they were regularly taking sleep medication.

5.4 Participant Recruitment

Participants were recruited through a number of different sources. Information regarding the final sample is provided later in this chapter.

1) Recruitment via the NHS

Initially, it was anticipated that the majority of participants would be recruited via this route, and so this was the first-line method of recruitment. Subsequent to ethical approval, the researcher met with several different midwives from local NHS trusts, who agreed to assist with recruitment by handing out advertisements to participants. The researcher also regularly attended antenatal clinics at Royal Bournemouth Hospital, in order to inform pregnant women of the study, and to hand out relevant information. Advertisements were also placed in the majority of general practice surgeries in the local area, subsequent to managerial approval.

2) Local Community

The researcher also researched local venues and groups attended by pregnant women. This included visiting a number of yoga and birth-preparation classes. From the contacts

established through this route, the researcher was also invited to place an advert in a local monthly parenting magazine. Leaflets for the study were placed in a number of local venues including cafes, shops and play centres.

3) Online

Given that recruitment via the above two methods had been slow, the study was advertised online via a number of social networking and community websites, including 'Facebook,' 'Netmums' and 'Gumtree'. This method turned out to be an effective form of advertisement, recruiting the majority of study participants. This highlights the utility of social media as a source of recruitment within research of this nature.

4) Coffee Morning and Word of Mouth

A coffee morning at a soft play centre was arranged in order to thank the women who had already taken part in the study, and to invite other women in the community who were interested in finding out more. This also provided an opportunity for women to meet other new mothers in the community. Given the relationships formed with participants, several also referred pregnant friends who subsequently took part.

5.5 Study Location

The primary location of the study was in the local areas of Bournemouth and Poole. This was due to the fact that the researcher had to visit the participants in their home (in the evenings) to carry out the sleep study. However, in order to maximise recruitment, participants were also recruited from East Berkshire, West Hampshire and Surrey.

5.6 Screening Tools

In order to assess eligibility, potential participants were asked to undergo two forms of screening: a screening questionnaire and a telephone screening interview.

5.6.1 Screening Questionnaire

Participants that were interested in taking part in the study were asked to complete a screening questionnaire (see Appendix 2). This asked questions relating to current and previous history of psychiatric illness, sleep disorders and medications. This was considered the 'first line' of screening.

5.6.2 The MINI International Neuropsychiatric Interview

After completing the screening questionnaire, eligible participants were also required to undergo a further form of screening, via the Mini International Neuropsychiatric Interview version 6.0 (MINI; Sheehan et al., 1998). This is a structured screening interview designed to act as a shorter replacement for the Structured Clinical Interview for DSM-IV Diagnosis, which was the current version in operation at the time of the study (SCID; Spitzer, Williams, Gibbon, & First, 1992). It covers nineteen different psychiatric disorders. The MINI has high validity and reliability and takes around fifteen minutes to complete (Sheehan et al., 1998). According to a well-known database, the Institute for Scientific Information (ISI) Web of Knowledge, the MINI has been cited in over 2,000 published journal articles. It has also been used in previous studies exploring sleep and PPD (Posmontier, 2008). The purpose of this screening was to ensure that participants were not currently experiencing symptoms indicative of psychiatric illness, and that they had no history of psychiatric illness except from major depression. This interview was conducted over the telephone by one of the researcher's supervisors who has received formal training and has extensive experience of assessing patients using this measure.

5.7 Procedure

Potential participants were provided with an information sheet (see Appendix 3) and were invited to discuss the study either face-to-face, via telephone or email. Following this, they were asked to complete the short screening questionnaire. If they met the inclusion criteria, participants were then asked to undergo the MINI screening interview, which was conducted over the telephone by the supervisor. If participants were eligible to take part, a date was arranged for the overnight sleep study, once they had reached the third trimester (28 weeks plus). For the PSG sleep study, the researcher visited the participant in their home in the evening, accompanied with a research assistant. On the day of the study, participants were asked to restrain from caffeine (i.e. tea, coffee and any other caffeine-containing substances). The procedure for setting up the PSG was described in section 5.2.1.1. Prior to this, the researcher discussed the procedure and invited the participant to ask any additional questions, before taking informed consent (see Appendix 4). The participant was provided with a solution and instructions on how to remove the electrodes the next morning.

The participant was then given three packs of questionnaires, which contained the measures for each study time-point. Pre-paid, addressed envelopes were included, and participants were asked to return each pack via post after completion. An overview of the measures at each time-point is provided in Figure 13. The researcher returned in the morning to collect the equipment. A note of the participants due date was made, and they were subsequently contacted near to this date to check progress. Reminders were sent out via telephone and email at each of the time points.

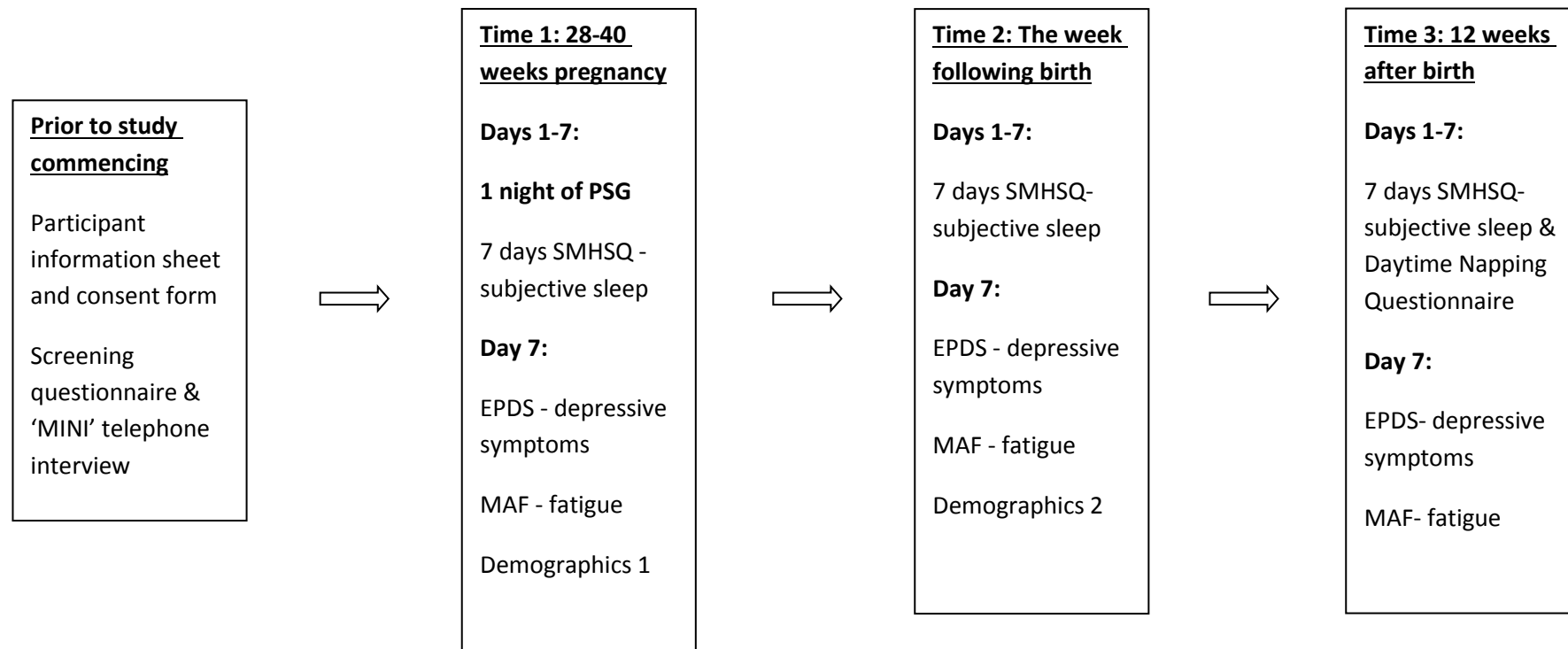


Figure 13. Summary of measures taken at each time-point of the study

5.8 Non-Pregnant Control Group

A control group of non-pregnant women were recruited in order to examine differences in sleep between pregnant and non-pregnant women, and whether pregnant women were less accurate in their subjective sleep perceptions. The same inclusion criteria were applied to this sample as previously described, with the exception that these women had not given birth within the previous year. The sample consisted of female adults of childbearing age, which was considered 18 to 45 years. The procedure for the control group was as follows.

Potential participants were given a participant information sheet and were invited to discuss the study with the researcher. They were then asked to complete a screening questionnaire similar to that in the pregnancy sample in order to assess whether they met the inclusion criteria. In order to match the pregnant sample, participants were not eligible if they were currently depressed. The stringent screening in the main study was necessary because the aim of the study was to examine the longitudinal relationship between sleep and depression, however, since the control group were only taking part in the cross-sectional element of the design, it was felt that a validated screening questionnaire was adequate to meet the demands of the study. Therefore, the Hospital Anxiety and Depression Scale (HADS, Zigmond & Snaith, 1983) was chosen as a screening measure for current depression.

The HADS is a well-validated and commonly used measure that has been applied to both clinical and general population samples (Bjelland, Dahl, Haug, & Neckelmann, 2002). A review article examining over 750 papers using the HADS concluded that the scale has at least as good screening properties for anxiety and depression as more comprehensive instruments (Bjelland et al., 2002). A benefit of the scale over others is that it is very quick to complete. The HADS is formed of two subscales, measuring anxiety and depression. For the purpose of this study, only the depression subscale was analysed. The depression scale consists of seven questions relating to how the individual has been feeling over the past week. Each answer is measured on a scale of 0-3 (0 represents absence of symptoms, 3 represents a high level of symptoms). The highest possible score for each subscale is 21. A depression cut-off score of ≥ 8 is the most frequently achieved cut-off for achieving high sensitivity and specificity (Bjelland et al., 2002) and has been shown to have sensitivity and specificity of 0.90 in a community sample, based on ICD-9 diagnosis (Abiodun, 1994). This cut-off point was used to screen for depression in the study.

Subsequent to screening, a date was arranged for the participant to take part in the PSG sleep study. Written informed consent was taken from each participant before the study. The procedure for setting up the PSG recording was the same as that described previously. As with the pregnant group, the non-pregnant control group were asked to complete the SMHSQ as a measure of subjective sleep, upon rising. This was used to compare with the PSG data, in order to address the aim of whether or not pregnant women were less accurate in their subjective sleep perceptions, compared to non-pregnant women.

5.9 Ethical Issues

The American Psychological Association (2002) describes five key principles that should inform psychological practice and research. These are: beneficence and nonmaleficence, fidelity and responsibility, integrity, justice and respect for people's rights and dignity. These are also the ethical guidelines adopted by the NHS, and therefore followed in the current study. Descriptions of each of these principles as well as their relevance to the current study are provided below.

Beneficence and Nonmaleficence: This principle refers to the need for psychologists to do no harm and to strive to benefit those that they work with. There is a need to safeguard and protect the welfare and rights of participants and to minimise risks or potential harm.

There were no anticipated risks associated with taking part in the study. However, the researcher ensured that each participant fully understood the full details of the study before taking part. It was important that the researcher explained that the PSG posed no threat to the women or their foetuses, since this was of concern to some participants. Participants were not offered payment for the study, but were offered a copy of their sleep reports if they wished. Benefits of the study were explained through the importance of research of this nature in order to better identify women who are at risk of developing postpartum depression. It was acknowledged that completing the study may raise the participant's awareness of their mental health. However, since it is common for perinatal women to be asked these types of questions by health visitors, it was felt that this was acceptable and in line with clinical practice. The participant information sheet contained signposts for where participants could find further information on sleep and mental health.

The participant information sheet provided details relating to each aspect of the study, including questions relating to the procedure, confidentiality, risks and benefits of taking

part and contact details of the researcher. Participants were told that they could withdraw from the study at any time, without needing to give reason.

Fidelity and Responsibility: This principle relates to the need for psychologists to establish trusting relationships with those that they work with, and to be aware of professional and scientific responsibilities. Professional standards should be upheld at all times. In order to serve the best interests of participants, it may be appropriate to consult with, or refer to, other relevant professionals or institutions.

In the process of designing the study, the researcher liaised with a number of experts within the fields of perinatal mental health and sleep. Of particular importance was the consideration of what would occur if participants reported significant mental health problems, either through the screening questionnaire, telephone interview or EPDS. The researcher felt that it was appropriate that if a participant showed symptoms of severe mental illness, as reported by the MINI screening interview, they would be referred to their relevant healthcare provider. This was also applied if a participant responded with a positive answer to question 10 on the EPDS ('the thought of harming myself has occurred to me').

Integrity: This refers to the need for psychologists to act with honesty, accuracy and truthfulness. This includes not stealing, cheating, misrepresenting information and avoiding unclear commitments.

The study did not include any deception, and the researcher ensured that participants were clear about the nature of the study before taking part. The process of informed consent was carefully considered throughout the study. The researcher ensured that the participant had adequate time to read information about the study and to ask questions before giving written consent.

Justice: This refers to acting with fairness and ensuring equal justice to all people, and recognising that all people should be able to access and benefit from the contributions of psychology. Psychologists must use judgement and take precautions to ensure that they work within the boundaries of their competence.

Participants were invited to be notified when publications arose from the study, as this was something that many participants expressed an interest in. The researcher also

ensured that they had good links with local perinatal mental health services, so that they could be contacted if any issues arose that were beyond the researcher's capacity.

Respect for People's Rights and Dignity: The last principle relates to respecting the dignity and worth of all individuals, and the rights to confidentiality, privacy and self-determination. Psychologists should respect the dignity and worth of all people, and the rights of individuals to privacy, confidentiality, and self-determination. It also refers to the need to respect differences of age, culture, religion, nationality and socio-economic status.

All participant data in the study were given identified by a unique ID number. Participant names and details were not written on any of the measures. All research data was kept in a secure locked cabinet at the university, of which only the researcher had access. Confidentiality would only be broken if the researcher deemed the participant or themselves to be at serious risk, such as if severe mental illness was detected, as previously described.

Since the study involved a clinical population who were currently receiving care from the National Health Service, the researcher was required to obtain ethical approval through the Integrated Research Application System (IRAS). As part of this, the researcher attended a course on 'Good Clinical Practice,' which outlined the ethical and scientific quality standards that apply in clinical research. At the time of obtaining ethical approval, the application was governed by the local research ethics committee (Southampton A, study reference number 11/SC/0158). Subsequent to obtaining national approval, the researcher was also required to obtain Research and Development (R&D) approval from each of the areas in which recruitment would occur. This ensured that no burden was placed upon staff at these sites, and that no extra costs were experienced as a result of the study. The researcher received a letter of access for each of the sites, which enabled her to recruit participants in antenatal units. R&D approval was obtained from Royal Bournemouth and Christchurch NHS Foundation Trust, Poole Hospital NHS Foundation Trust, Dorset Healthcare University Foundation Trust and Frimley Park Hospital NHS Foundation Trust.

The process of obtaining ethical approval was time-consuming, taking almost one year. Although this was unavoidable, it subsequently limited the time-frame in which participants could be recruited onto the longitudinal study.

5.10 Statistical Considerations

Details of each statistical analysis are described at the start of each results section. Data were analysed using the Statistical Package for Social Sciences (SPSS, IBM, version 19). To summarise, descriptive statistics were used to describe the participant characteristics. Between-group analyses (independent t-tests and one-way ANOVAs) were used to compare sleep variables between pregnant and non-pregnant women. Correlations were used to examine the relationship between subjective and objective sleep variables. Data were checked for normality using Shapiro-Wilks tests, and if significant, z-scores of skew and kurtosis were examined. Non-parametric tests were used where appropriate. Repeated-measures t-tests and one-way ANOVAs were used to examine changes in perinatal women's sleep, fatigue and depression across time. Correlations were carried out to examine relationships between sleep, fatigue and depression at each time-point, as well as prospectively. Based on a limited sample size and the number of correlations examined, interpretation of correlations was based upon Cohen's effect sizes, whereby 0.3 is considered a medium effect size and 0.5 is considered a large effect size (Cohen, 1988). Based on patterns of correlations, multiple linear regression analyses were used to examine the variables that predicted postpartum depression.

5.11 Sample Characteristics

This chapter describes the demographic details of the participants that took part in the study.

5.11.1 Pregnant Sample

Overall 29 pregnant women were recruited into the longitudinal study. This sample size is in line with previous studies that have used polysomnography to investigate sleep and postpartum depression (e.g. Lee, McEnany & Zaffke, 2000, $n=31$, Coble et al., 1994; $n=34$, Godfroid, Hubain, Dramaix & Linkowski, 1997, $n=28$), as well as those using actigraphy, which is a less demanding measure (e.g. Tsai & Thomas, 2012, $n=22$, Lee & Kimble, 2009, $n=20$). However, what makes this study unique over others is that it not only uses polysomnography and has three time-points, but that it also uses measures of subjective sleep and fatigue to provide a comprehensive view of the relationship between perinatal sleep and depression.

5.11.2 Demographics of Pregnancy Sample

The average age of participants was 31.28 years (range = 25-42, SD= 4.61) and the mean number of gestational weeks was 32.33 (range = 28-38, SD= 3.37). The majority of women were employed, although most women (excluding two) had finished working by the time of the study. As can be seen in Table 14, roughly half of the participants were primiparas (first time mothers). The majority were married or living with their partner, and all participants reported being very content or content with their relationship.

Table 14. Demographics of the Pregnancy Sample

	Variable	Number	Percentage
Parity	Primiparous	15	51.7
	Multiparous	14	48.3
No. of children	0	15	51.7
	1	11	37.9
	2	2	6.9
	3	1	3.4
Occupation	Employed	19	65.5
	Self employed	2	6.9
	Home-maker	7	24.1
	Unemployed	1	3.4
History of depression	Yes	6	20.7
	No	23	79.3
Family history of depression	Yes	9	31.0
	No	20	69
Marital status	Single	2	6.9
	Living with partner	12	41.4
	Married	15	51.7
Relationship satisfaction	Very content	24	82.8
	Content	5	17.2
Highest level of education	Secondary school	3	10.3
	Some additional training (e.g. apprenticeship)	10	34.5
	Undergraduate university	9	31.0
	Postgraduate university	7	24.1

5.11.3 Postpartum demographics

Characteristics of the participants in the postpartum period are described in Table 15. Five women did not complete week 1 postpartum measures and are therefore not included in the table. A further five women did not complete week 12 measures. The data of these women was excluded from the final longitudinal analyses reported in Chapters 7 and 8. The majority of women had a vaginal birth, and all women reported breast feeding their infant. Three quarters of the sample reported that their infant slept in a separate bed in the same room, while a quarter co-slept with their infant. All but two women reported that they were receiving practical help at home, and the majority reported that their baby was sleeping more than four hours a night. An equal number of women gave birth to a girl and a boy. By week twelve the majority of women reported that they had not yet returned to work.

Table 15. Postpartum Demographic Information

	Variable	Number	Percentage
Type of birth	Vaginal	16	66.7
	Caesarean	8	33.3
Type of feeding	Breast	20	83.3
	Bottle	4	16.7
Infant sleeping > 4 hours a night	Yes	16	66.7
	No	8	33.3
Infant gender	Girl	12	50.0
	Boy	12	50.0
Practical help at home	Yes	22	91.7
	No	2	8.3
Baby sleeping location	Co-sleeping	6	25.0
	Separate bed in room	18	75.0

5.11.4 Non-Pregnant Sample

The control group comprised of 24 non-pregnant women, with a mean age of 28.21 years (19-43, SD= 6.98). This group were recruited to compare differences in sleep, measured using PSG, in pregnant and non-pregnant women. Of this sample, 18 (75%) were primiparous and 6 (25%) were multiparous. Only three women (12.5%) reported a personal history of depression, and three women (12.5%) also reported a family history of depression.

5.12 Chapter Summary

This chapter described the methodology used in the study and provided relevant justifications. It also provided a description of the sample. The next part of the thesis details the results of each different part of the study.

PREFACE TO CHAPTERS 6, 7 AND 8

The following three chapters of this thesis describe the results of the main study. Given that this was a large study which addressed several aims, the results are described in three parts. These are outlined below.

Chapter 6: Sleep in Pregnant and Non-Pregnant Women

Chapter 6 describes the cross-sectional part of the study comparing subjective and objective sleep in pregnant and non-pregnant women, using between-group analyses. The use of a non-pregnant control group in this part of the study allowed for the specific comparison of sleep architecture in pregnant and non-pregnant women, and assessment of whether the groups differed in their accuracy of subjective sleep perceptions. It also examines the factors that affect subjective sleep quality.

Chapter 7: Longitudinal Changes to Sleep, Fatigue and Depression throughout the Perinatal Period

Chapter 7 describes longitudinal changes in sleep, depression and fatigue across the three time-points in the study, using repeated measures analyses. The purpose of this chapter is to examine how these factors changed over time, and whether similar patterns of change emerged between the variables

Chapter 8: Relationships between Sleep, Depression and Fatigue throughout the Perinatal Period

Finally, Chapter 8 examines the cross-sectional and longitudinal relationships between subjective sleep, objective sleep, fatigue and depression. The aim of this analysis was to identify the variables that were most strongly and consistently related to postpartum depression scores. Based on this, the variables most consistently related to week 1 and 12 postpartum depression scores were entered into stepwise regression models to tease apart which variables were most predictive of postpartum depression scores.

CHAPTER 6: SLEEP IN PREGNANT AND NON-PREGNANT WOMEN

The aims of this part of the study were to examine differences in sleep between third trimester pregnant women and non-pregnant women and to explore the relationships between subjective and objective measures of sleep. Both pregnant (n = 29) and non-pregnant (n = 24) women underwent one night of home polysomnography and completed the St Mary's Hospital Sleep Questionnaire upon waking. Results showed that pregnant women experienced poorer sleep, both objectively and subjectively. In terms of sleep architecture, pregnant women had significantly poorer sleep efficiency, more stage 1 and 2 sleep and less REM sleep. However, no significant differences were found in relation to slow-wave sleep. Although pregnant women were more accurate in estimating number of awakenings, they were significantly less accurate in estimating their total sleep time. Furthermore, number of awakenings was the factor that most strongly related to subjective sleep quality, suggesting that this is what drives reports of poor sleep amongst women in late pregnancy.

6.1 Introduction

As described in Chapter 1 (section 1.6), pregnancy is a time during which sleep changes are common. Sleep can be disrupted by a number of physiological and physical changes. For example, hormonal changes may affect sleep architecture (Wilson et al., 2011), while physical changes can result in less comfortable sleep and more awakenings (Baratte-Beebe & Lee, 1999; Wilson et al., 2011). Research suggests that women experience poorer sleep during the third trimester compared to other trimesters (Baratte-Beebe & Lee, 1999; Hedman, Pohjasvaara, Tolonen, Suhonen-Malm, & Myllylä, 2002; Lee, Zaffke, & McEnany, 2000; Schweiger, 1972).

The extent to which sleep problems are reported during pregnancy has prompted the inclusion of 'pregnancy-associated sleep disorder' as a diagnosis within the International Classification of Sleep Disorders (American Association of Sleep Disorders, 1997). However, the extent to which sleep changes that occur during pregnancy are 'normal' remains unclear (Santiago et al., 2001). A recent review found that poor sleep during pregnancy was related to longer labour, more pain and discomfort during labour, higher rates of preterm delivery and caesarean section (Chang, Pien, Duntley, & Macones, 2010). This suggests that sleep disruptions should be given more attention. However, despite the

potential adverse outcomes of poor sleep, the effects of pregnancy on sleep architecture remain unclear.

Recent years have seen an increase in the number of studies within this area (e.g. Jomeen & Martin, 2007; Ko, Chang, & Chen, 2010; Signal et al., 2007; Wilson et al., 2011), however, fewer than ten studies have used examined women's sleep during pregnancy using polysomnography (PSG, Brunner et al., 1994; Coble et al., 1994; Driver & Shapiro, 1992; Hertz et al., 1992; Karacan et al., 1968; Lee et al., 2000; Wilson et al., 2011). Of these, only three have recruited a control group of non-pregnant women (Hertz et al., 1992; Karacan et al., 1968; Wilson et al., 2011). Furthermore, small sample sizes and varied methodologies have resulted in conflicting findings regarding the effect of pregnancy upon sleep architecture (see Chapter 1, section 1.5.4). For instance, percentages of slow-wave sleep range from 9-28% in the first trimester, 10-36% in the second trimester, and 8-36% in the third trimester (Brunner et al., 1994; Coble et al., 1994; Driver & Shapiro, 1992; Hertz et al., 1992; Karacan et al., 1968; Lee et al., 2000; Wilson et al., 2011).

To date, there do not appear to be any published studies that specifically compare the relationship between polysomnographic and subjective sleep during pregnancy. As highlighted in Chapter 1, previous research suggests that individuals may not be accurate in their perceptions of sleep (Akerstedt et al., 1997; Baker et al., 1999). Accuracy may be further compromised by poor sleep quality (Tsuchiyama, Nagayama, Kudo, Kojima, & Yamada, 2003), which is common during pregnancy (Ross, Murray, & Steiner, 2005). Therefore, it is possible that pregnant women are less accurate in their sleep perceptions than non-pregnant women. The relationship between poor sleep and greater inaccuracy of sleep perceptions could be due to the well-established finding of cognitive impairment as a result of sleep deprivation (Durmer & Dinges, 2005). This further emphasises the need to combine both objective and subjective measures of sleep in order to gain a clearer representation of women's sleep during this period.

In order to understand the relationship between poor sleep and associated outcomes, we first need to examine the effects of pregnancy on women's sleep, and gain a clearer understanding of the accuracy of women's subjective sleep perceptions during pregnancy. Additionally, since much of the research within this area relies upon measures of subjective sleep quality, it is important to understand the specific sleep factors (including EEG variables) that affect pregnant women's perceptions of their overall sleep quality.

6.2 The Current Study

The purpose of this part of the study was to investigate both objective and subjective sleep in third trimester women compared to non-pregnant women, and to address limitations of previous research by examining the relationship between these measures. The objectives of the study are detailed below.

- 1) To compare subjective (sleep diary) and objective (PSG) aspects of sleep in pregnant and non-pregnant women.
- 2) To investigate the accuracy of pregnant and non-pregnant women in terms of their subjective sleep perceptions compared to PSG-measured sleep.
- 3) To compare whether pregnant women are less accurate than non-pregnant women in their subjective sleep perceptions.
- 4) To investigate which subjective and objective sleep factors in pregnant women are associated with subjective perceptions of sleep quality.

6.3 Method

Participants included 29 pregnant women in their third trimester of pregnancy, and a control group of 24 non-pregnant women (refer to Chapter 5, section 5.11 for participant details). Both groups of participants underwent one night of ambulatory PSG at their home (described in Chapter 5, section 5.2.1.1) and were asked to complete the St Mary's Hospital Sleep Questionnaire (SMHSQ) the following morning (see section 5.2.1.2).

6.4 Overview of Statistical Analyses

Between-group analyses were carried out to examine differences in each of the sleep variables between pregnant and non-pregnant women. Data were checked for normality using Shapiro-Wilk tests and were checked for skewness and kurtosis. Mann-Whitney U tests were carried out for the variables that were not normally distributed. All other variables were analysed using independent *t*-tests. In order to examine the accuracy of sleep perceptions, correlations were examined between objective and subjective measures. Shapiro-Wilks tests revealed that the variables were not normally distributed; therefore Spearman's correlations were used. Fisher's Z-test was computed to examine whether correlations between objective and subjective measures were different between groups (e.g. whether pregnant women were less accurate in their sleep perceptions). Another, convergent, measure was derived from difference scores between objective and subjective sleep. Participants were categorised in a dichotomous manner according to whether or not

they showed a discrepancy of more than 30 minutes between subjective and objective total sleep time. Yates' continuity correction was used to calculate odds ratios for both groups regarding the extent to which they were likely to misperceive their sleep by more than 30 minutes. Correlational analysis determined the factors that were most strongly related to the discrepancy in total sleep time perceptions in the group as a whole (e.g. which factors led to poorer accuracy of sleep perceptions). Finally, correlations were carried out to examine the relationship between sleep quality and each of the other sleep variables, to establish the factors most strongly related to subjective sleep quality in each group. This relationship was of particular interest in relation to the pregnant group, since subjective sleep quality has been related to the development of postpartum depression (as described in Chapter 3). Therefore it is important to understand what produces this perception.

6.5 Results

6.5.1 Sleep Variables in Pregnant and Non-Pregnant Women

The mean values for each of the sleep variables for pregnant and non-pregnant women are presented in Table 16. In terms of the variables that were measured both subjectively and objectively, pregnant women reported significantly shorter objective and subjective total sleep time (TST), more subjective and objective number of awakenings, and shorter subjective, but not objective sleep onset latency; although the latter approached significance ($p = .065$).

In terms of the subjective variables, pregnant women also reported spending significantly longer time napping during the daytime and also reported poorer sleep quality. In respect of PSG sleep stage variables, pregnant women showed significantly poorer sleep efficiency, increased stage 1 sleep and decreased REM sleep, in comparison to non-pregnant women. There was also a tendency for them to spend a greater proportion of time in stage 2 sleep ($p = .054$). There were no significant differences between percentage of time spent in stage 3 sleep or REM latency. While there was a trend for pregnant women to have a higher arousal index, this did not reach significance. Differences in subjective sleep quality and PSG-measured sleep efficiency are presented in Figures 14 and 15.

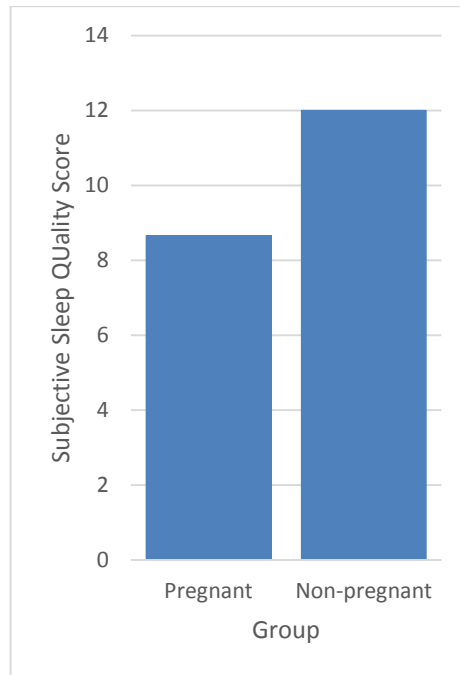


Figure 14. Mean subjective sleep quality scores in pregnant and non-pregnant women.

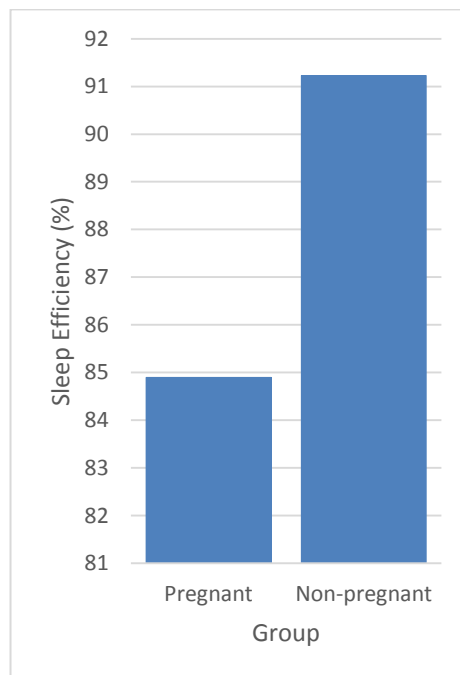


Figure 15. Mean polysomnographic sleep efficiency in pregnant and non-pregnant women.

Overall, pregnant women experienced significantly poorer sleep on the majority of sleep variables. This included less total sleep time, more difficulty falling asleep, more nightly awakenings, and poorer sleep efficiency. Furthermore, these differences were confirmed

using both subjective and objective measures. Pregnant women also reported poorer subjective sleep quality and more daytime napping, and also showed significant differences to their sleep architecture compared to non-pregnant women. This suggests that not only do pregnant women report poorer sleep, but that there are fundamental differences in their sleep architecture that may underlie these perceptions. The relationships between the subjective and objective measures of sleep are explored in more detail in the following sections.

Table 16. Differences in Subjective and Objective Sleep Measures in Pregnant and Non-Pregnant Women

	Controls (n= 24) (Mean \pm SD)	Pregnant (n=29) (Mean \pm SD)	p-value
Comparable Objective / Subjective variables			
Total Sleep (minutes, objective)	467.44 (\pm 64.92)	434.00 (\pm 64.67)	.034 ¹
Total Sleep (minutes, subjective)	461.25 (\pm 87.11)	420.52 (\pm 69.95)	.033 ¹
SOL (minutes, objective)	21.33 (\pm 24.17)	26.76 (\pm 24.85)	.065 ²
SOL (minutes, subjective)	19.89 (\pm 15.40)	38.52 (\pm 43.95)	.045 ²
Awakenings (objective)	5.00 (\pm 3.82)	9.79 (\pm 4.30)	.001 ²
Awakenings (subjective)	2.25 (\pm 1.22)	3.71 (\pm 1.40)	.001 ¹
PSG			
Sleep efficiency (%)	91.23 (\pm 9.03)	84.90 (\pm 10.10)	.001 ²
Stage 1 (%)	8.28 (\pm 4.23)	10.38 (\pm 4.78)	.026 ²
Stage 2 (%)	47.21 (\pm 7.15)	49.86 (\pm 5.24)	.054 ²
Stage 3 (%)	18.55 (\pm 7.95)	17.45 (\pm 5.17)	.282 ¹
REM (%)	25.92 (\pm 4.64)	22.33 (\pm 3.34)	.001 ¹
Awake (%)	4.78 (\pm 8.38)	10.72 (\pm 9.33)	.001 ²
REM latency (minutes)	74.08 (\pm 25.95)	76.00 (\pm 21.37)	.229 ²
Arousal Index	5.27 (\pm 1.60)	6.16 (\pm 2.87)	.091
Subjective sleep			
Daytime naps (mins)	2.50 (\pm 12.25)	16.72 (\pm 42.05)	.046 ¹
Sleep quality	12.00 (\pm 3.15)	8.66 (\pm 4.51)	.003 ¹

Note. ¹t-test ²Confirmed with additional Mann-Whitney test. SOL = sleep onset latency.

6.5.2 Relationships between Objective and Subjective Measures of Sleep

Table 17 shows the correlations between the aspects of sleep that were measured both objectively and subjectively. Overall, there were strong correlations between both subjective and objective total sleep time and sleep onset latency in both groups, although these correlations were stronger in the control group than in the pregnant group. However, these differences were not significant according to Fisher's Z test ($Z = -1.29, -.92$, p 's = .20, .36, respectively). In contrast, subjective and objective number of awakenings were strongly correlated for pregnant women, but not for non-pregnant women. The difference between these correlations was statistically significant ($Z = 2.58$; $p < .01$); suggesting that pregnant women were more accurate in predicting how many times they woke up during the night. The reason that pregnant women may have been more accurate in estimating their number of awakenings is likely to be because not only did they experience significantly more nightly awakenings than non-pregnant women, but they also spent significantly increased time awake during the night. Given that an awakening in this study was scored as a period of alpha (indicative of wakefulness) lasting at least 15 seconds (in line with the American Academy of Sleep Medicine guidelines, Silber et al., 2007), some of the awakenings experienced by the non-pregnant group may have been too short to perceive (Baker et al., 1999).

Table 17. Spearman's Correlations between Objective and Subjective Measures

	Total sleep time (subjective & objective)	Sleep onset latency (subjective & objective)	Number of awakenings (subjective & objective)
Pregnant	.557**	.514**	.570**
Controls	.764**	.684**	-.109

* $p < .05$ ** $p < .01$

Although the correlations between subjective and objective total sleep time were not significantly different between groups, significant differences did arise when participants were subsequently categorised according to whether or not their subjective perception differed from PSG by more than 30 minutes. In this instance, Yates' continuity correction analysis indicated that pregnant women were significantly more likely to misperceive their total sleep time by more than 30 minutes, Yates' (1) = 4.23, $p = .04$, $\phi = .32$, odds ratio

= 3.80. There were no differences between pregnant and non-pregnant women in the extent to which they underestimated versus overestimated their total sleep time, Yates' (1) = 1.56, $p = .21$. However, referring back to Table 16, both groups had a tendency to underestimate their total sleep time when compared to PSG.

An additional analysis was carried out to examine the factors that most strongly related to the discrepancy between objective and subjective total sleep time in the group as a whole, to see whether sleeping poorly was associated with greater sleep state misperception. Indeed, results showed that a larger discrepancy between subjective and objective sleep TST (indicative of greater inaccuracy) was related to poorer sleep quality ($r(51) = -.489$, $p < .01$), increased subjective ($r(51) = .574$, $p < .01$) and objective number of awakenings ($r(51) = .421$, $p < .01$), increased sleep onset latency ($r(51) = .442$, $p < .01$), poorer sleep efficiency ($r(51) = -.511$, $p < .01$) and more percentage of time awake ($r(51) = .379$, $p < .01$). Therefore, poorer sleep appears to be associated with reduced accuracy in sleep perceptions, which may explain why pregnant women were less accurate at recalling their total amounts of sleep.

To summarise, these findings show that pregnant women were more accurate than non-pregnant women at estimating their nightly awakenings, which is likely to be due the fact that they experienced significantly more awakenings which lasted longer, and therefore were more likely to be recalled. In contrast, pregnant women were less accurate at estimating their total amount of night-time sleep, which appears to relate to the finding that their sleep was considerably poorer. Indeed, when sleep is of poor quality and fragmented, it may be more difficult to calculate exactly how much sleep was obtained.

Table 18. Spearman's Correlations between Sleep Variables and Subjective Sleep Quality in Pregnant Women and Controls

<i>Correlations between variables and subjective sleep quality</i>		
	Pregnant	Control
PSG		
Total sleep time (minutes)	.242	.379
Number of awakenings	-.393*	.206
Sleep onset latency (minutes)	-.077	-.224
Sleep efficiency (%)	.262	.379
Stage 1 (%)	-.077	-.243
Stage 1 (mins)	.060	-.027
Stage 2 (%)	.077	.022
Stage 2 (mins)	-.139	.444*
Stage 3 (%)	-.108	.332
Stage 3 (mins)	.079	.332
REM (%)	.315	.041
REM (mins)	-.329	.306
Awake (%)	-.264	-.024
REM latency	.020	.028
Subjective		
Total sleep time (minutes)	.348	.506*
Number of awakenings	-.420*	-.524**
Sleep onset latency (minutes)	.041	-.178

* $p < .05$ ** $p < .01$

6.5.3 Factors Relating to Sleep Quality

Table 18 shows the correlations between each of the sleep measures and women's subjective perception of their sleep quality. The aim of this analysis was to examine which particular aspects of sleep affect how one perceives their overall sleep quality. Some differences arose in the relationships between the sleep variables and subjective perceptions of sleep quality between groups. For instance, among non-pregnant women, more minutes spent in stage 2 sleep was significantly moderately associated with better sleep quality, yet this was not found in the pregnant sample. Furthermore, total sleep time appeared to have a stronger influence on the sleep quality perceptions of non-pregnant women. However, in both pregnant and non-pregnant women, the factor that was most

strongly related to subjective sleep quality was number of awakenings. In fact, this was also found to be the factor that was most strongly related to sleep quality in the general sample in Chapter 3, suggesting that this is not unique to pregnant women.

6.6 Overview of Findings

Compared to non-pregnant women, pregnant women experienced significantly poorer sleep on a number of subjective and objective dimensions. This included less total sleep time, almost twice as many awakenings, and longer sleep onset latency. These findings were confirmed both subjectively and objectively. Pregnant women also reported more daytime napping and poorer subjective perceptions of sleep quality. In terms of sleep architecture, they also had significantly more stage 1 and 2 sleep, poorer sleep efficiency and less REM sleep. However, no significant differences were found in relation to slow-wave sleep (stages 3 and 4).

Given that this appears to be only the fourth study that has compared polysomnographic sleep in pregnant and non-pregnant women, it is important to consider how these findings fit in the context of others. The finding that pregnant women experienced less REM sleep and more stage 1 sleep was also reported by Hertz et al. (1992) and Wilson et al. (2011). However, no differences were reported in relation to slow-wave sleep. While this supports the findings of Hertz et al. (1992), both Karacan et al. (1968) and Wilson et al. (2011) found that third trimester women had less slow-wave sleep. Therefore, while there seems to be consistency in the finding that pregnant women have more stage 1 sleep and less REM sleep, the findings relating to slow-wave sleep remain mixed.

However, there are two advantages to this study over others. Firstly, while the previous studies each used laboratory-based polysomnography, this study used home-based ambulatory polysomnography which is likely to provide a more naturalistic reflection of women's sleep. Secondly, the scoring of polysomnographic data in this study was carried out according to the most up-to-date and well-established guidelines of the American Academy of Sleep Medicine (AASM) guidelines (Silber et al., 2007). The major difference between this scoring system in comparison to the former Rechtschaffen and Kales (1968) system (used in previous studies) is that stages 3 and 4 sleep are now combined, due to a lack of evidence regarding fundamental differences between these stages (Iber et al., 2007). Therefore, when using the most up-to-date scoring criteria, and measuring women's sleep naturally (outside of the laboratory), third trimester women appear to show less REM sleep and more stages 1 and 2 sleep compared to non-pregnant women.

The difference in amounts of stages 1 and 2 sleep between pregnant and non-pregnant women is likely, to some extent, be caused by increased awakenings in pregnant women, since awakenings disrupt sleep cycles. After waking, an individual has to go back through the lighter stages of sleep (stages 1 and 2), and therefore is likely to receive less slow-wave sleep. However, although there was a trend for pregnant women to receive less slow-wave sleep, this finding was not significant. One possibility for this difference may be due to the different scoring systems used in previous studies. The two previous studies that did find reduced slow-wave sleep in pregnant women only found differences in relation to stage 4 sleep, whereas in the current study stages 3 and 4 were measured as a single stage. This highlights the need for consistency in the way in which sleep is scored.

As described in Chapter 1 (1.2.1), the function of REM sleep is a topic has been the subject of a great deal of debate. Some have suggested that the state of REM sleep and accompanied dreaming serve a mood regulatory function, helping an individual to 'work through' emotional memories (Cartwright et al., 1998; Walker & van der Helm, 2009). Given that late pregnancy is often associated with anxiety which often translates into anxious dreams (Nielsen & Paquette, 2007), based on this theory we may expect to see more REM sleep amongst pregnant women, yet the opposite was found. However, it is likely that hormonal and physiological factors influence sleep during this time. For example, previous research has found that late pregnancy is associated with an increase in cortisol (Cousins et al., 1983) and oestrogen (Branchey et al., 1971) which can reduce amounts of REM sleep (Born, Späth-Schwalbe, Schwakenhofer, Kern, & Fehm, 1989).

Clearly, there is more research needed to understand the functionality of changes in sleep architecture during pregnancy. Further research is also needed to examine whether changes to sleep during pregnancy are 'normal,' or whether women who show particular sleep deficits are at greater risk of adverse health outcomes. Chapter 8 examines whether particular aspects of sleep, such as REM latency, are related to the development of postpartum depression. This is important, since depression has been characterised by REM abnormalities such as increased amounts of REM sleep and decreased REM latency (Kupfer & Ehlers, 1989), which have been found to predict future episodes of depression (Giles, Jarrett, Roffwarg, & Rush, 1987; Giles, Kupfer, Rush, & Roffwarg, 1998; Kupfer, 1984).

Another unique aspect of this study is that it was the first study to specifically compare subjective and objective sleep perceptions in pregnant women, using polysomnography.

When examining the relationship between subjective and objective measures of sleep, pregnant women were significantly less accurate in estimating their total sleep time, compared to non-pregnant women. Given that they experienced poorer sleep quality, more awakenings and poorer sleep efficiency, and that these factors correlated with greater inaccuracy in total sleep time perceptions, this suggests that poor sleep in late pregnancy may result in sleep state misperception. This is in line with previous research suggesting that those with more disturbed sleep are likely to be less accurate in their sleep perceptions (Tsuchiyama et al., 2003), which may relate to reduced cognitive performance as a result of sleep disruption (Insana et al., 2013). However, it may also be that when sleep is of poor quality and fragmented, it is simply more difficult to ascertain the exact amount of sleep that was obtained.

Despite this, correlations between objective and subjective measures were relatively strong, even among the pregnant group. In particular, pregnant women were considerably more accurate in estimating their number of awakenings compared to non-pregnant women. This is likely to be due to longer and more frequent awakenings among pregnant women that were therefore more likely to be remembered. Furthermore, number of awakenings was the factor that was most strongly related to sleep quality. Overall, this suggests that perceptions of sleep quality are strongly based upon the number of awakenings over a given night, and that since pregnant women both experience more awakenings and are more aware of these awakenings, they subsequently report poorer sleep quality.

6.7 Chapter Summary

Overall, this chapter reported that pregnant women experienced significantly poorer sleep on the majority of objective and subjective sleep domains. In relation to sleep stages, pregnant women experienced less REM sleep and more stages 1 and 2, but did not differ in relation to slow-wave sleep. They also experienced nearly twice as many awakenings as non-pregnant women. This suggests that not only do pregnant women report poorer sleep, but that there are also fundamental differences in their sleep architecture when compared to non-pregnant women. Due to poorer sleep, pregnant women appear to be significantly less accurate in estimating total sleep time, which highlights the need to incorporate objective measure of sleep. Increased nightly awakenings is the factor that is most strongly related to subjective sleep quality amongst pregnant women, more so than sleep

stages or sleep quantity. Therefore, increased awakenings in pregnant women appears to be what drives reports of poor sleep quality at this time.

Having identified intricate differences between the sleep architecture of pregnant and non-pregnant women, and examined the relationships between subjective and objective measures of sleep, the next chapter explores longitudinal changes to women's sleep over the course of the perinatal period.

CHAPTER 7: LONGITUDINAL CHANGES TO SLEEP, FATIGUE AND DEPRESSION THROUGHOUT THE PERINATAL PERIOD

The aim of this part of the study was to examine changes in sleep, fatigue and depression, from late pregnancy through to one and twelve weeks postpartum. A week of St Mary's Hospital Sleep Questionnaires followed by the Multidimensional Assessment of Fatigue Scale and the Edinburgh Postnatal Depression Scale were completed at each time-point. Results showed that women reported the poorest sleep quality, reduced sleep quantity and were most fatigued during the first postpartum week, compared to the other time-points. Women reported over one and a half hours less total sleep than during pregnancy, even when including daytime napping. This highlights the scope of changes to sleep patterns that occur over a relatively short period of time in the perinatal period. There were no significant changes to depression scores between time-points.

7.1 Introduction

Sleep changes are common during pregnancy (refer to Chapter 1, section 1.6 for an overview). In the study described in Chapter 6, pregnant women experienced poorer sleep on a range of subjective and objective measures, compared to non-pregnant women. Surprisingly few studies have been carried out to examine longitudinal changes in sleep throughout the perinatal period, and several of these studies are limited by small sample sizes (Brunner et al., 1994, $n = 9$, Driver & Shapiro, 1992, $n = 5$, Kang, Matsumoto, Shinkoda, Mishima, & Seo, 2002, $n = 10$; Matsumoto, Kang, & Seo, 2003, $n = 10$, Shinkoda, Matsumoto, & Park, 1999, $n = 4$). Some of these studies have measured sleep using polysomnography (PSG) (Brunner et al., 1994; Driver & Shapiro, 1992; Karacan, Williams, Hirsch, McCaully, & Heine, 1969; Lee et al., 2000). PSG was used in Chapter 6 as it provided in-depth information relating to specific sleep stages and sleep architecture, which was useful in order to provide an in-depth comparison of sleep between pregnant and non-pregnant women. However, while PSG is useful for providing in-depth information relating to sleep architecture, it can only be used for short periods of time. Therefore, sleep diaries and / or actigraphy may be preferable in order to provide a better reflection of changes to perinatal sleep patterns over time.

Using actigraphy and sleep diaries, Signal et al. (2007, $n = 19$) found that the first postpartum week was associated with 1.5 hours less sleep per night, 70% more napping

and three times as many sleep episodes in 24 hours, compared to the second trimester. Differences were also noted between nulliparas and multiparas, with nulliparas experiencing poorer sleep efficiency, more time in bed and more awakenings during pregnancy, and less time in bed and fewer sleep episodes at one week postpartum. Similarly, Kang et al. (2002, $n = 10$) found that early postpartum sleep was associated with decreased sleep efficiency, less TST and increased awakenings, compared to late pregnancy. However, the focus of these studies has tended to be on quantitative aspects of sleep and has not included measures of subjective sleep quality. Given that poor subjective sleep quality has been related to postpartum depression (PPD) (Goyal, Gay, & Lee, 2007; Hiscock & Wake, 2001), it is important to examine changes in women's subjective perceptions of sleep quality over time. Furthermore, these studies did not examine fatigue, which is common during pregnancy and the postpartum period (Lee & Zaffke, 1999; Webster, 1994), and may contribute to postpartum depression (Doering Runquist, Morin, & Stetzer, 2009, see Chapter 3, section 3.6). The preliminary study described in Chapter 4 found that fatigue was the strongest predictor of depression in a general sample, and that fatigue was more strongly related to sleep quality than sleep quantity. Therefore, research is needed to examine possible changes in these variables throughout the perinatal period.

Before examining the relationships between sleep, fatigue and depression in Chapter 8, the aim of this chapter was to explore the longitudinal changes in these variables from late pregnancy through to twelve weeks postpartum. In contrast to previous studies, this study examined both sleep quality and quantity, included measures of depression and fatigue, and assessed whether these factors differed in relation to parity.

7.2 Method

Participants included 29 pregnant women who were recruited in their third trimester of pregnancy (see Chapter 5, section 5.11 for a description of the sample). Participants were asked to complete the St Mary's Hospital Sleep Questionnaire (SMHSQ, see 5.2.1.2) for seven consecutive days during the third trimester of pregnancy, at one and twelve weeks postpartum. At the end of each seven day period, participants were asked to complete the Edinburgh Postpartum Depression Scale (EPDS, see 5.2.3) and the Multidimensional Assessment of Fatigue Scale (MAF, see 5.2.2). Demographic information was collected during pregnancy and at one week postpartum (see 5.2.4.1).

7.3 Overview of Statistical Analyses

A series of one-way repeated- measures one-way ANOVAs were conducted to examine changes in sleep, fatigue and depression over the three time-points: pregnancy, one week postpartum and twelve weeks postpartum. Data were checked for normality, and where necessary non-parametric alternatives (Friedman's ANOVA) were also carried out. If sphericity was not assumed, adjustments were made using Greenhouse-Geisser. Statistical information and effect sizes (Cohen's *d*) are presented in tables throughout the chapter. Post-hoc (Bonferonni) tests were carried out to locate the source of difference (Wilcoxon signed-rank tests were used if data were not normally distributed). Mean scores are presented for the total number of participants included at each stage of the study, but only participants who completed all three time-points (*n*=19) were included in the statistical tests. At the beginning of the study 29 pregnant women participated, this reduced to 23 women at one week postpartum and 19 women and twelve weeks postpartum, due to participants not returning their questionnaires.

7.4 Results

7.4.2 Changes in Subjective Sleep across the Three Time-Points

Mean values of women's subjective sleep across the three time-points are presented in Table 19.

Table 19. Mean Subjective Sleep Variables, Fatigue and Depression Scores at Each Time-Point

	Mean (SD)							
	Pregnancy (n=29)	Week 1 (n=26)	Week 12 (n=19)	<i>df</i>	<i>F</i>	<i>p</i>	Post hoc (Bonferroni)	Effect size (Cohen's <i>d</i>)
Night-time sleep (minutes)	426.41 (57.72)	309.21 (94.74)	410.41 (48.76)	1,41, 25.41	23.51	<.001	Pregnancy > Week 1 Week 12 > Week 1	1.14
Naps (minutes)	20.12 (27.67)	37.53 (41.67)	6.35 (16.06)	1,41, 25.44	9.27	.001	Week 1 > Pregnancy Week 12 < Week 1	1.55
Total sleep (minutes)	446.53 (68.93)	343.82 (105.91)	393.53 (105.87)	2, 36	4.16	.024	Pregnancy > Week 1	.48
Sleep onset latency (minutes)	41.04 (40.81)	33.30 (70.95)	25.89 (30.18)	1,19, 21.49	0.79	.406	-	-
Sleep quality	10.26 (3.16)	9.54 (3.28)	12.22 (2.87)	2, 36	4.26	.022	Week 12 > Week 1	.45
Number of awakenings	3.45 (1.39)	3.50 (0.87)	2.19 (1.33)	2, 36	7.13	.002	Pregnancy > Week 12 Week 1 > Week 12	.63
Fatigue	25.21 (7.32)	27.90 (8.89)	21.74 (9.80)	2, 24	5.20	.011	Week 1 > Week 12	.55
Depression	5.15 (3.90)	6.54 (6.32)	6.05 (5.52)	2, 24	1.07	.352	-	-

Night-Time Total Sleep Time (see Figure 16)

As can be seen in Table 19, in terms of total sleep time (TST), women received an average of around 7 hours in late pregnancy, around 5 hours during the first week postpartum, and slightly less than 7 hours at twelve weeks postpartum. Mean TST during pregnancy ($p = <.001$) and at week 12 ($p = <.001$) was significantly longer than at 1 week. There was no significant difference between TST during pregnancy and week 12.

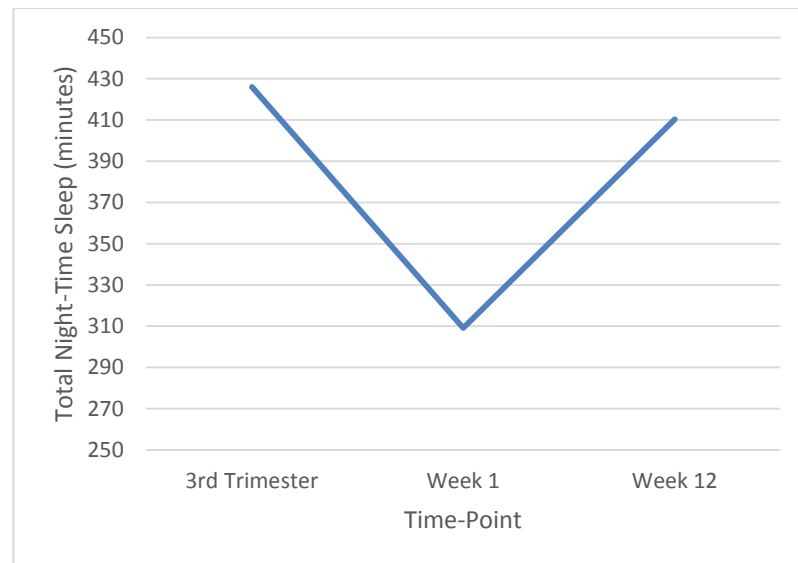


Figure 16. Mean night-time sleep across the three time-points.

Daytime Naps (see Figure 17)

As expected, daytime naps appeared to increase as TST decreased. The amount of time spent engaging in daytime naps increased significantly from late pregnancy to week 1 ($p = .023$) and decreased significantly from week 1 to week 12 ($p = .011$). However, using non-parametric tests, the only significant differences were between weeks 1 and 12 ($z = -3.24$, $p = .001$). There was no significant difference in the amount of time spent napping during pregnancy and week 12.

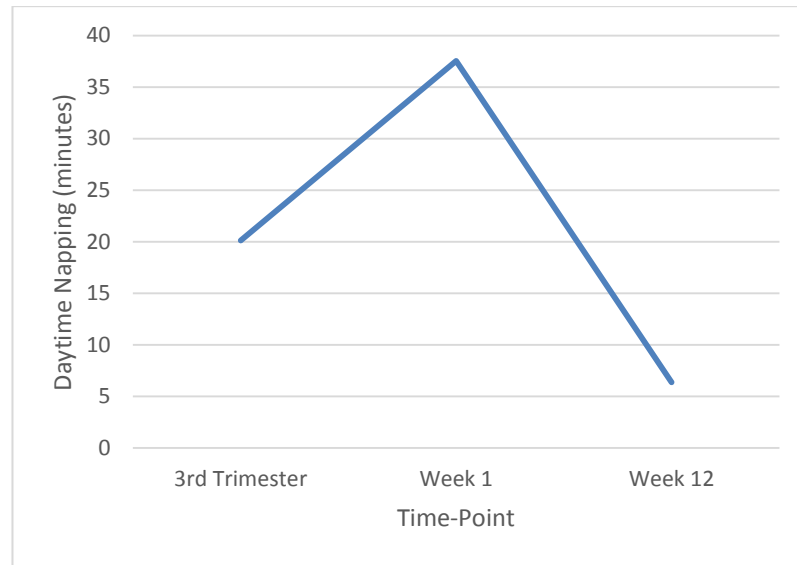


Figure 17. Mean daytime napping across the three time-points.

Total Sleep (night-time plus daytime naps)

When night-time and day-time sleep were combined, women experienced significantly more total sleep during pregnancy than at week 1 ($p=.012$). However, there were no significant differences between weeks 1 and 12, or between pregnancy and week 12. This suggests that the most prominent changes in total sleep occurred between pregnancy and the early postpartum period.

Sleep Onset Latency

In contrast to the majority of sleep variables, sleep onset latency appeared to be longest during the third trimester, and continued to fall during the subsequent time-points. However, these changes were not significant.

Sleep Quality (see Figure 18)

Average sleep quality ratings improved significantly from week 1 to week 12 ($p=.031$). These ratings were higher during week 12 than during pregnancy (indicative of better sleep), however this was not significant. In addition, there was no significant difference between sleep quality during pregnancy and the early postpartum period, suggesting that women perceived their sleep quality to be equally poor during these periods, in comparison to later in the postpartum period.

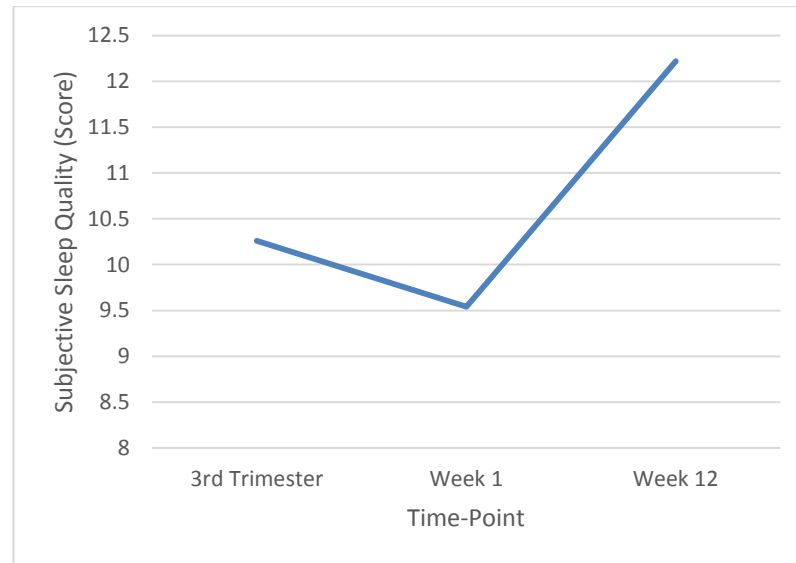


Figure 18. Mean subjective sleep quality score over the three time-points.

Number of Awakenings

The average number of nightly awakenings that women reported did not differ significantly from pregnancy to week 1, but decreased significantly by week 12. There were significantly more awakenings during pregnancy ($p = .047$) and week 1 ($p < .001$) than during week 12.

Overview of Sleep Changes

Perinatal women's sleep patterns changed considerably from late pregnancy through to twelve weeks postpartum. These changes were reflected by medium to strong effect sizes. Overall, women reported the poorest sleep during the first week postpartum on the majority of domains, compared to late pregnancy and 12 weeks postpartum. This was characterised by less sleep, more awakenings, more daytime napping, more fatigue and poor sleep quality.

When comparing sleep during late pregnancy and 12 weeks postpartum, in many respects, sleep during late pregnancy appeared to be poorer, with a tendency for more reported fatigue, poorer sleep quality and more difficulty falling asleep (although these findings did not reach significance). Women also experienced significantly more awakenings during late pregnancy compared to week 12. However, one aspect of sleep that was not poorer was sleep *quantity*. In fact, women tended to longer total sleep time during pregnancy than

at 12 weeks postpartum, despite the fact that they reported poorer sleep quality. Given that we know from Chapter 6 that subjective sleep quality is strongly related to number awakenings, this may be why women tended to report poorer sleep quality during pregnancy, despite reporting more overall sleep.

7.4.3 Changes in Fatigue across the Time-Points

Mean fatigue scores are also shown in Table 19. There were no significant differences between pregnancy and week 1, suggesting that women were equally fatigued during these time-points. However, fatigue significantly decreased from week 1 to week 12 ($p=0.023$).

7.4.4 Changes in Depression across Time-Points

Figure 19 shows distributions of depression scores across the time-points. From this graph it appears that while the average number of women with low depression scores remained fairly consistent over time, the percentage of women with higher depression scores increased marginally across time. EPDS scores ranged from 0-16 during late pregnancy, 0-20 during the first week postpartum, and 0-20 at 12 weeks postpartum.

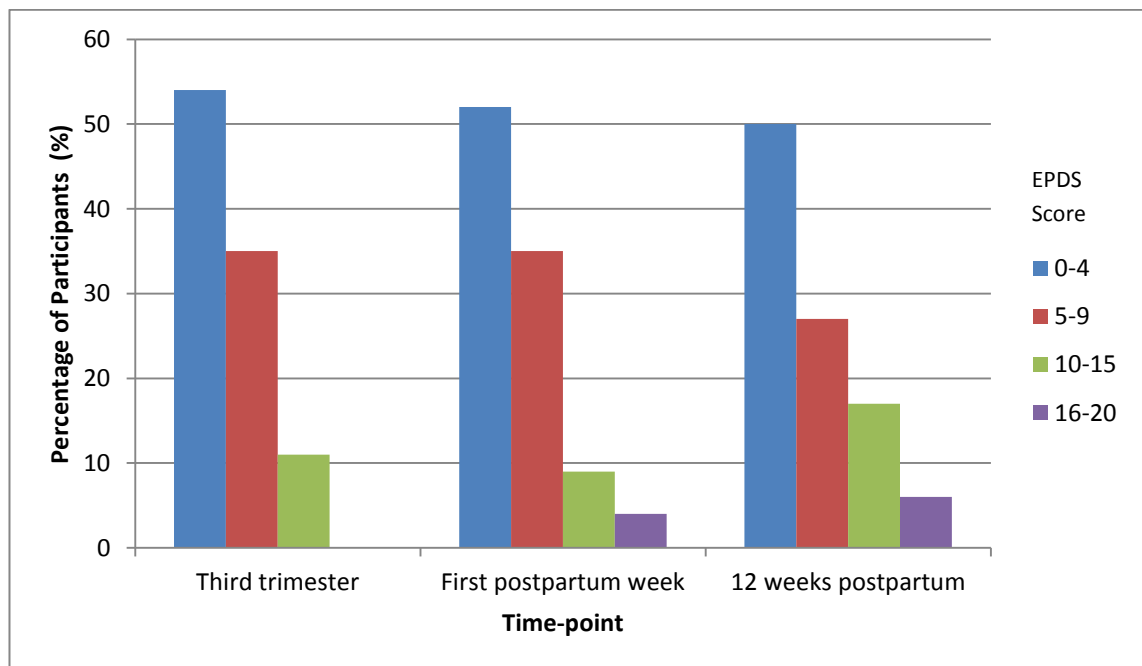


Figure 19. Distribution of EPDS scores across time.

Mean Edinburgh Postnatal Depression Scale (EPDS) scores are shown in Table 19. When categorising participants according to a score of ≥ 10 , three participants were classified as depressed during late pregnancy and one week postpartum, compared to five participants at 12 weeks postpartum. Of the three women who were classified as depressed during pregnancy, one woman was no longer depressed at week 1 (EPDS = 5) but did not complete week 12 questionnaires, and a further woman did not complete either week 1 or 12 questionnaires. The third woman was classified as depressed during each subsequent time-point. Out of the five women classified as depressed at 12 weeks postpartum, one women exceeded the EPDS cut-off at both prior time-points, and one women exceeded the cut-off at week one and week 12, but not during pregnancy. Two of these women approached the cut-off scores during late pregnancy (EPDS = 9), however the final woman who was classified as depressed during week 12 had low scores at both previous time-points. Trajectories of EPDS scores in the five women scoring ≥ 10 at 12 weeks postpartum are shown in Figure 20.

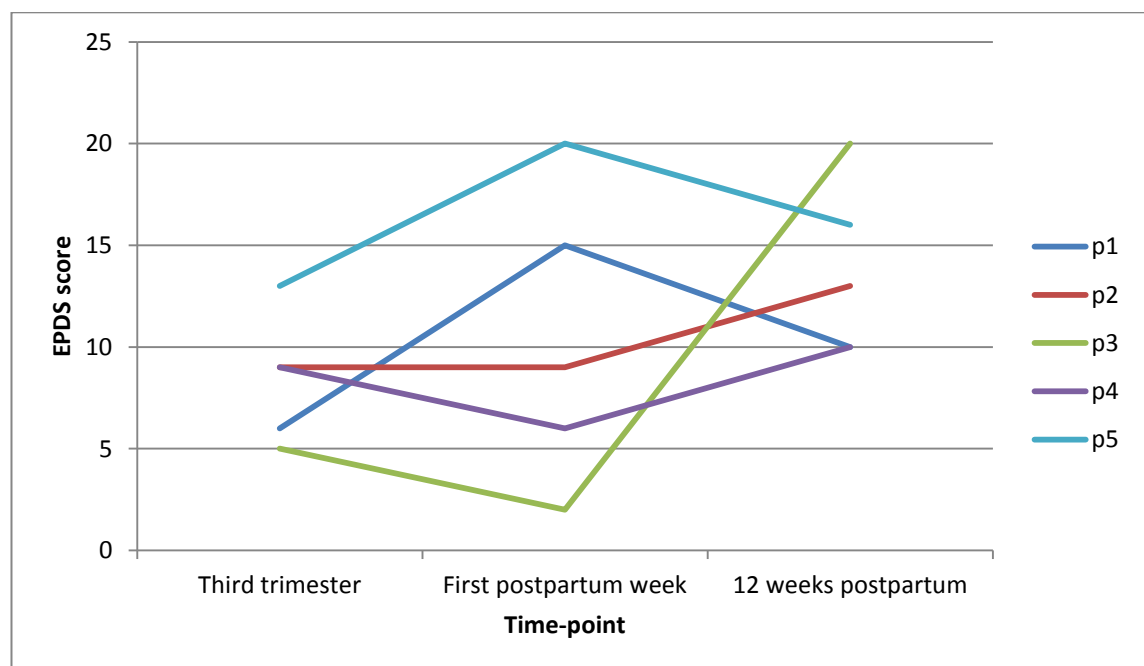


Figure 20. Trajectory of mean EPDS scores across time among the five women scoring ≥ 10 on the EPDS at 12 weeks postpartum.

It is interesting to note that although no participants reported a diagnosis of depression at baseline (either through self-report or the MINI screening interview), three participants achieved the EPDS cut-off for depression during the third trimester, highlighting potential

differences in sensitivity between these measures. While depression scores appeared to be highest in the first week postpartum, thus mirroring changes in sleep and fatigue, these differences were not significant. This suggests that depressive symptoms remained relatively stable across time-points.

Figures 21 and 22 show trajectories of total sleep (including daytime napping) and sleep onset latency across the three time-points among the women who scored ≥ 10 at 12 weeks postpartum. It appears that the majority of these women experienced the shortest amount of sleep during late the first postpartum week, except one women who appeared to show the opposite pattern; experiencing the most sleep during the first postpartum week. However, this is also the period when daytime napping was most apparent.

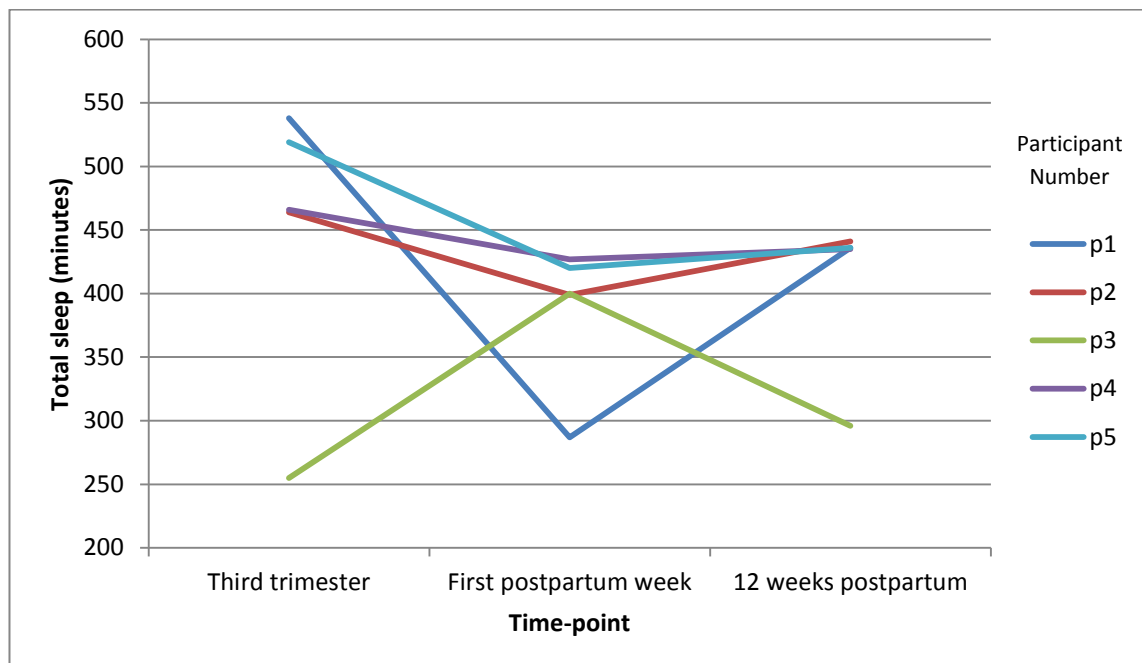


Figure 21. Total amount of sleep across time among women scoring ≥ 10 on the EPDS at 12 weeks postpartum.

Sleep onset latency was relatively stable across time among the majority of women (see Figure 22), with a slight decrease at one week postpartum. However, one participant who reported increased sleep onset latency at each time-point showed a different pattern, experiencing the most difficulty falling asleep during the first postpartum week. A detailed exploration of the relationship between sleep and postpartum depression is provided in Chapter 8.

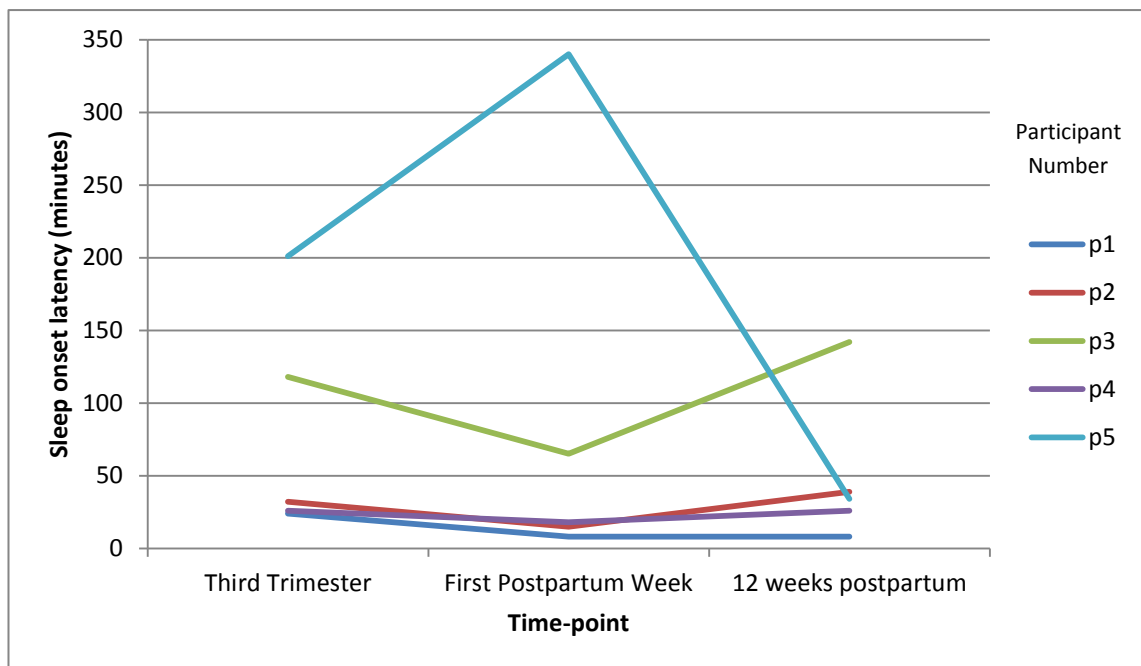


Figure 22. Subjective sleep-onset latency across time among women scoring ≥ 10 on the EPDS at 12 weeks postpartum.

7.5 Effects of Parity

A series of independent *t*-tests were carried out to examine whether any differences any differences arose between primiparas and multiparas, in relation to subjective sleep, fatigue and depression at any of the time-points. Primiparas ($n=12$) had significantly higher week one postpartum EPDS scores (mean=7.33, SD=5.76) than multiparas ($n=10$, mean=3.8, SD=2.78), $t(20) = 1.77, p=.046$), but did not differ on pregnancy or week 12 scores. Primiparas also spent significantly longer napping in the first postpartum week (mean = 51.43 minutes, SD= 47.64) compared to multiparas (mean =18.21 minutes, SD=19.52), $t(15.13) = 2.20, p=.020$. No other differences were found between groups.

7.6 Overview of Findings

The aims of this chapter were to examine changes in subjective sleep, fatigue and depression throughout the perinatal period. Previous research has tended to focus on quantitative aspects of sleep rather than asking women about how they perceive their sleep quality. Furthermore, no other studies appear to have examined changes to sleep alongside changes to fatigue and depression.

The results of this study support those of previous research in the finding that women experienced the poorest sleep during the first postpartum week, compared to the other time-points (Gay et al., 2004; Kang et al., 2002; Signal et al., 2007). Women slept nearly two hours less at night and spent nearly twice as long napping in the first postpartum week compared to late pregnancy. They also reported poorer subjective sleep quality and increased symptoms of fatigue. This supports the findings of Wambach (1998) suggesting that fatigue is highest during the first postpartum week, in comparison to later in the postpartum period. As women's total sleep time decreased, they engaged in more daytime napping to compensate. However, even when night-time and day-time sleep were combined, the amount of sleep experienced during the early postpartum period was over one and a half hours less than during late pregnancy. By 12 weeks postpartum the majority of sleep variables had improved, indicative of relative recovery from early postpartum sleep disruption.

Some interesting findings emerged in relation to differences between patterns during pregnancy and 12 weeks postpartum. Despite a tendency to report more sleep during late pregnancy than at 12 weeks, women reported significantly more awakenings and tended to report poorer subjective sleep quality. This suggests that changes to subjective sleep quality do not reflect sleep quantity, but instead appear to reflect sleep continuity, thus supporting the findings of Chapter 6. Previous research has shown that the way in which women perceive their overall sleep quality predicts symptoms of postpartum depression, even when controlling for infant sleep disruption (Hiscock & Wake, 2001). Therefore, it is important to consider not only quantitative changes to sleep, but also how women perceive their sleep quality.

Although depression scores did not change significantly across time, there was a trend for higher levels of depression during the first postpartum week. This suggests that changes in sleep could potentially contribute to early postpartum depression symptoms. However, further research is needed to investigate of the relationship between these variables. This will be addressed in the following chapter. Almost a quarter of participants reported depression scores ≥ 10 on the EPDS at weeks 1 and 12, which is higher than previously established prevalence rate of around 13% (O'Hara & Swain, 1996). However, early postpartum scores would have been likely to reflect postpartum blues rather than depression (Ross, Murray, & Steiner, 2005). A lack of significant change in depression scores suggests that women's mood remained relatively stable over the course of the

perinatal period. This highlights that the importance of considering antepartum depression as a risk factor for postpartum depression (O'Hara & Swain, 1996).

In relation to parity, total sleep time was similar across both nulliparas and multiparas, supporting the findings of both Lee et al.(2000) and Signal et al.(2007). However, first-time mothers had significantly higher levels of depression during the first week postpartum. This is in line with previous studies, suggesting that first-time mothers have an increased risk of developing PPD (Glavin, Smith, & Sørum, 2009). Compared to experienced mothers, primiparas also spent significantly longer time napping during the first week postpartum. This contrasts to Signal et al. (2007) who found that nulliparas had fewer sleep episodes over a 24-hour period at one week postpartum compared to multiparas, but did not find any significant differences in relation to napping. However, this may be due to difference in measurement between this study and that of Signal et al. (2007), who referred to the frequency rather than the length of naps. The key difference between primiparas and multiparas is therefore perhaps not in the quantity of naps, but in the length of these naps. Multiparas may not be able to spend as long napping as primiparas, since they may have to care for other infants.

In summary, compared to late pregnancy and three months postpartum, women reported the most dramatic changes to their sleep and most fatigue during the first postpartum week. Despite attempts to compensate for lack of sleep through daytime napping, women experienced over one and a half hours less sleep compared to late pregnancy and reported significantly poorer sleep quality. Given that Chapter 6 highlighted the extent to which sleep is poorer in pregnant women compared to non-pregnant women, the finding that early postpartum sleep is even poorer than late pregnancy emphasises the severity of sleep changes during this time. This begs the question of whether or not these dramatic changes to sleep may precipitate changes in mood. This will be explored in Chapter 8. By using a prospective sleep measure, this part of the study was able to capture the degree of changes to sleep patterns that occur over a relatively short period of time.

7.7 Chapter Summary

This appears to be the first study to simultaneously examine changes to subjective sleep quality, quantity, fatigue and depression throughout the perinatal period. Chapter 6 demonstrated that sleep during late pregnancy is significantly worse than the sleep of non-pregnant women. However, this chapter showed that the early postpartum period is further characterised by considerable sleep changes that should not be overlooked.

Women should be informed about the type of sleep changes that they should expect to experience during this time, along with ways of reducing sleep disruption. Given what is known about the effects of sleep disruption on mental and physical well-being, the analyses so far suggest that more attention should be given to perinatal sleep. However, the finding that depression scores did not change significantly across time suggests that depression during pregnancy is a strong predictor of postpartum depression. The following chapter describes the final part of the analysis, examining relationships between sleep, fatigue and the development of postpartum depression.

CHAPTER 8: RELATIONSHIPS BETWEEN SLEEP, FATIGUE AND DEPRESSION THROUGHOUT THE PERINATAL PERIOD

The aim of this part of the study was to examine relationships between sleep, fatigue and postpartum depression. Pregnant participants (n=29) underwent one night of polysomnography during the third trimester of pregnancy, and completed a week of St Mary's Hospital Sleep Questionnaires followed by the Multidimensional Assessment of Fatigue Scale and the Edinburgh Postnatal Depression Scale at three time-points: the third trimester, one week postpartum and twelve weeks postpartum. The variables that were most consistently associated with depression scores among each of the time-points were fatigue and sleep onset latency, suggesting that these are key symptoms of perinatal depression. Sleep onset latency and total amounts of sleep during late pregnancy significantly predicted week 1 EPDS scores, when controlling for depression during pregnancy, suggesting that these are significant risk factors for the development of early postpartum depressive symptoms. In contrast, the only variable that significantly predicted week 12 EPDS score was depression during pregnancy, suggesting that while sleep during late pregnancy is important, the strongest predictor of postpartum depression appears to be depression during pregnancy. In contrast, there were no significant relationships between sleep stages measured through polysomnography, and the development of postpartum depression. The implications of these findings are discussed.

8.1 Introduction

The previous two chapters have highlighted the scope of sleep changes that occur throughout the perinatal period. The aim of this final results chapter is to examine whether any of these aspects of sleep are related to the development of postpartum depression (PPD). Previous research investigating relationships between sleep and postpartum depression was examined in Chapter 3. To recap, research suggests that women experiencing symptoms of postpartum depression report poorer sleep than non-depressed women (Da Costa, Dritsa, Rippen, Lowensteyn, & Khalifé, 2006; Dorheim, Bondevik, Eberhard-Gran, & Bjorvatn, 2009; Groer & Morgan, 2007; Huang, Carter, & Guo, 2004). However, some studies using objective measures of sleep have not confirmed these findings (Dorheim et al., 2009; Godfroid, Hubain, Dramaix, & Linkowski, 1997). For example, Dorheim et al. (2009) found that while women with postpartum depression (PPD) reported poorer sleep than non-depressed women when sleep was measured

retrospectively, no differences emerged using prospective sleep diaries or actigraphy. This highlights the importance of using both objective and subjective measures, particularly given that perinatal women may be less accurate in their sleep perceptions (see Chapter 6).

While it is important to understand how sleep is related to current symptoms of PPD, it is perhaps even more important to understand whether sleep disruption can play a role in the development of PPD. Although there is some evidence for this (e.g. Bei, Milgrom, Ericksen, & Trinder, 2010; Lee, McEnany, & Zaffke, 2000; Wolfson, Crowley, Anwer, & Bassett, 2003), the current literature is limited by studies that have:

- (i) failed to control for depression at baseline (Wilkie & Shapiro, 1992)
- (ii) relied upon subjective (often retrospective) measures of sleep (Dennis & Ross, 2005; O'Hara & Swain, 1996; Wilkie & Shapiro, 1992; Wolfson et al., 2003).
- (iii) only measured PPD symptoms in the early postpartum period (Bei et al., 2010; Cooc Calcagni et al., 2012; Deepika Goyal et al., 2009; O'Hara & Swain, 1996).

Furthermore, only one study has specifically examined the longitudinal relationship between sleep and PPD using polysomnography (Lee et al., 2000), the 'gold standard' measure of sleep (Kushida et al., 2005). Given that certain aspects of sleep architecture, such as reduced REM latency, have been found to predict those at risk of major depressive disorder (Giles et al., 1990, 1987, 1998), further research using polysomnography is needed.

The aim of this study was therefore to investigate the relationships between objective sleep (using PSG), subjective sleep, fatigue and depression, during pregnancy and at weeks 1 and 12 after birth, and to establish which specific sleep-related factors were most predictive of PPD. Given that particular aspects of sleep appear to be more strongly related to depression than others (see Chapter 4), there is a need to measure a range of sleep variables. The specific objectives were to:

- (i) examine cross-sectional relationships between the different measures of sleep and fatigue and depression.
- (ii) examine longitudinal relationships between these variables.
- (iii) examine which variables were most predictive of PPD symptoms, after controlling for baseline depressive symptoms.

8.2 Method

This part of the study focused on the pregnant group only, examining changes in sleep and depression measures during pregnancy and postpartum. Full details of participant characteristics and the study procedure can be found in Chapter 5. Participants completed one night of polysomnography (objective sleep) during the third trimester of pregnancy, followed by a week of St Mary's Hospital Sleep Questionnaires (SMHSQ, subjective sleep), and a copy of the Edinburgh Postnatal Depression Scale (EPDS) and the Multidimensional Assessment of Fatigue (MAF) scale. These questionnaires were also completed during the first postpartum week, and at twelve weeks. Therefore, while objective sleep was measured at a single time point (during late pregnancy), subjective sleep and fatigue were measured at each of the three time-points. Demographic information was completed during pregnancy and following childbirth (see Chapter 5.2.4.1).

8.3 Overview of Statistical Analyses

Correlations were carried out to examine relationships between sleep, fatigue and depression at each time-point, as well as prospectively. Tests of normality showed that EPDS scores at week 1 and 12 were slightly skewed. However, after identifying an outlier, which was subsequently removed from the data set due to having a z-score of >2.58 (Mayers, 2013), the data was normally distributed and therefore Pearson's correlations were used. After examining patterns of correlations between variables, stepwise multiple linear regression analyses were used to examine the variables that predicted postpartum depression at weeks 1 and 12. The stepwise method was used in order to ascertain the extent to which a particular variable predicted depression, while controlling for other variables. All participants took part in the initial PSG sleep study and completed the subsequent SMHSQ sleep questionnaire in the morning. However, due to the common issue of attrition in longitudinal studies, 23 women were included at one week postpartum and 19 women were included at twelve weeks postpartum.

8.4 Results

The first part of this section describes the cross-sectional relationships between sleep, fatigue and depression at each time-point (third trimester, 1 week postpartum, 12 weeks postpartum). First described are the relationships between objective sleep and EPDS scores during pregnancy, followed by the relationships between subjective sleep, fatigue and EPDS symptoms during pregnancy. Following from this is the examination of the relationships between subjective sleep, fatigue and EPDS scores during the 1st week

postpartum, and then again at 12 weeks postpartum. The aim was to gain an understanding of the cross-sectional nature of the relationship between sleep and depression before examining prospective, longitudinal relationships. This would provide an indication of which aspects of sleep appear to be symptomatic of depression, compared to those which may be predictive of depression.

8.4.1 Cross-Sectional Relationships

8.4.1.1 Relationships between Sleep, Fatigue and Depression during Pregnancy

a) Objective Sleep and Depression during Pregnancy

Correlations were carried out to examine the relationships between objective sleep factors (measured using PSG) and depression during pregnancy. The results are displayed in Table 20. As can be seen from the pattern of correlations, the only objective sleep variable that was significantly related to EPDS scores during pregnancy was total sleep time. Greater total sleep time was associated with greater depression scores ($r(26) = .414$, $p = .018$). Percentage of stage 2 sleep was moderately negatively correlated with EPDS scores, although this only approached significance ($r(26) = -.38$, $p = .055$).

Table 20. Pearson's Correlations between Objective PSG Variables and EPDS Scores during Pregnancy

	Objective Sleep Pregnancy									
	TST	SE	SoL	Wake (no.)	Wake (%)	S1%	S2%	S3%	REM%	REM latency
EPDS Pregnancy	.414*	.223	.194	-.148	-.221	.115	-.380	.304	-.020	.160

Note. TST= total sleep time, SE= sleep efficiency, SoL= sleep onset latency, S1,2,3 = stages 1,2,3. * $p < .05$,

** $p < .01$

b) Subjective Sleep and Depression during Pregnancy

Table 21. Pearson's Correlations between Subjective Sleep, Fatigue and Depression during Pregnancy

	Subjective Sleep Pregnancy						
	TST (night)	Wake (no.)	SoL	Naps	Total sleep	SQ	Fatigue
EPDS Pregnancy	.340	-.019	.484*	-.102	.257	-.081	.528**

Note. TST= total sleep time, SoL= sleep onset latency, SQ= sleep quality, * $p < .05$, ** $p < .01$

The same analyses were carried out with the subjective sleep variables. Greater subjective total sleep time was also related to greater depression during pregnancy, however this relationship was not as strong as its objectively measured counterpart. However, it is important to note that subjective sleep variables were taken over a week, whereas objective variables were taken from a single night. Table 21 shows that EPDS scores during pregnancy were moderately positively related to sleep onset latency ($r(26) = .446$, $p = .023$) and strongly related to fatigue ($r(26) = .527$, $p = .006$).

Overview of Cross-Sectional Relationships between Subjective Sleep, Objective Sleep, Fatigue and Depression during Pregnancy

Differing relationships emerged between subjective and objective sleep in relation to depression during pregnancy. Higher depression scores were related to increased objective, but not subjective TST, longer subjective but not objective sleep onset latency and increased levels of fatigue. Fatigue and subjective sleep onset latency were the variables most strongly related to depression.

8.4.1.2 Relationships between Sleep, Fatigue and Depression during the First Postpartum Week

Table 22 is similar to the previous table, however this time it presents the relationships between subjective sleep, fatigue and EPDS scores during the early postpartum period, as opposed to late pregnancy. As can be seen in Table 22, depression was associated with greater fatigue and increased sleep onset latency one week postpartum. There was also a moderate positive relationship between amount of time spent napping and depression scores, however, this did not reach significance.

Table 22. Pearson's Correlations between Subjective Sleep, Fatigue and Depression during the First Postpartum Week

	Subjective Sleep Week 1						Fatigue
	TST (night)	Wake (no.)	SoL	Naps	Total sleep	SQ	
EPDS Week 1	-.132	-.046	.695**	.387	-.043	-.270	.424*

Note. TST= total sleep time, SoL= sleep onset latency, SQ= sleep quality, * $p < .05$, ** $p < .01$

8.4.1.3 Relationships between sleep, fatigue and depression at 12 weeks postpartum

As can be seen in Table 23, similar to during pregnancy and the postpartum period, sleep onset latency was the variable that was most strongly related to week 12 depression scores; increased depression was associated with longer sleep onset latency. While fatigue was positively correlated with depression, unlike at the other time-points, this relationship did not reach significance ($p = .263$).

Table 23. Pearson's Correlations between Subjective Sleep, Fatigue and Depression at 12 Weeks Postpartum

	Subjective Sleep Week 12						Fatigue
	TST (night)	Wake (no.)	SoL	Naps	Total sleep	SQ	
EPDS Week 12	-.157	-.227	.518*	-.197	-.390	-.025	.279

Note. TST= total sleep time, SoL= sleep onset latency, SQ= sleep quality, * $p < .05$, ** $p < .01$

8.4.2 Summary of Cross-Sectional Analyses

Across each time-point, subjective sleep onset latency and fatigue were the variables that were most consistently associated with depression scores, suggesting that these factors are particularly relevant to depression. The only objective sleep variable that was significantly (positively) related to depression during pregnancy was total sleep time.

8.5 Prospective Relationships between Sleep, Fatigue and Depression

The following analyses examine the prospective relationships between the sleep variables and postpartum depression scores. First described are the relationships between objective

sleep during pregnancy, and weeks 1 and 12 EPDS scores. This is followed by an examination of the relationships between subjective sleep and fatigue during pregnancy and weeks 1 and 12 EPDS scores. The final part of this section describes the relationships between subjective sleep and fatigue during week 1 postpartum and EPDS scores at week 12. The aims of these analyses were to identify which variables were most strongly related to postpartum depression scores, and at which time-point these relationships were strongest.

8.5.1 Relationships between Subjective Sleep, Objective Sleep and Fatigue during Pregnancy, and Postpartum Depression Scores

a) Objective Sleep during Pregnancy and Postpartum Depression Scores at Weeks 1 and 12

Table 24 shows correlations between objective sleep variables during pregnancy and week 1 and 12 EPDS scores. Increased objective sleep onset latency during pregnancy was moderately associated with increased depression scores at weeks 1 and 12, although the week 12 relationship did not reach significance ($p=.097$). Although there were mild to moderate correlations between the objective sleep stage variables and postpartum depression scores, none of these variables reached significance, except that less stage 2 sleep during pregnancy was moderately-strongly associated with greater week 12 depression scores, which approached significance ($p=.060$).

Interestingly, when looking at the overall patterns of correlations between the pregnancy sleep stage variables and EPDS scores, they appear to be more strongly related to week 12 depression scores than either pregnancy or week 1 depression scores. This suggests that particular aspects of sleep architecture during pregnancy (such as decreased stage 2 sleep) may indeed relate to the development of postpartum depression, which warrants further research. However, in contrast to previous research suggesting that reduced REM latency is a risk factor for depression (Kupfer, Bulik, & Grochocinski, 1984), these results suggest the opposite: increased REM latency was associated with increased depressive symptoms. However, again, this relationship was not significant.

Table 24. Pearson's Correlations between Objective PSG Variables during Pregnancy, and Postpartum Weeks 1 and 12 EPDS scores

	Objective Sleep Variables during Pregnancy									
	TST	SE	SoL	Wake (no.)	Wake (%)	S1%	S2%	S3%	REM%	REM latency
EPDS										
Week 1	.125	.017	.501*	-.091	-.086	.190	-.257	.267	-.295	.377
EPDS										
Week 12	.252	-.088	.404	.386	.115	.295	-.452	.324	-.197	.374

Note. TST= total sleep time, SE= sleep efficiency, SoL= sleep onset latency, S1,2,3 = stages 1,2,3, * $p < .05$, ** $p < .01$

b) Subjective Sleep during Pregnancy and Postpartum Depression Scores at Weeks 1 and 12

Table 25 shows correlations between subjective sleep during late pregnancy and week 1 and 12 postpartum depression scores. Subjective total sleep time during pregnancy was strongly positively correlated with EPDS scores at week 1. Therefore, sleeping longer during pregnancy appears to be related to increased risk of early postpartum depressive symptoms. Similar to objective sleep during pregnancy, increased subjective sleep onset latency during pregnancy was strongly associated with depression both at weeks 1 and 12. However, in contrast to the cross-sectional relationships, fatigue during pregnancy was not prospectively associated with depression at either postpartum time-point. This suggests that fatigue reflects a symptom of current depression rather than a factor associated with its development.

Table 25. Pearson's Correlations between Subjective Sleep and Fatigue during Pregnancy and Depression and week 1 and 12 EPDS scores

	Subjective Sleep Variables During Pregnancy						Fatigue
	TST (night)	Wake (no.)	SoL	Naps	Total sleep	SQ	
EPDS							
Week 1	.528*	.082	.563*	.221	.617**	.108	.123
EPDS							
Week 12	.392	.399	.518*	.379	.530*	-.350	.215

Note. TST= total sleep time, SoL= sleep onset latency, SQ= sleep quality, * $p<.05$, ** $p<.01$

8.5.2 Relationships between Subjective Sleep and Fatigue during the First Postpartum Week, and Week 12 Postpartum Depression Scores

As can be seen in Table 26, increased sleep onset latency in the first postpartum week was also moderately associated with greater week 12 EPDS scores. There were no significant relationships between any of the other sleep variables and week 12 depression. Again, this suggests that increased sleep onset latency during pregnancy appears to be a significant risk factor for postnatal depression.

Table 26. Pearson's Correlations between Subjective Sleep, Fatigue and Depression during the First Postpartum Week and Week 12 Depression Scores

	Subjective Sleep Variables During Week 1 Postpartum						Fatigue
	TST (night)	Wake (no.)	SoL	Naps	Total sleep	SQ	
EPDS							
Week 12	.089	-.224	.558*	.301	.121	-.278	.163

Note. TST= total sleep time, SoL= sleep onset latency, SQ= sleep quality, * $p<.05$, ** $p<.01$

8.5.3 Overview of Prospective Relationships between Sleep, Fatigue and Depression across the Three Time-Points

On the basis of the correlations at each time point, it appears that prospectively, sleep onset latency is a key variable that is associated with depression at both postpartum time-points. Therefore, as well as being a sign of current depression, increased sleep onset latency may also relate to future levels of depression. It also appears that total amount of sleep obtained during late pregnancy is strongly associated with depression at both postpartum time-points; particularly during the early postpartum phase, signalling that dramatic changes in total amounts of sleep from late pregnancy to the early postpartum period may precipitate early depressive symptoms. Objective sleep variables were not significantly associated with postpartum depression, with the exception of sleep onset latency during pregnancy. The fact that fatigue was strongly associated with depression when examined in a cross-sectional but not prospective manner suggests that while fatigue appears to be a key symptom of perinatal depression, it does not appear to contribute to its development.

8.6 Consistency of Depression Scores over Time

Table 27 shows that there were strong correlations between EPDS scores at each time-point. The strongest relationship was between EPDS scores during pregnancy and twelve weeks postpartum. This suggests that depressive symptoms were relatively consistent across time-points, but that depression during pregnancy may be a particularly important indicator of later postpartum depressive symptoms.

Table 27. Pearson's Correlations between EPDS Scores during Pregnancy and at 1 and 12 Weeks Postpartum

	EPDS Week 1	EPDS Week 12
EPDS Pregnancy	.499**	.801**
EPDS Week 1		.684*

* $p < .05$, ** $p < .01$

8.7 Predicting Postpartum Depression Score

8.7.1 Regression Model to Examine which Variables Prospectively Predicted Week 1 EPDS Scores

Multiple linear regression was used to examine which pregnancy sleep variables were most predictive of postpartum depression. The purpose of this analysis was to ascertain the extent to which pregnancy sleep variables independently predicted early postpartum depression scores, when accounting for depression scores during pregnancy. It was important to account for initial depression scores, since the relationship between poor sleep and depression is bi-directional (Riemann et al., 2001), and therefore sleep disturbance during pregnancy could be a symptom of current depression rather than an independent predictor of postpartum depression. Given the number of participants, it was important to be selective with respect to the variables that were entered into the model. Based on the previously reported correlations, the pregnancy sleep variables that were most strongly related to week 1 depression scores were entered into a stepwise linear regression model. These were: subjective sleep onset latency and total amount of sleep. EPDS score during pregnancy was added to the model in order to examine whether these sleep variables would predict postpartum depression after accounting for prior symptoms. Due to the close relationship between subjective and objective sleep onset latency ($r = .514$), subjective sleep onset latency was used in this analysis, as it held a stronger relationship with EPDS scores.

Table 28. Stepwise Regression Analyses with EPDS Week 1 Score as the Outcome Measure

Predictor variable	R ²	Adjusted R ²	R ² change	F	Gradient	t	p
Model	.554	.504		11.01**			
Pregnancy Total sleep			.338**		.054	3.15	.006
Pregnancy sleep onset latency (subjective)			.216**		.055	2.95	.009

**p<.01; *p<.05

The result of the regression analysis (see Table 28) showed that total amount of sleep and sleep onset latency during pregnancy significantly predicted EPDS scores at week 1, accounting for 55.4% of the sample outcome variance (adjusted $R^2=.504$). Total amount of sleep during pregnancy accounted for the greatest variance (33.8%), followed by subjective sleep onset latency which independently accounted for 21.6% of the variance. The model excluded EPDS score during pregnancy as it did not significantly contribute to the model. This suggests that total amount of sleep and sleep onset latency during the last trimester of pregnancy were better predictors of early postpartum depression score than depressive symptoms during pregnancy.

In order to assess whether the same outcome would be found when using objective sleep onset latency, the regression was repeated using this variable in place of subjective sleep onset latency. Almost identical findings were found when using objective sleep onset latency instead of subjective sleep onset latency ($R^2=.498$), highlighting the accuracy of this subjective measure. Therefore, increased total sleep time and longer sleep onset latency in pregnancy increased the risk of developing early PPD symptoms, independent of whether the woman experienced depressive symptoms during pregnancy.

Given the strong cross-sectional relationship that existed between sleep onset latency and depression in the early postpartum period ($r = .695$), Pearson's correlation was used to examine the relationship between sleep onset latency during pregnancy and in the early postpartum. A strong relationship was found between sleep onset latency at both of these time-points ($r = .851$, $p < .001$), suggesting that women who experienced difficulty falling asleep during late pregnancy experienced similar difficulties during the early postpartum period.

8.7.2 Regression Model to Examine which Variables Prospectively Predicted Week 12 EPDS Scores

A similar regression analysis was carried out to examine which variables significantly, prospectively, predicted week 12 postpartum depression score, after controlling for depression during pregnancy and 1 week postpartum. Again, given the sample size selected variables were entered into the model. These were the variables from pregnancy and / or week one postpartum that were most strongly correlated with week 12 EPDS score. The following variables were entered into the model: subjective sleep onset latency (pregnancy), total amount of sleep (pregnancy), sleep onset latency (week 1), EPDS (pregnancy) and EPDS (week 1). The results are presented in Table 29.

Table 29. Stepwise Regression Analyses with EPDS Week 12 Score as the Outcome Measure

Predictor variable	R ²	Adjusted R ²	R ² change	F	Gradient	t	p
Model	.641	.619		28.59**			
EPDS (pregnancy)			.641		1.08	5.35	>.001

**p<.01; *p<.05

The results of the regression analysis show that EPDS score during pregnancy was the only variable that significantly predicted week 12 EPDS scores, accounting for 64.1% of the sample outcome variance (adjusted $R^2=.619$). This suggests that the relationships between sleep onset latency (during pregnancy and at week 1), total sleep during pregnancy, and week 12 depression scores, were mediated by depression during pregnancy. Combined with the findings of the previous analysis, this suggests that although total amount of sleep and increased sleep onset latency during pregnancy contribute to early PPD symptoms, alone, they do not predict depression scores later in the postpartum period. Furthermore, depressive symptoms during pregnancy seem to be a better predictor of later postpartum depression scores than early postpartum symptoms. This could be due to the fact that depressive symptoms in the early postpartum period are common and relate to the postpartum blues, yet in the majority of women symptoms will disappear after 7-10 days (O'Hara, 1987). In contrast, depression during pregnancy may reflect a more stable measure.

8.8 Overview of Results

The aims of this chapter were to examine cross-sectional relationships between the different measures of sleep, fatigue and depression; to examine longitudinal relationships between these variables; and to examine which variables were most predictive of PPD symptoms, after controlling for baseline depressive symptoms. This was the first study to report upon these relationships using both prospective, subjective measures of sleep and polysomnography.

This study found that:

- (i) The factors that were most consistently moderately correlated with depression at each time-point were increased sleep onset latency and fatigue.
- (ii) Sleep stages (as measured through polysomnography) during pregnancy appeared to be more strongly related to week 12 postpartum depression scores, than pregnancy or week 1 scores, however these variables did not reach significance.
- (iii) Increased total amount of sleep and increased sleep onset latency during late pregnancy significantly predicted week 1 depression scores, over and above depressive symptoms during pregnancy.
- (iv) The strongest predictor of week 12 depression score was depressive symptoms during late pregnancy.

These findings highlight that particular aspects of sleep appear to be more strongly related to postpartum depressive symptoms, and that there is a difference between these relationships when measured cross-sectionally in comparison to prospectively. Each of these findings will now be discussed in turn.

(i) Cross-sectional relationships

Previous studies have found that women with postpartum depression experience poorer sleep than non-depressed women (Dorheim, Bondevik, Eberhard-Gran, & Bjorvatn, 2009; Huang et al., 2004). The majority of studies have relied upon retrospective measures of sleep which provide a global score, and therefore cannot provide specific information about which aspects of sleep are most relevant (Da Costa et al., 2006; Dorheim et al., 2009; Huang et al., 2004). However, no studies to date have examined the cross-sectional relationships between sleep and postpartum depression using both prospective, subjective measures and polysomnography. By doing so, this study was able to tease apart the specific variables that appeared to be most relevant to depression during the perinatal period.

The main findings in the cross-sectional analyses were that increased sleep onset latency and fatigue were the factors most consistently associated with depression. These findings have also been reported by others (e.g. Groer & Morgan, 2007; Posmontier, 2008; Swanson, Pickett, Flynn, & Armitage, 2011). For example, Swanson, Flynn, Wilburn, Marcus and Armitage (2010) found that difficulty falling asleep was the only

insomnia-related symptom that significantly predicted EPDS scores. Furthermore, this was confirmed by Posmontier (2008) using actigraphy.

The finding that perinatal depression was not significantly associated with sleep quantity has also been mirrored by others (e.g. Dorheim, Bondevik, Eberhard-Gran & Bjorvatn, 2009; Huang, Carter & Guo, 2004). Similar relationships between sleep and depression were also reported in the general sample in Chapter 4, where depression was more strongly related to sleep quality than sleep quantity. In both of these studies, sleep onset latency and fatigue were strongly associated with depression. This suggests that these factors are not unique to perinatal depression.

However, what was particularly interesting in this study was that, in contrast to previous studies (e.g. Da Costa et al., 2006; Dorheim et al., 2009; Huang et al., 2004), subjective sleep *quality* was not significantly associated with depression scores. The reason for this could be due to differences in the way that sleep was measured in this study, compared to previous studies. The previous studies that reported poorer sleep quality amongst depressed perinatal women used the PSQI, a retrospective measure which refers to the previous month (Da Costa et al., 2006; Dorheim et al., 2009; Huang et al., 2004). Since depression is characterised by negative thinking style, a cognitive bias towards negative information and negative autobiographical memory (Beck, 1964; Gotlib, 1983; Hamilton & Gotlib, 2008), depressed individuals may be more likely to recall their sleep as poor. In contrast, the present study employed a prospective measure of sleep, which may be less likely to be distorted by cognitive biases. In light of this, Dorheim, Bondevik, Eberhard-Gran and Bjorvatn (2009) found that women with PPD reported poorer sleep when measured retrospectively (PSQI) but not when measured prospectively (using actigraphy and sleep diaries). These findings suggest that reports of poor sleep quality in previous studies of women with PPD may in fact be a reflection of negative thinking rather than sleep deficits.

While certain aspects of these results are similar to those reported in the general population sample (Chapter 4), one difference emerged. Although sleep quality was not related to depression amongst perinatal women, it was related to depression in the general population sample, despite the fact that the same tool was used to measure subjective sleep. One reason for this apparent discrepancy could be due to the fact that both pregnant and non-pregnant individuals appear to base their perceptions of sleep quality on the number of nightly awakenings (see Chapter 6). In the study reported in

Chapter 6, pregnant women experienced significantly more awakenings than non-pregnant women, and were also more aware of these awakenings. However, in contrast to the general population sample, awakenings were not strongly associated with perinatal depression. The reason that nightly awakenings contribute to depression in the general population but not in perinatal women may be due to the fact that nightly awakenings are to some extent characteristic of the perinatal period, and a substantial number of these awakenings are likely to be due to infant awakenings rather than the mother waking up of her own accord. In contrast, in non-pregnant individuals, nightly awakenings may reflect personal difficulties with maintaining sleep and may be indicative of insomnia, and therefore more are likely to have a negative effect on mood. However, it is also possible that a relationship between sleep quality, awakenings and postpartum depression may have arisen had the sample been larger.

(ii) Relationships between polysomnographic sleep and depression

Another unique aspect of this study is that sleep was also measured objectively during late pregnancy, using PSG, considered the 'gold standard' measure of sleep (Kushida et al., 2005). In terms of cross-sectional findings, increased total sleep time in the third trimester as well as decreased amounts of stage 2 sleep were moderately related to increased symptoms of depression during pregnancy. None of the other sleep stage variables were significantly related to depression.

What is particularly interesting is that sleep architecture during pregnancy appeared to be more strongly related to week 12 postpartum depression scores, than pregnancy or week 1 scores. There were moderate to strong correlations between the percentage of time spent in stages 1, 2 and 3 sleep and REM latency during pregnancy, and week 12 depressive symptoms. More stage 1 sleep, less stage 2 sleep, more slow-wave sleep and increased REM latency appeared to be associated with greater postpartum depression scores at week 12. Although these correlations did not reach significance, they suggest that particular aspects of sleep architecture during pregnancy may be indicative of postpartum depression. These relationships may have reached significance given a larger sample.

However, these patterns do not seem to fit with what is known about the relationship between sleep architecture and depression. In particular, reduced REM latency and decreased slow-wave sleep appear to be typical of depression (Kupfer et al., 1984; Lee et

al., 1993; Pillai, Kalmbach, & Ciesla, 2011), with reduced REM latency shown to be a marker for individuals who are at risk of developing depression (Giles et al., 1990; Kupfer et al., 1984; Modell & Lauer, 2007). Opposite findings appeared to emerge within this study, with positive relationships between both REM latency, amounts of slow-wave sleep and depressive symptoms.

Only one other study has cross-sectionally examined sleep in women with postpartum depression using polysomnography (Godfroid, et al., 1997). Interestingly, this study also reported an increase of slow-wave sleep in women with postpartum depression. However, this study has been criticised since women slept in a sleep laboratory, away from their infant and therefore may have relished the opportunity to catch up on some much needed sleep. Only one study has examined longitudinal relationships between sleep architecture during pregnancy and postpartum depression. Lee et al. (2000) found that a reduction in REM latency from late pregnancy through to the early postpartum period was associated with negative affect. However, this type of analysis was not possible in the present study given that polysomnography was only carried out during pregnancy.

Clearly, more research is needed to examine whether relationships between sleep architecture and postpartum depression mirror those seen in major depressive disorder. It is also important to note that the majority of the sample in the present study did not experience a high level of postpartum depressive symptoms, and therefore it is also possible that a lack of variance in postpartum depression scores limited the reported effects. Previous research suggests that sleep architectural changes are dependent upon depression severity (Kupfer et al., 1984). Therefore, future studies would benefit from recruiting women with a history of depression who are more likely to experience postpartum depression.

(iii) Predicting early postpartum depression symptoms

Overall, there were differences between the variables that predicted depression scores at one and twelve weeks postpartum. The strongest predictors of depression scores during the first postpartum week were increased sleep onset latency, and increased amounts of sleep during the third trimester of pregnancy. This relationship remained significant whilst controlling for depression during pregnancy, suggesting that certain aspects of sleep during pregnancy can provide an important indication of women at risk of developing early PPD symptoms.

The findings suggest that women who receive more sleep during pregnancy are at greater risk of developing early PPD symptoms. This is not the first study to document this relationship. For example, Wolfson et al. (2003) found that women with PPD at 2-4 weeks postpartum had significantly greater amounts of sleep during the third trimester of pregnancy than women without PPD. Okun, Hanusa, Hall and Wisner (2009) also found that women with a history of PPD who experienced early recurrence of symptoms (less than 4 weeks postpartum) received better sleep quality during the third trimester than those who recurred later in the postpartum period. Similarly, Lee et al. (2000) found that women with negative postpartum affect experienced more drastic changes in their amounts of sleep from pre-postpartum. Specifically, these women slept significantly longer during the third trimester, but significantly shorter in the early postpartum period, compared to women with positive affect.

The findings of Chapter 7 showed that women experienced around one and a half hours less sleep in the early postpartum period, compared to during late pregnancy. Therefore, it is quite possible that women who slept for longer during late pregnancy experienced more drastic changes to their sleep from pre to postpartum, which could have resulted in an increase in depressive symptoms.

(iv) Predicting week 12 postpartum depression scores

In contrast to symptoms of postpartum depression during the first postpartum week, when examining the factors that predicted week 12 depression scores, the sleep variables were no longer significant after accounting for depression scores during pregnancy. This suggests that although increased amounts of sleep and difficulty falling asleep during late pregnancy are predictors of early PPD symptoms (indicative of postpartum blues), the strongest predictor of week 12 PPD symptoms is depression during pregnancy. This highlights the importance of early detection of perinatal depressive symptoms. It also supports the decision in the latest version of the DSM-5 to change the 'postpartum onset' specifier to 'peripartum onset,' given that many women who develop PPD will exhibit symptoms during pregnancy (American Psychiatric Association, 2013).

On the other hand, this does not mean that sleep during pregnancy is not important to PPD. Increased sleep onset latency and longer sleep duration were significant predictors of week 1 depression scores, and there was a strong relationship between depression scores at weeks 1 and 12. Therefore, addressing sleep during pregnancy could help to

reduce the overall severity of PPD symptoms. Possible strategies to improve these aspects of sleep are discussed in the following chapter.

8.9 Chapter Summary

Overall this chapter described the final part of the study, which examined the relationships between sleep, fatigue and depression throughout the perinatal period. This was the first longitudinal study to analyse a range of both subjective and objective sleep variables and examine their relationship to both early and later symptoms of postpartum depression. This study found that increased sleep onset latency and fatigue are the factors that are most strongly associated with depression during the perinatal period. However, the factors that appear to play a role in the development of early PPD symptoms include increased sleep onset latency and increased amounts of sleep during pregnancy. Both sleep and depression during late pregnancy are important factors in the development of postpartum depression, and should therefore be given more attention. In contrast, the polysomnographic sleep variables showed little relationships with postpartum depression.

The following chapter provides a full discussion of the results of this study, and summarises how the findings of this thesis have contributed to knowledge within this research area. It also describes implications of the research, evaluates the methodology used in the thesis, and provides suggestions for future research.

CHAPTER 9: GENERAL DISCUSSION

9.1 Overview of Key Findings

The primary study described in this thesis was the first to examine the longitudinal relationship between sleep and postpartum depression (PPD) using both subjective, prospective measures of sleep and objective polysomnography (PSG). The need for this type of study was acknowledged in a review by Ross, Murray and Steiner (2005) who stated that longitudinal studies using PSG are warranted in order to 'confirm the hypothesis that sleep disruptions during pregnancy and the early postpartum period are associated with future risk for postpartum depression' (p. 253). The majority of studies to date have: failed to control for depression at baseline (e.g. Wilkie & Shapiro, 1992); relied upon subjective (often retrospective) measures of sleep (Dennis & Ross, 2005; O'Hara & Swain, 1996; Wilkie & Shapiro, 1992; Wolfson et al., 2003); used actigraphy as an objective measure rather than PSG (Bei, Milgrom, Ericksen, & Trinder, 2010; Dorheim, Bondevik, Eberhard-Gran, & Bjorvatn, 2009; Posmontier, 2008); and only measured PPD symptoms in the early postpartum period (Bei, Milgrom, Ericksen, & Trinder, 2010; Coe Calcagni, Bei, Milgrom, & Trinder, 2012; Deepika Goyal, Gay, & Lee, 2009; O'Hara & Swain, 1996).

As well as examining the relationship between sleep and PPD, this thesis also provided a detailed examination of sleep during the perinatal period, in order to provide further information relating to the nature of sleep changes during this time. Furthermore, this was the first study to specifically compare the relationship between subjective and objective measures of sleep in a perinatal sample.

The key findings of this thesis were:

1. Women experience significant changes to their sleep during the perinatal period. Firstly, by comparing sleep in pregnant and non-pregnant women using both objective (PSG) and subjective measures, this study was able to add to the limited literature examining sleep architectural changes associated with late pregnancy. Compared to non-pregnant women, pregnant women in their third trimester experienced significantly less total sleep time, more awakenings and more difficulty falling asleep. These findings were confirmed both subjectively and objectively. They also reported more daytime napping and poorer subjective perceptions of sleep quality. In terms of sleep architecture, pregnant women had

significantly more stages 1 and 2 sleep, poorer sleep efficiency and less REM sleep. However, no significant differences were found in relation to slow-wave sleep (stages 3 and 4). The factor of sleep most strongly associated with subjective sleep quality was nightly awakenings, suggesting that this appears to be what drives reports of poor sleep quality among perinatal women, and thus highlighting the importance of sleep continuity.

2. When examining longitudinal changes to subjective sleep throughout the perinatal period, women reported the poorest sleep quality, reduced sleep quantity and most fatigue during the first postpartum week, compared to late pregnancy or 12 weeks postpartum. This period was characterised by over one and a half hours less total sleep than during pregnancy, even when including daytime napping. This highlights the scope of changes to sleep patterns that occur over a relatively short period of time in the perinatal period, particularly in the transition from pre- to postpartum.
3. Among both pregnant and non-pregnant individuals, symptoms of depression appear to be most strongly related to increased reports of fatigue and increased sleep onset latency, suggesting that these are useful factors for screening current symptoms of depression. This also raises the question of whether perinatal depression is unique in this respect. However, whether these reflect a symptom or 'cause' of depression cannot be inferred from cross-sectional analyses alone.
4. The final part of the thesis identified increased sleep onset latency and increased total amount of sleep during late pregnancy as significant predictors of early postpartum depression scores, after controlling for baseline symptoms of depression. This suggests that these are important markers of early postpartum depression. However, the strongest predictor of postpartum depression scores at 12 weeks was depressive symptoms during pregnancy.

9.2 Explanations of Key Findings

Within primary care, symptoms of depression are typically viewed as either psychological (low mood, loss of interest, poor concentration, and associated anxiety) or somatic (fatigue, sleep disturbance, changes in appetite, general aches and pains; Kapfhammer, 2006; Tylee & Gandhi, 2005). The term 'somatic' can refer to symptoms that are physical, bodily, vegetative, medically unexplained or masked (Tylee & Gandhi, 2005). Despite the

fact that over two thirds of patients in primary care present with somatic symptoms, it is typically psychological factors that are used to detect depression and receive the most attention (Tylee & Gandhi, 2005). Current theoretical investigations of the relationships between sleep and depression appear to reflect the perceived somatic nature of sleep disturbances. For example, recent research has suggested that disturbed sleep and fatigue among those with depression have a neurochemical basis, modulated by neurotransmitters such as norepinephrine and serotonin (Demyttenaere, De Fruyt, & Stahl, 2005; Kapfhammer, 2006). Other recent research has pointed towards inflammation as a potential mediator of the relationship between sleep disturbance and depression, recognising specific pro-inflammatory cytokines such as interleukin-6 that may modulate both sleep and mood-regulatory processes (Makhija & Karunakaran, 2013; Motivala, Sarfatti, Olmos, & Irwin, 2005).

It is only in more recent years that research has begun investigating the relationship between poor sleep and the development of postpartum depression, and theoretical explanations of this relationship are lacking. However, some researchers have postulated that inflammation (Okun, Hall, & Coussons-Read, 2007; Okun, Roberts, Marsland, & Hall, 2009) and / or circadian factors (Sharkey et al., 2013) may play an underlying role in this relationship. In contrast, there is a paucity of research examining the role of cognitive and psychological factors in the relationship between sleep problems and fatigue among those with both major and postpartum depression.

Within this thesis, the factors that were most strongly related to depression in both pregnant and non-pregnant individuals were fatigue and increased sleep onset latency. Given that both of these factors involve cognitive elements (Deale, Chalder, Marks, & Wessely, 1997; Moss-Morris & Petrie, 2001; Roth, 2007) it is possible that the relationships between sleep disturbance and depression may indeed be modulated by cognitive factors. Difficulty falling asleep is a classic symptom of insomnia (Roth, 2007), the aetiology of which is known to be largely cognitive (Espie, 2007; Harvey, 2002). Cognitive models of insomnia have suggested that worry and rumination about life stresses can initially result in insomnia-related symptoms, such as difficulty falling asleep. However, once sleep difficulties are experienced, an individual may start to worry about sleep itself, and the potential consequences of not getting enough sleep. As a consequence this can lead to hyper-arousal and selective attention to internal and external cues, which make it more difficult to fall asleep (Harvey, 2002).

While the cross-sectional findings of this thesis suggest that the particular aspects of sleep associated with depression are similar in both major and perinatal depression, these findings cannot provide information regarding the temporal nature of this relationship; i.e. whether fatigue and difficulty falling asleep reflect a cause of consequence of depression. However, when examining the factors that were prospectively associated with the development of postpartum depressive symptoms, the relationship between fatigue and postpartum depression was no longer significant. This suggests that fatigue is more likely to reflect a symptom of depression, rather than a factor associated with its development.

The longitudinal part of this thesis highlighted the uniqueness of sleep during the perinatal period. Not only do pregnant women report poorer sleep, but there are fundamental differences in their sleep architecture when compared to non-pregnant women. Furthermore, between late pregnancy and the early postpartum period, women experience sudden and drastic changes to their sleep. The final aim was to assess whether particular aspects of sleep during this period increased women's risk of developing symptoms of PPD.

Previous research has suggested that poor 'sleep quality' appears to be characteristic of women at risk of, or currently experiencing, postpartum depression (Bei et al., 2010; Da Costa, Dritsa, Rippen, Lowensteyn, & Khalifé, 2006; Dorheim, Bondevik, Eberhard-Gran, & Bjorvatn, 2009; Huang, Carter, & Guo, 2004). However, this thesis has emphasised the complexity of sleep and the various ways in which it can be measured. The majority of these studies have relied upon a global, retrospective measures of sleep quality, typically the Pittsburgh Sleep Quality Index (PSQI). It was argued that this measure may not provide an accurate or detailed enough understanding of this relationship, given that a) depression is associated with negative cognitive biases that may distort perceptions of sleep (Beck, 1967), and b) perinatal women experience vast changes to their sleep over relatively short periods of time, which would not be accurately represented by a measure that refers to sleep over the previous month. This was supported by Dorheim et al. (2009) who found relationships between sleep and PPD when sleep was measured retrospectively using the PSQI, but not when measured prospectively. Therefore, the claim that poor subjective sleep quality predicts postpartum depression appears to be based upon limited research using somewhat biased measures.

Furthermore, studies to date have tended to rely upon subjective measures, and the majority of those that have used an objective measure have used actigraphy rather than

PSG (Bei et al., 2010; Dorheim et al., 2009; Posmontier, 2008). However, actigraphy does not provide information on sleep architecture, which may be particularly relevant to depression. For example, reduced REM latency has been shown to reflect a risk factor for the development of depression (Giles et al., 1987, 1998), and there is preliminary evidence to suggest that alterations in REM sleep may also play a role in the development of PPD (Coble et al., 1994; Lee, McEnany, et al., 2000).

Out of all of the sleep variables examined within this thesis (including prospective sleep diary measures and PSG variables), those that were most predictive of early (one week) PPD symptoms were increased sleep onset latency and increased total amount of sleep during late pregnancy. This relationship was significant even after controlling for depression during pregnancy, suggesting that these may reflect potential risk factors for PPD. In contrast, there was not sufficient evidence to suggest that aspects of sleep architecture measured through PSG were associated with the development of PPD. The relevance and implications of each of these factors are described in turn.

Increased Sleep Onset Latency

Following the findings of this study, there appears to be increasing evidence to suggest that difficulty falling asleep is an important factor associated with the development of PPD (Goyal, Gay, & Lee, 2007; Swanson, Pickett, Flynn, & Armitage, 2011). Goyal, Gay and Lee (2007) concluded that trouble falling asleep may be the most relevant screening question in detecting women at risk of postpartum depression. Furthermore, in an investigation of the relationship between insomnia and PPD, Swanson, Pickett, Flynn and Armitage (2011) found that difficulty falling asleep was the strongest predictor of PPD. Swanson et al. (2011) also reported that symptoms of insomnia amongst pregnant women were related to anxiety. Pregnancy is a time during which anxiety is common (Huizink, Mulder, Robles de Medina, Visser, & Buitelaar, 2004; Skouteris, Wertheim, Rallis, Milgrom, & Paxton, 2009; Swanson et al., 2011). Specific anxieties may relate to fear of giving birth, fear of bearing a handicapped child and concerns about one's appearance (Huizink et al., 2004). Therefore, it is not surprising that as parturition approaches, women may experience increasing anxiety, which may impede the ability to fall asleep with ease. In the present study, there was strong consistency between pregnancy and postpartum sleep onset latency, suggesting that women who struggled to fall asleep during pregnancy continued to do so after birth, when there may be even more pressure to fall asleep quickly in order to

maximise sleeping opportunities. Increased sleep onset latency was also associated with depression at each of the postpartum time-points.

While the relationship between insomnia and major depression is well-established (Chang et al., 1997; Ford & Kamerow, 1989), few studies have examined whether cognitive treatments of insomnia may prevent the risk of depression. Furthermore, there is a paucity of research investigating insomnia among perinatal women. Cognitive-behavioural therapy for insomnia (CBT-I) has been shown to be an effective treatment for individuals with insomnia (Harvey & Tang, 2003) and also those with co-morbid insomnia and depression (Manber et al., 2008). However, only one study to date has examined the use of CBT-I as a treatment for perinatal women (Swanson, Flynn, Adams-Mundy, Armitage, & Arnedt, 2013). In a pilot study, Swanson et al. (2013) examined the effectiveness of a five week CBT-I intervention with twelve postpartum women with insomnia and depression. Improvements were seen in relation to fatigue, sleep quality, awakenings, mood and insomnia severity, suggesting that this is an effective treatment for postpartum women. A larger trial examining the effectiveness of CBT-I in perinatal women is currently underway at Stanford University (Rachel Manber, personal communication, June 2013).

Increased amounts of Sleep during Late Pregnancy

The second factor that significantly predicted early postpartum depression symptoms was increased amount of sleep during late pregnancy. From first glance this finding could seem counter-intuitive; as one may expect that increased amounts of sleep would reduce the risk of depression. However, as with the former finding, this is not the first study to suggest that sleeping more in late pregnancy may increase the risk of PPD. f et al. (2003) found that women with PPD at 2-4 weeks postpartum had significantly greater amounts of sleep during the third trimester of pregnancy than women without PPD. Similarly, Lee et al. (2000) found that women with negative postpartum affect experienced more drastic changes in their amounts of sleep from pre-postpartum. Specifically, these women slept significantly longer during the third trimester, but significantly shorter in the early postpartum period, compared to women with positive affect.

This thesis showed that women experienced around one and a half hours less sleep in the early postpartum period, compared to during late pregnancy, supporting the findings of Signal et al. (2007). Therefore, it is quite possible that women who sleep longer during late pregnancy experience more drastic changes to their sleep from pre to postpartum, which

could result in an increase in depressive symptoms. Although circadian factors were not specifically examined in this thesis, it is possible that such factors could provide an explanation for these findings. Dramatic changes to sleep patterns from pre to postpartum would have affected women's exposure to light and dark cues and therefore would be likely to affect the circadian system. The phase-shift hypothesis of depression postulates that mood disturbances result from a phase delay or advance of the suprachiasmatic nucleus and related circadian rhythms that control temperature, cortisol, melatonin and REM sleep, relative to other rhythms (Germain & Kupfer, 2008). One way of measuring circadian phase is to measure dim light melatonin onset (DLMO), which is considered to be the period during which the biological 'sleep gate' opens (Shochat et al., 1997). Using this method, Sharkey, Pearlstein and Carskadon (2013) found significant changes to circadian phase position among postpartum women (particularly phase delay and evening circadian preference), which were correlated with postpartum depressive symptoms. While this was only a preliminary study with a small sample, the findings indicate that changes to sleep patterns during the perinatal period affect circadian systems, and that these changes may be associated with future risk for postpartum depression. A larger study is currently underway at Brown University (Katherine Sharkey, personal communication, June 2013).

Although more research of this nature is needed, this finding raises the question of how (or whether) women should prepare for the sleep changes that likely await them in the early postpartum period. For example, a gradually advancing sleep schedule combined with morning bright light has been shown to be effective at advancing circadian rhythms before eastwards flights, with the potential to prevent or reduce subsequent jetlag (Eastman, Gazda, Burgess, Crowley, & Fogg, 2005). While this may seem a crude comparison, it does raise the possibility that if circadian phase shifts are associated with PPD then how do we go about reducing this risk?

Bright light therapy is another possible treatment that could be used to help to stabilise the circadian system. For example, bright light treatment has been shown to be effective at reducing negative symptoms of chemotherapy (shown to disrupt circadian rhythms) among breast cancer patients (Ancoli-Israel et al., 2012; Neikrug et al., 2012). Furthermore, administering bright light in the morning and reducing exposure to bright light during the evening hours can increase evening levels of melatonin, reducing difficulties in falling asleep (which predicted PPD in this study) (Gooley, 2008). Only two studies to date have

examined the use of bright light therapy as a treatment for women with postpartum depression, both of which had small sample sizes ($n=2$, $n=15$) and reported conflicting results (Corral, Wardrop, Zhang, Grewal, & Patton, 2007; Corral, 2000). However, significant improvements to depressive symptoms have been shown using a randomised controlled trial among women with antepartum depression (Wirz-Justice et al., 2011). Future studies would benefit from examining whether continued administration of bright light therapy through late pregnancy and the postpartum period could help to stabilise the circadian system, and therefore reduce the impact of sleep disruption, and the likelihood of developing PPD.

Depression during Pregnancy

Despite moderate correlations between sleep variables during late pregnancy and depressive symptoms at 12 weeks postpartum, the only variable that significantly predicted week 12 postpartum depression scores was depressive symptoms during pregnancy. This highlights the importance of early detection of perinatal depressive symptoms, and supports the decision in the latest version of the DSM-5 to change the 'postpartum onset' specifier to 'peripartum onset,' given that many women who develop PPD will exhibit symptoms during pregnancy (American Psychiatric Association, 2013). Furthermore, depression during pregnancy was a better predictor of week 12 depression score than depression during the first postpartum week. Given the hormonal changes associated with birth and postpartum blues (O'Hara, Schlechte, Lewis, & Wright, 1991), it may be that depression during pregnancy provides a more stable and accurate measure of those at risk for PPD.

9.3 Methodological Issues and Future Research

Remarkably, this was the first study of its kind to adopt both subjective measures of sleep and PSG in order to examine longitudinal relationships between sleep and PPD. However, one of the reasons for this may be the practical difficulty of obtaining such data, given the demands that this type of study places upon participants who are busy preparing for the significant life event of having a baby. Therefore, the majority of research to date has used small samples, and thus current assumptions are based upon a relatively limited data set. This study used a sample size that was comparable, if not larger than other studies (e.g. Hertz et al., 1992; Lee, McEnany, & Zaffke, 2000; Tsai & Thomas, 2012). What sets this study apart from others is the use of a longitudinal design, the use of both subjective and objective measures and the use of a control group of non-pregnant women. However,

further studies are needed to confirm the reliability of these findings and increase power, since this has been overlooked in the majority of studies to date. Multi-centre studies that adopt carefully structured recruitment strategies are needed in order to gain the necessary sample size, so that more rigour can subsequently be applied to theory and practice. Future studies should also consider strategies to recruit participants from a range of cultural and socio-economic backgrounds, since it is likely that women who volunteer to take part in such studies are those that have sufficient social support or a higher educational background.

By using PSG this study was able to add to the very limited research examining the relationship between sleep architecture and PPD. While this method did not uncover any significant findings in relation to PPD, some trends did emerge in relation to particular variables, such as a relationship between decreased stage 2 sleep during pregnancy and the development of PPD. Furthermore, the findings also add to the literature on the effects of late pregnancy on sleep architecture. However, future studies adopting a control group of non-pregnant women should also consider controlling for menstrual phase, given that the menstrual cycle can affect sleep architecture, which could have confounded some of the differences seen in sleep architecture between pregnant and non-pregnant women seen in this study (Lee et al., 2000; Parry et al., 2006).

While this study did not report significant relationships between EEG measures and PPD, the data was analysed using traditional sleep scoring methods, rather than looking at signal frequencies through spectral analysis. Previous research has found that those with depression show reduced power density in slow-wave activity (Borbély et al., 1984). Some research has suggested that spectral analysis may have the potential to uncover changes in sleep that may not be picked up through typical sleep scoring (Edinger & Krystal, 2003; Hertz et al., 1992). Therefore, future studies may benefit from incorporating this type of analysis in order to see whether any differences emerge.

This was the first study to specifically report upon the relationship between subjective and objective sleep among perinatal women. The findings showed that pregnant women appear to be somewhat less accurate in their estimations of total sleep time, which was related to the finding that their sleep was significantly poorer. While this could be due to reduced cognitive functioning as a result of sleep disruption, it may also be that when sleep is of poor quality and fragmented, it is simply more difficult to recall the exact

amount of sleep that was obtained. This highlights the need to incorporate objective measures of sleep, particularly among populations that experience disturbed sleep.

In contrast, pregnant women were considerably more accurate at estimating their number of awakenings compared to non-pregnant women. This may be due to longer and more frequent awakenings among pregnant women that were therefore more easily remembered. Furthermore, number of awakenings was the factor that was most strongly related to sleep quality among both pregnant and non-pregnant women, suggesting that perceptions of sleep quality are strongly based upon the number of awakenings. In contrast, the findings did not support the viewpoint that subjective sleep quality is dependent upon amounts of slow-wave sleep (Keklund & Akerstedt, 1997; Saletu, 1975). In order to build upon these findings, future studies incorporating subjective and measures of sleep should directly report correlations between these measures.

This thesis also highlighted the importance of prospective versus retrospective measures of sleep. While more global measures of sleep, such as the PSQI, may provide a good starting point in which to examine whether poor sleep is related to a particular disorder, more detailed, prospective measures are needed to develop a better understanding of such relationships. In particular, this thesis has shown that specific aspects of sleep appear to more relevant to depression than others, which would not have been found if using a global measure. A better understanding of the specific aspects of sleep that are most relevant to depression will also allow for the exploration of possible theoretical underpinnings, which are currently lacking. Furthermore, it is also important to consider the time-frame in which particular measures of both sleep and depression refer to. This is particularly important in relation to postpartum depression where both sleep and mood can change over short periods of time, therefore it is important to have a measure that is time-specific.

This study used the most validated measure of postpartum depression, the Edinburgh Postnatal Depression Scale (EPDS). However since this is designed as a screening scale rather than a clinical diagnosis, future studies may benefit from incorporating postpartum clinical interviews. The research investigating sleep and PPD, to date, has used an array of measures of PPD (described in Chapter 3). Many of these measures are not specifically designed for use in postpartum women. However, a major issue is that currently there are only a handful of scales that have been validated for use in this sample. Even if using a DSM-5 based clinical diagnosis, a woman will only be diagnosed with PPD if symptoms

arise within 4 weeks of birth, despite research showing that symptoms develop much after this time (Gjerdingen, Crow, McGovern, Miner, & Center, 2011; Jones & Cantwell, 2010). Therefore it is important for future research to identify alternative ways of measuring PPD.

9.4 Implications

Some possible interventions based on the findings of this thesis have already been described. In particular, the use of CBT-Insomnia and bright light therapy may offer potential benefits for perinatal women, both in relation to the treatment and prevention of PPD. However, to date, there are little to no randomised-controlled trials (RCTs) to demonstrate these effects. Since perinatal women are often reluctant to take antidepressant medication due to fears of harming the baby, it is estimated that only 15% of women with postpartum depression receive treatment (Ng, Hirata, Yeung, Haller, & Finley, 2010). Women are warned about the potential harmful effects of medication during pregnancy (Campagne, 2007) and therefore women with a history of depression may decide to stop taking medication during their pregnancy. Research into non-pharmacological interventions, such as these, is therefore important.

The main RCTs that have been carried out are based upon behavioural sleep interventions. Several trials have found behavioural interventions to be effective in reducing reports of infant sleep problems (Hiscock & Wake, 2002; Hiscock et al., 2007; Stremler et al., 2006; Wolfson, Lacks, & Futterman, 1992) and improving maternal mood (Hiscock & Wake, 2002; Harriet Hiscock, Bayer, Hampton, Ukoumunne, & Wake, 2008). The majority of these trials have targeted postpartum women and have compared behavioural sleep interventions to a usual care group with basic sleep information (Hiscock et al., 2007; Stremler et al., 2006, 2013). For example, Hiscock and Wake (2002) carried out an intervention with 156 postpartum women with infants aged between 6-12 months. Participants were randomised to either a control group with written information about infant sleep, or a three-part intervention where women received three private consultations providing tailored sleep management plans and information about controlled crying. Those in the intervention group subsequently reported less infant sleep problems and reduced maternal depression.

To date, however, the majority of interventions have focused on infant sleep as the primary outcome. This thesis has highlighted the importance of maternal sleep quality in relationship to the development of postpartum depression, and particularly the

importance of sleep during late pregnancy. A recent study by Stremmler et al. (2013) involved a large multi-site intervention with 246 primiparous women in the early postpartum period, half of whom were randomised to a behavioural-educational sleep intervention while the other half received usual care. The intervention included a 45-60 minute meeting with a trained sleep nurse, a 20-page booklet elaborating on sleep strategies and three phone calls. The intervention covered maternal sleep hygiene, strategies for increasing sleep opportunities, maternal relaxation techniques and infant sleep strategies. While the intervention was effective in the pilot study (Stremmler et al., 2006), no significant differences were seen in relation to maternal or infant sleep or health between the two groups in the larger study. The authors speculated that the women who took part were likely to be highly motivated, and the completion of sleep-diaries by both groups may have helped participants in the usual care group to improve their sleep. Furthermore, fewer women reported the use of particular strategies such as progressive muscle relaxation and deep breathing, which suggests that more guided practice may have been needed. It is also possible that the intervention was carried out too early in the postpartum period, given that this is a period of intense change and adjustment and therefore implementing sleep strategies may not be a priority for many families.

Since late pregnancy appears to be a time during which sleep problems are common, interventions during pregnancy could be useful. One such intervention is currently being carried out by researchers at the University of Sydney, whereby a brief psycho-educational sleep intervention in late pregnancy is being compared to a control group in order to examine whether the intervention improves sleep and reduces symptoms of postpartum depression (Kempler, Sharpe, & Bartlett, 2012). If successful, such low cost interventions have the potential to be integrated into standard perinatal healthcare.

Other non-pharmacological interventions that may be useful in helping both sleep and depression during this period include mindfulness and / or mind-body practices. The practice of mindfulness (paying attention to the current state of the mind) has been shown to be effective at reducing symptoms of anxiety and depression in clinical samples (Hofmann, Sawyer, Witt, & Oh, 2010), as well as improving sleep (Britton, Haynes, Fridel, & Bootzin, 2012; Shapiro, Bootzin, Figueredo, Lopez, & Schwartz, 2003). Research has also highlighted the benefits of yoga during pregnancy. For example, Satyapriya, Nagendra, Nagarathna and Padmalatha (2009) found that yoga reduced perceived stress in pregnant women and improved adaptive autonomic response to stress by increasing

parasympathetic activity. Beddoe, Paul Yang, Kennedy, Weiss and Lee (2009) found that women practicing mindful yoga during pregnancy experienced reductions in pain and reduced stress and anxiety. Furthermore, women who begin practicing yoga during their second trimester of pregnancy experience fewer awakenings, less time awake at night and less sleep disturbance, suggesting that yoga not only improves mood but also improves sleep (Beddoe, Lee, Weiss, Kennedy, & Yang, 2010). Pregnancy yoga has also been associated with decreased reported pain during labour and shorter duration of labour (Chuntharapat, Petpichetchian, & Hatthakit, 2008). A recent study by Field, Diego, Delgado and Medina (2013) found that pregnant women who partook in a 20 minutes group session of yoga / tai chi for 12 weeks reported significantly lower depression, had less negative affect, lower anxiety and fewer sleep disturbances, compared to a waitlist control group.

Although more research is needed to assess the efficacy of mind-body interventions during pregnancy, a review by Beddoe and Lee (2008) concluded that there is sufficient evidence to suggest that these practices lead to significant health benefits when used in conjunction with conventional prenatal care (Beddoe & Lee, 2008). Future RCTs with larger sample sizes and more structured protocols are needed to investigate whether mind-body interventions have the potential to improve sleep and reduce depression among perinatal women.

9.5 Conclusion

Overall this thesis has documented one of the first studies to examine the longitudinal relationship between sleep and postpartum depression, using both subjective and polysomnographic measures. The findings showed that while the sleep of third trimester women was considerably poorer than that of non-pregnant women (both objectively and subjectively), the most significant changes occur in the transition between late pregnancy and the early postpartum period. Furthermore, depression, increased amounts of sleep, and reports of difficulty falling asleep during late pregnancy significantly predicted postpartum depression scores, suggesting that these may reflect markers of women at risk. Future studies incorporating larger sample sizes are needed to build upon these findings, as are studies that examine whether circadian and cognitive factors may underlie these relationships. Such studies will help to build rationale for specific interventions targeting sleep that are likely to be most effective at reducing the risk of developing postpartum depression.

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APPENDICES

Appendix 1. St Mary's Hospital Sleep Questionnaire

This questionnaire refers to your sleep over the past 24 hours. Please try and answer every question.

Participant ID number:

Participant Initials:

Date:

At what time did you:

1) Settle down for the night?..... 2) Fall asleep last night?

3) Finally wake this morning?..... 4) Get up this morning?.....

5) Was your sleep (tick box)

Very light ☐

Light ☐

Fairly light ☐

Light average ☐

Deep average ☐

Fairly deep ☐

Deep ☐

Very deep ☐

6) How many times did you wake up? (tick box)

Not at all ☐

Once ☐

Twice ☐

Three times ☐

Four times ☐

Five times ☐

Six times ☐

More than **six** times ☐

How much sleep did you have...

7) Last night?hrs.....mins

8) During the day, yesterday?hrs.....mins

9) How well did you sleep last night? (tick box) If not well what was the trouble (eg restless, etc)?

Very badly	<input type="checkbox"/>		
Badly	<input type="checkbox"/>		1 _____
Fairly badly	<input type="checkbox"/>	→	2 _____
Fairly well	<input type="checkbox"/>		3 _____
Well	<input type="checkbox"/>		
Very well	<input type="checkbox"/>		

10) How clear-headed did you feel after
getting up this morning? (tick box)

Still very drowsy indeed	<input type="checkbox"/>
Still moderately drowsy	<input type="checkbox"/>
Still slightly drowsy	<input type="checkbox"/>
Fairly clear-headed	<input type="checkbox"/>
Alert	<input type="checkbox"/>
Very alert	<input type="checkbox"/>

11) How satisfied were you with last night's
sleep? (tick box)

Very unsatisfied	<input type="radio"/>
Moderately unsatisfied	<input type="radio"/>
Slightly unsatisfied	<input type="radio"/>
Fairly satisfied	<input type="radio"/>
Completely satisfied	<input type="radio"/>

12) Were you troubled by waking early and being unable to get off to sleep again? (tick box)

NO ☐ YES ☐

13) How much difficulty did you have in getting off to sleep last night? (tick box)

None or very little ☐

Some ☐

A lot ☐

Extreme difficulty ☐

14) How long did it take you to fall asleep last night? hrs..... mins....

Appendix 2. Pregnancy Screening Questionnaire

Screening questionnaire: The relationship between sleep and mood during pregnancy and the postpartum period



In order to assess whether you would be eligible for the study, I would be very grateful if you would take the time to answer the following questions:

(Please put an 'X' next to the appropriate box)

- 1) Are you currently diagnosed as suffering from an episode of depression?
Yes:

No:
- 2) Have you had one or more episode of depression before, which was diagnosed by a GP or clinician?
Yes:

No:
- 3) Are you currently diagnosed as suffering from bipolar disorder (manic depression)?
Yes:

No:
- 4) Have you ever previously been diagnosed as suffering from bipolar disorder?
Yes:

No:
- 5) Are you currently diagnosed as suffering from any other psychiatric disorder (if yes, please specify which disorder):
Yes: Disorder:

No:
- 6) Have you ever been diagnosed with any other psychiatric disorder? (if yes, please specify which disorder)
Yes: Disorder:

No:

7) Are you currently taking any antidepressant medication?

- Yes
- No

8) If you answered yes, please state the name and dose of the medication you are taking:

Name of antidepressant (e.g. Flouxetine 20mg): _____

Dose (e.g. 1 tablet twice a day in the morning and evening): _____

9) Are you currently taking any sleep medication?

Yes:

No:

10) Are you currently suffering from a diagnosed sleep disorder? If so, please specify which disorder:

Yes:	Please specify:	Insomnia
		Restless legs syndrome
		Sleep disordered breathing
		Narcolepsy
		Parasomnias
		Delayed sleep phase subtype
		Other

No:

If you have any queries relating to any of these questions, please contact the researcher:

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PARTICIPANT INFORMATION SHEET

Assessing the relationship between poor sleep and mood during pregnancy and the postpartum period

Chief investigator: Lauren Kita – PhD student

We would like to invite you to participate in our research. Before you decide we would like to explain what the research is about, why it is important, and what your participation will involve. The researcher will go through the information sheet with you in case you have any questions. It will take approximately 10 minutes. You can talk about the study with others if you wish. Please ask any questions you may have. You can contact the principal researcher or the project supervisor for any questions. If you decide not to participate it will not affect your treatment. Part 1 tells you the purpose of this research and what will happen to you if you take part. Part 2 gives you more detailed information about the conduct of the research.

PART 1

What is the purpose of the study?

Sleep is often disturbed in women during pregnancy and after the birth of a child. Research suggests that poor sleep may be a risk factor for depression. The purpose of the study is to assess whether poor sleep and fatigue during pregnancy leads to poor mood in the period after childbirth.

Why have I been invited?

You have been invited because you are currently pregnant. You may or may not have a history of depression.

Do I have to take part?

It is up to you to decide. Your participation in this study is entirely voluntary.

We will describe the study and go through this information sheet, which we will then give to you. If you agree to take part we will ask you to sign a consent form. If you agree to take part and later decide that you do not want to continue, you can withdraw at any time without giving a reason.

What will happen to me if I take part?

Before the study you will be required to take part in a short telephone interview which looks at mood and depressive symptoms. This will take no more than 20 minutes to complete. If the screening interview indicates that you have depression or symptoms of another psychiatric disorder, we will refer you to your health professional and you may have to be withdrawn from the study.

The first part of the study will take part in the third trimester of your pregnancy. You can discuss a date with the research team that is best for you. You will be sent a pack of questionnaires. One of the questionnaires asks questions about your sleep on the previous night, and you will be asked to fill this in every morning for seven days. On the last day, you will be asked to complete two short questionnaires on fatigue and mood, and one demographics questionnaire. This asks questions relating to personal information such as your education level and the number of children you have. During one night of this period you will also be required to undergo a night of sleep recording. Please see the section below for more details.

The second part of the study will be performed one week after you have given birth to your child. You will be asked to notify the researcher once you have given birth. You will receive another pack of questionnaires as before. The same procedure will apply but without any sleep recording.

The final part of the study will take part 12 weeks after you gave birth. You will be asked to complete the same process as before. However, you will not be required to complete a demographics questionnaire or complete a sleep recording.

What is sleep recording?

Sleep recording uses a device to measure brain activity, muscle movements and eye movements. A number of sensors will be attached to your scalp and face in order to monitor your sleep during the night. This provides

information about sleep timing and also the different stages of sleep that we pass through during the night.

What will the recording involve?

The researcher will visit you in your home to fit the device, or you may come to the university if you would prefer. The researcher will try to arrange a time so that the midwife will also be present. If this is not possible a fellow member of the research team will accompany the researcher. Electrode fitting will take around one hour. Electrodes will be placed on your scalp, around and behind your eyes, behind your ears and under your chin. The device is portable and allows you to move around freely. You may get up at any time in the night; the device is attached to a strap which can be placed around your shoulder. You do not need to do anything apart from sleep, as normal.

In the morning you can remove the electrodes yourself. The researcher will show you how to do this.

Is it safe?

This type of recording is considered completely safe. It does not involve exposure to radiation, nor does it involve any injections.

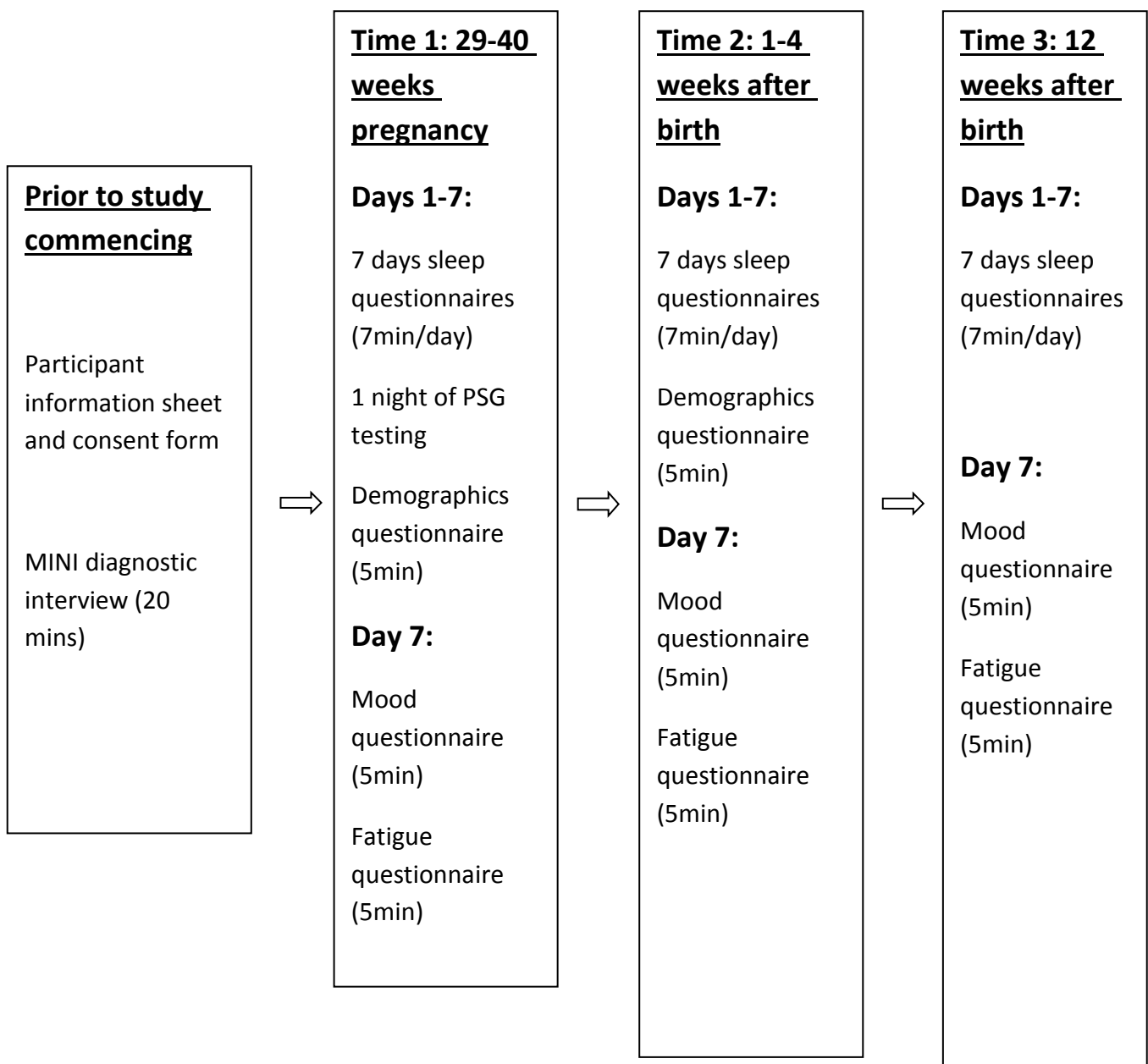
Do I need to prepare for the sleep study?

It is best to avoid alcohol, caffeine, sedatives and stimulants on the day of the test. No other preparation is required.



How long will the study last?

The study is split into three parts. The sleep recording will only take place at Time 1 (during pregnancy). At all other times you will be sent a pack of questionnaires to complete at home, which you can return to us in a prepaid envelope. Two of the questionnaires need to be completed continuously for 7 days during each time point.



What are the possible disadvantages and risks of taking part?

You may find it more difficult than usual to sleep whilst your sleep is being recorded. However, we encourage you to relax as much as possible and not to worry. You should carry on with your routine as you normally would do so. If you need to get up in the night, you can. The researcher will show you how to carry the device and how to plug it in once you return to bed. You might find some of the items on the questionnaire to be personal; however we will discuss the types of questions with you before you start. If you decide that you no longer wish to take part, you can do so without giving a reason.

What are the possible benefits of taking part?

We cannot promise that the study will help you personally. However, the results should help our understanding of the relationship between sleep and mood during pregnancy and after childbirth. This may help health professionals to identify women who may be at risk of experiencing poor mood and depression after the birth of their baby.

What if there is a problem?

Should you wish to make a complaint about the way you have been dealt with during the study or any possible harm you might suffer, you can. Detailed information is provided in part 2.

Will my taking part in the study be kept confidential?

Yes. We will follow ethical and legal practice and all information about you will be handled in confidence. The details are included in Part 2.

If the information in Part 1 has interested you and you are considering participation, please read the additional information in Part 2 before making any decision.

PART 2

What will happen if I don't want to carry on with the study?

If, for any reason, or at any time, you decide that you no longer wish to be involved in this study you are free to withdraw without giving a reason. Deciding to withdraw from the study will not affect your antenatal or postnatal care. If you decide to withdraw we would use the data collected up to your withdrawal with your permission. All data will be anonymous and will not contain any identifiable personal information.

What if there is a problem?

If you have a concern about any aspect of this study you should ask to speak with the researchers who will do their best to answer your questions. Contact details are at the end of this information sheet.

Will my taking part in this study be kept confidential?

If you join the study some of your medical records and the data collected for the study may be looked at by a NHS health professional. They may also be looked at by staff employed by the sponsor to check that the study is being carried out correctly. All will have a duty of confidentiality to you as a research participant and we will do our best to meet this duty.

All data collected from the study will be anonymised with a unique ID number that will be used on all documentation. No personal identifiable information will be used for the purposes of data collection for this study. All personal data will be destroyed at the end of the study.

All completed questionnaires, tests and other collected data will be stored at Bournemouth University in a secure locked cabinet for a period of 5 years after the end of the trial and then destroyed.

All electronic data will be kept on a secure computer and access to the data will be secured with passwords known only to the research team. No personal information will be reported in any publications that may result from this research.

If at any time during the study it is indicated that you are suffering from severe symptoms of depression, we are obliged to notify the health professional that is currently dealing with your antenatal / postnatal care.

Involvement of the General Practitioner/Family doctor.

If you agree to participate we will inform your GP that you are taking part in this study. We will ask your consent to inform your GP. If you do not wish your GP to be informed you may still take part in the study.

What will happen to the results of the research study?

The results of this research will be used as a part of a PhD dissertation. They might also be published in relevant scientific journals and presented at scientific conferences. No identifiable personal information will be used. When available, a summary of the results of the study may be obtained from the principal researcher.

Who is organising and funding the research?

This research is organized by Bournemouth University as a part of a PhD study.

Who has reviewed the study?

All research in the NHS is looked at by independent group of people, called a Research Ethics Committee, to protect your interests. This study has been reviewed and given favourable opinion by an NHS Research Ethics Committee.

Further information and contact details

You can contact the principal investigator for more information about this study:

Lauren Kita (Principal investigator): 01202 965049 / Mobile number 07923861452 (for any problems during sleep recordings) /
lkita@bournemouth.ac.uk

Dr. Andrew Mayers (Project Supervisor): 01202 961871 /
amayers@bournemouth.ac.uk

Some useful websites if you feel that you are experiencing sleep deprivation and / or depression:

- **NHS postnatal depression information:**
<http://www.nhs.uk/Conditions/Postnataldepression/Pages/Introduction.aspx>
- **American Academy of Sleep Medicine, information on sleep during pregnancy and postpartum:**
<http://yoursleep.aasmnet.org/topic.aspx?id=40>.

If you would like to talk to someone:

- **Samaritans 08457 90 90 90**
www.samaritans.org

Thank you for taking the time to read this information sheet. If you decide to take part in the study you will be given a copy of this information sheet and the signed consent form to keep.

Appendix 4. Participant consent form**CONSENT FORM****Assessing the relationship between poor sleep and mood during pregnancy and the postpartum period**

Lauren Kita (PhD researcher) / Dr Andrew Mayers

Centre Number:

Study Number:

Patient Identification Number for this trial:

Please INIT
each

1. I confirm that I have read and understand the information sheet dated..... (version.....) for the above study. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily. ☐
2. I understand that my participation is voluntary and that I am free to withdraw at any time without giving any reason, without my medical care or legal rights being affected. ☐
3. I give consent for my GP to be made aware of my participation in the study. ☐
4. I understand that relevant sections of my medical notes and data collected during the study may be looked at by individuals from the NHS Trust or the regulatory authority, where it is relevant to my taking part in this research. I give permission for these individuals to have access to my records. ☐
5. I agree to take part in the above study. ☐

Name of Participant Date Signature

Name of Person taking Date Signature
Consent

When completed, 1 for patient; 1 for researcher site file; 1 (original) to be kept in medical notes