

THE IMPACT OF LONG-TERM PARTIAL SLEEP DEPRIVATION
IN UNIPARA MOTHERS

By

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DECLARATION

I declare that this dissertation is my own, unaided work. It is being submitted for the Degree of Master of Science at the University of the Witwatersrand, Johannesburg.

It has not been submitted before for any degree or examination to any other university.

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ABSTRACT

Most sleep deprivation studies show increased body mass, increased hypertension, increased Type 2 Diabetes, obesity, cardiovascular issues and even death. There is a paucity of research in sleeping patterns of first time Caucasian mothers of babies between six and twelve months old in South Africa. The objective was to determine the impact of long-term partial sleep deprivation on metabolism and mood in these mothers.

A qualitative and quantitative study using a small sample of thirty one mothers was undertaken. Interviews assessed age, education, anthropometric data, family history, medication use, and baby sleeping habits. Participants were also asked to complete the Pittsburgh Sleep Quality Index (PSQI) and the Becks Depression Inventory II (BDI-II) to assess the mothers sleep quality and depressive state.

Medication use significantly decreased sleep quality. Oral contraceptive use and depression (BDI-II score 14-40) were associated with a significantly higher PSQI score and significantly decreased number of hours sleep and sleep efficiency. Oral contraceptive users were significantly more depressed. The six mothers on antidepressants had significantly higher PSQI scores than those not on antidepressants, but hours of sleep and sleep efficiency were not significantly different.

This study showed that sleep deprivation did not impact mothers anthropometry. Interestingly medication use affected sleep quality more than baby arousals.

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LIST OF ABBREVIATIONS

AMA	Arm Muscle Area
BDI-II	Becks Depression Inventory II
BMI	Body Mass Index
DOS	Date of Survey
EEG	Electroencephalogram
LH	Luteinizing Hormone
MDD	Major Depressive Disorder
MUAC	Mid Upper Arm Circumference
NREM	Non-Rapid Eye Movement
OC	Oral Contraceptives
PSQI	Pittsburg Sleep Quality Index
REM	Rapid Eye Movement
SD	Standard Deviation
SWS	Slow wave sleep
TSF	Tricep Skinfold (thickness)
WHR	Waist hip ratio

CHAPTER ONE - Literature Review

This paper describes the sleep and anthropometry in a group of mothers and babies (between five and thirteen months old) in Gauteng, South Africa. It evaluates different ways of measuring sleep quality and the relationship between sleep and anthropometric variables in the mothers. Factors other than baby sleep that could influence maternal sleep were investigated. The literature review will discuss general sleep physiology in adults and complications adult women encounter with sleep. Infant sleep under one year of age and the problems that the babies and their parents may encounter are discussed. There are many problems caused by sleep deprivation, such as, appetite changes, hormonal fluctuations, body mass variations, metabolic changes, hypertension, immunity decreases, development of Type 2 Diabetes, cardiovascular issues, moodiness, pain tolerance variances, memory loss, anxiety, and depression. This paper examined the metabolic effects of mothers (good sleeping compared to poor sleeping) using basic anthropometric recordings and self reported data. It also analyzes the most commonly used medications women take, six months after child birth and what effect on sleep they have. The paper examines the effect of oral contraceptives, depression and antidepressants on sleep.

1. Normal Sleep

Sleep is a natural periodic state of rest for the mind and body, in which the eyes close and consciousness is completely or partially lost. There is a decrease in bodily movement and responsiveness to external stimuli. During sleep, the brain in humans and other mammals undergoes a characteristic cycle of brain-wave activity that includes intervals of dreaming (Harris C, 2005). Sleep is not a continuous state throughout the night but consists of repetitive sleep cycles Fig 1.1. (Dement W *et al*, 1957) interrupted by brief arousals that are not recalled in the morning (Knab B, 1988).

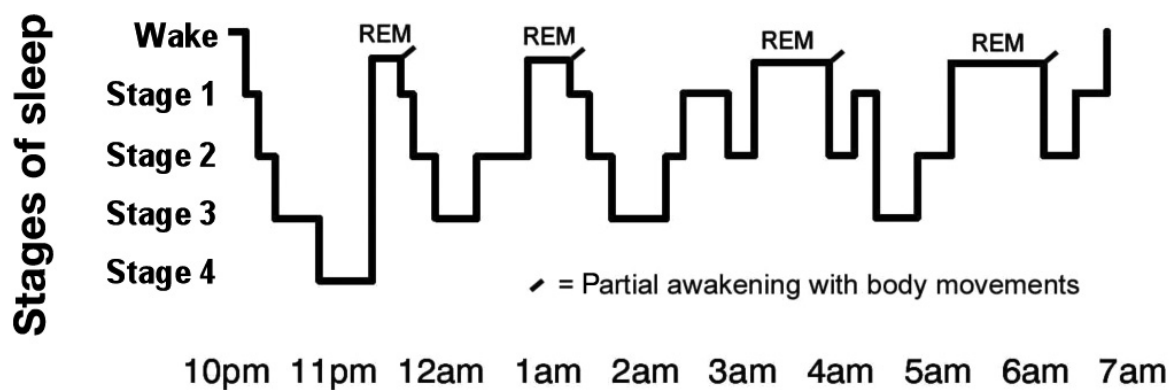


Figure 1.1. Sleep stage distribution during one typical night in an adult

The sleep cycle changes regularly throughout life and is influenced by several variables. The duration of slow-wave sleep is usually longest early on in a night's sleep (Koban M *et al*, 2006). Sleep cycles after the first cycle contain less slow-wave sleep, as the amount of time spent in sleep stages 3 and 4 shortens during the night. Sleep deprivation, breathing problems, illness, frequently changing sleep schedule, stress, medication, environment and age all affect the progression of the sleep cycle (Koban M *et al*, 2006). When it comes to sleep quantity and quality there have been differences found between genders.

1.1.Sleep in women

Healthy women appear to sleep better and to cope better with sleep loss or externally induced sleep disturbance, which might contribute to women's lower cardiovascular risks and greater longevity compared to men (Miller V *et al*, 2008). Women on average go to bed earlier and wake-up earlier than men (Adan A *et al*, 2002). A number of factors may affect women's sleep. Changes in hormonal levels, stress, body temperature, illness, lifestyle, and sleep environment may impact sleep (Moline M *et al*, 2004). The most common sleep study findings across the menstrual cycle include decreases in REM sleep, increases in stage 2 sleep and spindle frequency activity, and no changes in sleep onset latency and quality during the luteal phase compared to the follicular phase (Ishizuka Y *et al*, 1994). Sleep complaints commonly occur during the postovulatory luteal phase of the menstrual phase in healthy women (Manber R *et al*, 1997). Pregnancy and menstrual-related hormonal fluctuations may affect sleep patterns, mood, and reaction to stress.

1.2.Sleep in infants under one year of age

The development of sleep-wake patterns from multiple sleep episodes throughout the twenty four hour period to one consolidated sleep episode at night, and a few naps during the day is a complex process that challenges parents in the first year of life (Burnham M *et al*, 2002). The twenty four hour circadian cycle in infants becomes established at three to six months (Spruyt K *et al*, 2008) resulting in longer stretches of sleep at night and shorter naps during the day.

Babies six to twelve months old need about fourteen hours of sleep in a twenty four hour day with a normal range between nine hours and eighteen hours. The average amount of daytime sleep is about three to four hours split into a morning nap and an afternoon nap at three to six months of age. Some babies will nap for twenty minutes, others will sleep much longer than average (Pearl P, 2002). By six months, the baby's sleep architecture closely resembles that of an adult's. After an initial "settling" period that typically takes 10 to 20 minutes, the infant drifts from Stage 1 NREM sleep into Stage 2, 3 or 4. The infant returns to Stage 1 and cycles again (Montemitro E *et al*, 2008). After one to two cycles of NREM sleep, REM is entered at about 60 minutes. The first one third of the night is mostly deep sleep (NREM Stages 3 and 4). The last section of the night is predominately Stage 2 NREM and REM (Grigg-Damberger M *et al*, 2007).

Growing infants and young children spend around 50% of their total sleep time in REM sleep, compared to 20% for adults in a twenty four hour period. REM is believed to be important for normal brain development and may explain why infants spend 50% of sleep in active or REM sleep (Burnham M *et al*, 2002). Nighttime baby sleep should consolidate over the first year from many short (or long naps) into more regular naps and a longer period of time at night. At about four years old, the child should sleep in a single uninterrupted block of time at night. Figure 1.2. illustrates how sleeping patterns change in people of different ages (Website reference, as of 25 Dec 2012). As we get older, we spend less time sleeping. Infants spend well over 14 hours a day sleeping. As an infant grows, the duration of their periods of wakefulness increase. One year olds are beginning to have a more consolidated sleep schedule during the night, but still require naps during the day. Normal sleep is the quantity and quality of NREM and REM sleep necessary to refresh the child. As people age, naps consolidate into one block of sleep from before midnight until after 6am as an adult.

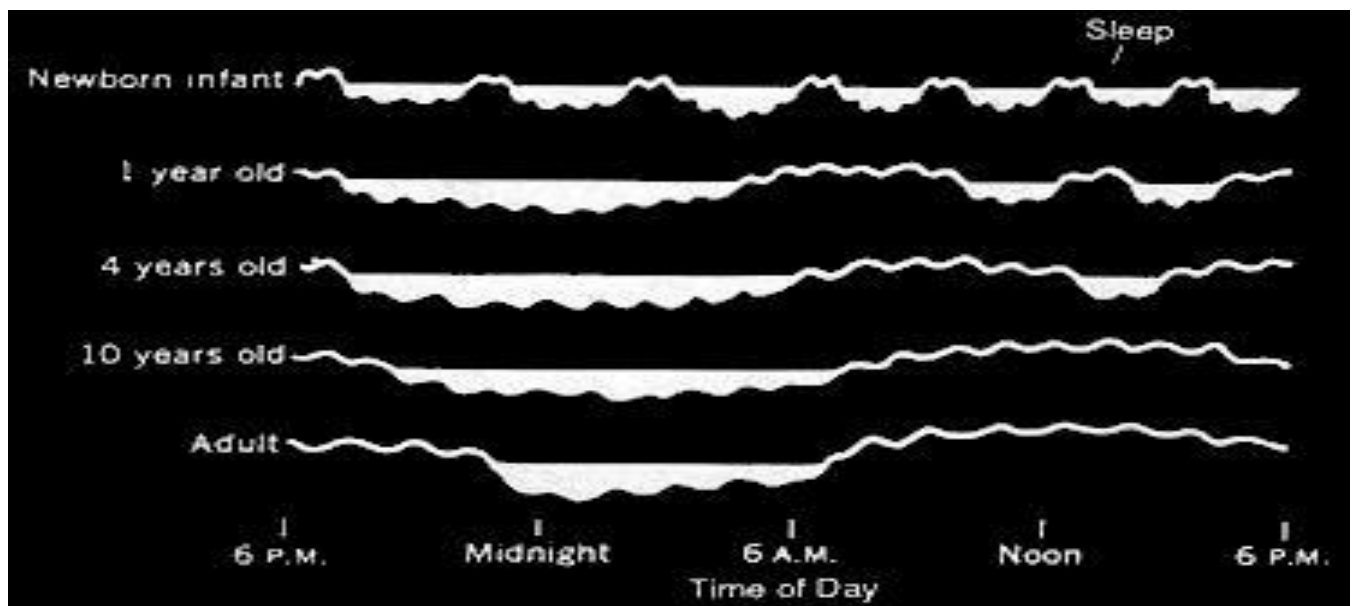


Figure 1.2. Sleep is illustrated by the white filled in sections and wakefulness by the white line.

1.3. Sleep problems in infants under one year of age

Babies do not inherently know how to fall back asleep on their own (International Paediatric Work Group of Arousals, 2005). A baby may depend on rocking or nursing to fall asleep. Many children are distressed when they awake and will cry for their parent (Adair R *et al*, 1993).

Male infants (3-6 months) old have been shown to have significantly more problems with sleeping continuously through the night compared to female infants in a study done by Bayer *et al* (2007). Mothers in this study also reported that these male infants were temperamentally difficult.

Sleep disorders during infancy can be due to behaviour or physical complications in the brain (Pearl P. 2002) and can be classified into two major categories, the parasomnias and the dyssomnias. Parasomnias involve behaviors or physiological events that interfere with sleep after sleep onset, and include disorders of arousal, partial arousal, or with transition between the stages of sleep (Mindell J *et al*, 1999). Common parasomnias include confusional arousals, sleepwalking, sleep terrors, nightmares, and rhythmic movement disorders. Parasomnias occur between 3 and 8 yrs of age and subside as the child matures (Dahl R. 1998). They fall outside the scope of this study and will not be discussed further.

Dyssomnias are defined as the disorders that make it difficult to initiate or maintain sleep. Delayed sleep-phase syndrome is a disorder in which the major sleep episode is delayed in relation to the desired clock time, resulting in symptoms of sleep onset or difficulty in awakening at the desired time (Mindell J. 1995). In advanced sleep-phase syndrome the

problem is that the major sleep episode is advanced in relation to the desired clock time, where subjects exhibit an early sleep onset (evening sleepiness) and an early awakening. Advanced sleep-phase syndrome is not common among children but delayed sleep-phase syndrome can develop before one year of age and into early childhood (Pearl P. 2002).

The most common dyssomnias in children under one year of age are sleep disruption, sleep-onset association disorder, inadequate sleep hygiene, sleep apnea and limit-setting sleep disorder.

Sleep disruption in children can be caused by noise, sickness or sleep hygiene. Sleep disruption also has serious implications for children. An inverse relationship has been found between sleep duration and anthropometrics in children in many studies (Anderson S *et al*, 2010, Locard E *et al*, 1992). If sleep duration increases then body mass, obesity, or measures of adiposity in infants, toddlers, and preschool age children decrease (Monasta L *et al*, 2010, Taveras E *et al*, 2008, Tikotzky L *et al*, 2010, Von Kries R *et al*, 2002, Sekine M *et al*, 2002, Tian Z *et al*, 2010).

Sleep-onset association disorder is a condition in which a child associates their ability to fall asleep with something in their environment or even a person. Examples of these associations include being held, rocked or nursed, and eating or drinking prior to bed. Other associations include falling asleep in the car and sleeping in a parent's bed. For the child with sleep-onset association disorder, sleep onset is impaired when these circumstances are absent. In addition, if the child wakes up at night, they are not able to fall back to sleep if "that something" or circumstance is absent. Between 25% and 50% of six to twelve month olds have this condition.

The most important sleep hygiene measure is to maintain a regular sleep and wake pattern seven days a week for your infant. It is also important to spend an appropriate amount of time in bed, not too little, or too much. This may vary by individual infant. Maintaining a schedule might be inconvenient and time consuming but with just one infant is a possibility. Therefore establishing good sleep routines should be a priority. Sleep hygiene also concerns the physical space the infant sleeps in. The comfort of the cot, the light, temperature and the cleanliness of the bedroom are all important factors.

Sleep apnea is a potentially serious disorder where breathing is interrupted repeatedly during sleep. There are three kinds of apnea: obstructive sleep apnea, which is caused by a blockage; central sleep apnea, in which there is no blockage but the brain fails to signal the muscles to breathe, and mixed apnea that is a combination of the two (Lin A *et al*, 2012).

Children with Down Syndrome and other congenital conditions that affect the upper airway have a higher incidence of sleep apnea. Over half of children with Down Syndrome will develop obstructive sleep apnea. Any baby can have sleep apnea, but it is much more common in babies who were born prematurely. In babies born to mothers up to 37 weeks pregnant, it is called apnea of prematurity. In babies born to mothers past 37 weeks pregnant, it is called apnea of infancy. The more premature a baby, the more likely the baby is to suffer from apnea.

Infants not having been taught to self soothe is called limit-setting sleep disorder and is a parent driven difficulty. The associations between infant sleep patterns and parental behaviours, cognitions and expectations suggest that parents play a pivotal role in the

development of the sleep patterns of infants (Anuntaseree W *et al*, 2008). If babies do not sleep well, by waking often, it interferes with the sleep of their mothers.

1.4. Interaction between maternal and baby sleep in the first year of life

Mothers' and babies' sleep in the first year of the infant's life is a complex and stressful balancing act. Sleep Onset Association Disorder and Limit Setting Sleep Disorder imply the involvement of the care-giver with the sleep problem (Touchette E *et al*, 2009). The links between parenting and baby sleep are bidirectional. Just as parenting plays a major role in infant sleep, infant sleep and sleep problems can influence parental mood and physiological well-being. Comforting a crying baby while coping with personal tiredness can challenge mothers after birth. Infant crying and increasing exhaustion can negatively affect family health. Maternal exhaustion has been identified as a predictor of postpartum depression (Bozoky I *et al*, 2002), and persistently crying infants are at a higher risk for shaken baby syndrome or other forms of child abuse (Reijneveld S *et al*, 2004).

The difference between tiredness and fatigue, is that the former is a physiological state occurring after extended wakefulness and/or exertion that is relieved by a period of sleep, and the latter is a pathological state which persists through the circadian rhythm and cannot be relieved through a single period of sleep (Milligan R *et al*, 1996). Fatigue decreases the ability to perform daily activities and hampers the well-being of the affected person. Mothers of young infants are sleep deprived along with their infants if they are not sleeping.

Parents' beliefs, expectations, emotions and behaviours related to infant sleep are influenced by a number of external factors (Sadeh A *et al*, 2010). A mothers' social cultural, environment, developmental history, memories, personality and psychopathology affect the way she raises her infant and thus possibly the infant's developmental characteristics and sleep patterns. Across the life cycle of women, the quality and quantity of sleep can be impacted by hormonal changes, vasomotor symptoms (Moline M *et al*, 2004) and child-care responsibilities.

2. Reduced Sleep

Internal and external factors can influence the balance of the sleep-wake cycle. Internal factors include advancing age (Atalay H. 2011), stress, anxiety, depression, menstrual phase, chronic pain or other discomfort (Schwartz J *et al*, 2008). External factors include socioeconomic status (Friedman E *et al*, 2007), food, beverages, caffeine, alcohol, nicotine, antihistamines, and prescription medications such as contraceptives, beta blockers, alpha blockers, and antidepressants are all known to interfere with sleep (Reddy A *et al*, 2010). Other external factors include the sleep environment such as bed partners, light, noise, temperature and bed comfort can also greatly affect the quantity and quality of sleep. In general, all of these factors tend to reduce the number of hours of sleep and the depth of sleep (Schwartz J *et al*, 2008), impacting the quantity and quality of sleep respectively.

There is abundant scientific evidence supporting the conclusion that sleep is an essential physiological need that must be satisfied to ensure survival. Experimental work on sleep restriction has determined that 7-8 hours of quality sleep is required for optimal performance (Banks S *et al*, 2007). Sleep disturbances affecting quality and quantity of sleep

are thought to impact hormones that can lead to psychological and physiological changes. Psychological changes can include emotional experiences, depression, pain and memory (Chuah L *et al*, 2010). The metabolic, endocrine and immune changes have been suggested to include body mass gain (Hasler G *et al*, 2004), development of obesity (Gangwisch J *et al*, 2005), Type 2 Diabetes (Yaggi H *et al*, 2006), heart disease (Ayas N *et al*, 2003) and even mortality in adult women (Patel S *et al*, 2004).

In a study mothers were surveyed 3 years post partum where short sleep had been defined as ≤ 5 hours in 24 hours and long sleep > 5 hours sleep per day at 6 months and 1 year postpartum. They found at 3 years postpartum significantly higher adiposity in short sleepers. This was measured by a significantly increased body mass retention, larger subscapular and tricep skinfold thickness and a higher waist circumference (Taveras E *et al*, 2011). There are also indications that lactation and the duration of breastfeeding can directly impact the mothers ghrelin and peptide YY levels affecting her adiposity at 3 years postpartum (Stuebe A *et al*, 2011). Breastfeeding was purposefully excluded from this study.

The optimum amount of sleep required every evening for adults is 7-8 hours however the amount that the population sleeps is changing over time (Kripke D *et al*, 2002). Between 1959 (Kripke D *et al*, 1979) and 1992 (Bliwise D *et al*, 1992) the average amount of sleep reported by middle age people decreased by about one hour per night (from 8-9 hours per night to 7-8 hours per night). A study examining the sleep duration from sleep diaries of full time workers from 1975 to 2006 (Knutson K *et al*, 2010), found a significant increase in the number of individuals who were sleeping less than 6 hours per night. A recent study from the National Health Interview Survey (Cincinnati, Ohio USA) which examined the sleep duration of individuals across several occupations ranging from manufacturing to public

administration found that the percent of workers who reported sleep duration of 6 hours or less per night increased from 24% to 30% (Luckhaupt S *et al*, 2010) in the last 20 years. These findings may demonstrate the development of widespread partial sleep deprivation or sleep restriction related to external environmental or social factors such as parenthood, needing to work more than one job or longer work shifts rather than a biologic change in need for sleep. Interestingly it has been shown that subjects frequently underestimate the impact of their sleep restriction and overestimate their performance when their sleep is restricted (Van Dongen H *et al*, 2003). There are a number of ways in which the total number of hours of sleep can be reduced.

There are three modalities of sleep loss. Total sleep deprivation (such as a shift work), chronic sleep restriction or partial sleep deprivation (due to work, social and domestic responsibilities, medical conditions or lifestyle) and sleep disruption that is common in sleep disorders such as sleep apnea or restless legs syndrome (Schwartz J *et al*, 2008). Chronic sleep restriction and sleep disruption (also known as sleep fragmentation) have the same impact on daytime cognitive functioning as a period of total sleep deprivation (Van Dongen H *et al*, 2003). Sleep quantity is affected by the number of awakenings at night, sleep latency, and sleep duration. Some survey studies have investigated sleep habits in populations using self-reported data on sleep habits and health. Studies focused on sleep quantity show that 7-8 hours of sleep at night is positively associated with good health and longevity (Frederick T *et al*, 1988). The effect of sleep quantity is not independent as there is also a positive association with good sleep quality and good health (Lugaresi E *et al*, 1983).

Reduced sleep quality and less time spent sleeping causes fatigue and dysfunction in the daytime. This is linked to reduced quality of life, depression, absenteeism from work, accidents, and increased health problems such as obesity, Type 2 Diabetes, cardiovascular problems and even death (Léger D *et al*, 2010). Short sleepers, with poor sleep quality have an increased risk of cardiovascular disease and coronary heart disease (Hoevenaer-Blom M *et al*, 2011). The symptoms of poor sleep quality and reduced time spent asleep are the same. There are intrinsic differences between genders in our 24 hour circadian rhythms (Duffy J *et al*, 2011).

2.1.Reduced Sleep in Women

Research on gender differences and gonadal steroid regulation of sleep and biological rhythms is still very new. It has been shown that ovarian steroids affect circadian rhythms in female rats and has been replicated in female humans (Mong J *et al*, 2011).

Women are much more likely to report sleep problems such as not getting enough sleep or being sleepy during the day (Cain S *et al*, 2010). The most common sleep problem in women is insomnia (Adan A *et al*, 2002). Other common sleep disorders are sleep disordered breathing, restless legs syndrome or periodic limb movement disorder (Moline M *et al*, 2004) and hormonal fluctuations that could be caused by oral contraception or other medications such as antidepressants.

Insomnia is more prevalent in women. Insomnia is defined as repeated difficulty with sleep initiation, maintenance, consolidation, or quality that occurs despite adequate time and opportunity for sleep resulting in daytime impairment. Common diagnosis includes taking longer than thirty minutes to fall asleep, staying asleep for less than six hours, waking more than three times a night, or experiencing sleep that is chronically non-restorative or poor in quality. Insomnia is usually a short-term condition but it can become chronic.

Acute insomnia lasts up to one month. It is often referred to as adjustment insomnia because it most often occurs in the context of an acute situational stress, such as a new job or an upcoming deadline or examination. This insomnia typically resolves when the stressor is no longer present or the individual adapts to the stressor.

Transient insomnia often recurs when new or similar stresses arise in the patient's life. Transient insomnia lasts for less than one week and can be caused by another disorder, changes in the sleep environment, stress, or severe depression (Pavlova M *et al*, 2011).

Chronic insomnia lasting more than one month can be associated with a wide variety of medical and psychiatric conditions. Chronic insomnia has numerous health consequences such as slower responses to challenging reaction-time tasks. Subjects with chronic insomnia have reduced quality of life, comparable to that experienced by patients with such conditions as diabetes, arthritis, and heart disease. Quality of life improves with treatment but still does not reach the level seen in the general population. Despite not getting adequate sleep, patients with insomnia often have difficulty falling asleep even for daytime naps (Pavlova M *et al*, 2011).

Insomnia can also be a risk factor for depression and a symptom of a number of medical, psychiatric and sleep disorders. Insomnia appears to be predictive of a number of disorders, including depression, anxiety, alcohol dependence, drug dependence and suicide (Pavlova M *et al*, 2011).

Primary insomnia is a diagnosis of exclusion. The differential diagnosis of primary insomnia requires ruling out several other conditions, including medical, psychiatric, or circadian-rhythm disorders (eg, delayed sleep-phase syndrome) or other sleep-related disorders, such as periodic limb movement disorder or restless-legs syndrome. The treatment of primary insomnia begins with education about the sleep problem and appropriate sleep hygiene measures. Cognitive behavioural therapy is now considered the most appropriate treatment for patients with primary insomnia. Cognitive-behavioral interventions could help target sleep related problems and unhelpful safety behaviors such as long catch-up naps. Cognitive-behavioral interventions can promote accepting and non-judgmental attitudes and might reduce the distress associated with the unnecessary struggles to maintain “normal” sleeping routines whilst dealing with motherhood (Harvey A *et al*, 2007).

Sleep disordered breathing can range from snoring to sleep apnea and is less common in women than in men. Sleep disordered breathing is more prevalent during the menopausal years due to the loss of progesterone which stimulates breathing, the redistribution of fat,

as measured by waist/hip circumference ratio changes and increased rates of obesity (Moline M *et al*, 2004).

Many studies have reported that women are affected by restless legs syndrome twice as often as males for mild, moderate and severe restless legs syndrome. Restless legs syndrome will generally become worse or might appear for the first time during pregnancy (Dzaja A *et al*, 2009). Parity increases the risk of restless legs syndrome later in life suggesting that pregnancy is a specific behavioral risk factor for developing restless legs syndrome. Iron metabolism and high estrogen levels might contribute to restless legs syndrome during pregnancy (Manconi M *et al*, 2012).

Hormonal fluctuations can interfere with sleep during early pregnancy, the growing fetus can make it difficult to sleep comfortably later on during in pregnancy, and newborn care typically involves numerous nighttime awakening periods after the baby is born. All of these changes in sleep patterns and the associated fatigue can affect a woman's physical and mental well-being, her relationships, her employment, and her ability to adjust to the new role of parent. Oral contraceptives and antidepressant medication also affect sleep and are discussed later in the review.

3.Measuring Reduced Sleep (Quality and Quantity)

Perception of sleep quality (subjective) using sleep diaries or questionnaires is not as accurate as the objective measurements of sleep using activity monitors or electroencephalogram (EEG) recordings. The objective measurement of sleep duration is a more accurate measure than subjective reporting

Sleep efficiency is the ratio of time spent asleep (total sleep time) to the amount of time spent in bed. Sleep quality is measured more subjectively using depth of sleep, how well rested one feels upon awakening and general satisfaction with sleep (Buysse D *et al*, 1989). The quality of sleep can be assessed in many ways. Some of these include the Epworth Sleepiness Scale, the Pittsburgh Sleep Quality Index and Patient-Reported Outcomes Measurement Information System (PROMIS™) to name a few. The Epworth Sleepiness Scale consists of 8 self-rated items, each scored from 0-3, that measure a subject's habitual "likelihood of dozing or falling asleep" in common situations of daily living. No specific time frame is specified. The Epworth Sleepiness Scale score represents the sum of individual items, and ranges from 0-24. Values >10 are considered to indicate significant sleepiness. The Epworth Sleepiness Scale is sensitive to change in clinical status, as evidenced by improvements following treatment of sleep apnea with continuous positive airway pressure (Nguyen A *et al*, 2006).

There are many questionnaires which can be used to check quality of sleep. Probably the most well-known is the Pittsburgh Sleep Quality Index which is a subjective measure of perceived quality of sleep over the past month (Appendix B). The subject fills in the answers independently and the questions differentiate poor from good sleep by measuring seven areas: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication and daytime dysfunction. The analysis of the answers gives a score from 0 to 21. A score of greater than 5 indicates a greater severity of sleep disturbance and a lower quality of sleep (Buysse D *et al*, 2008). The PSQI has been correlated with subject reported and clinician-identified sleep disorders. Research suggests that the PSQI is an internally consistent, valid measure of self-reported sleep condition problems (Gottlieb D *et al*, 2006).

The Patient-Reported Outcomes Measurement Information System (PROMIS™) Sleep Disturbance and Sleep-Related Impairment item banks are results from post-hoc computerized adaptive testing simulations, item discrimination parameters, item means, and clinical judgments. They were used to select the best-performing eight items for Sleep Disturbance and Sleep-Related Impairment. The final 8-item short forms provided less test information than the corresponding full banks, but correlated strongly with the longer forms. The short forms had greater measurement precision than the Pittsburgh Sleep Quality Index and the Epworth Sleepiness Scale, as indicated by larger test information values across the continuum of severity, despite having fewer total items that is a major advantage for both research and clinical settings (Yu L *et al*, 2011). PROMIS™ is a

program that utilizes both subjective and objective measurements of sleep. This study used the PSQI for its simplicity and validity.

3. Classifications of Reduced Sleep

The importance of sleep becomes apparent when sleep disruption occurs. Acute sleep deprivation over a short period of time has been shown to increase anxiety and depression and general distress (Babson K *et al*, 2010). Total sleep deprivation can cause a greater adverse effect than partial sleep deprivation. Partial sleep deprivation over more than 6 months has not been studied extensively. Sleep fragmentation prevents the normal progression of sleep stages causing a decrease in normal physiological sleep relative to the time in bed (Van Dongen H *et al*, 2003). Sleep restriction (or sleep debt) is reduced sleep duration and is the most common form of sleep deprivation (Schwartz J *et al*, 2008). Selective sleep stage deprivation occurs if sleep fragmentation is restricted to a specific sleep stage for example REM sleep in untreated obstructive sleep apnea (airflow cessation), or medications interfering with sleep structure (Van Dongen H *et al*, 2003). A vicious cycle of depression, anxiety and disturbed sleep can develop (Harvey A. 2002).

The consequences of sleep reduction can affect learning, memory processing, cellular repair, brain development and functioning including stress, anxiety, depression, hormones, immunity and metabolism (AlDabal L *et al*, 2011).

4. Consequences of Reduced Sleep

4.1. Cognitive

Sleep disruption changes almost all behavioral and neurocognition including attention, learning and memory, emotional reactivity, executive function and decision making (Killgore W. 2010). Decreased attention following various durations of sleep deprivation have been identified (Balkin T *et al*, 2008. Learning and memory is decreased when any type of sleep restriction is imposed on mammals such as rats, apes and humans. Recent neurobiological findings have shown the existing brain mechanisms in rats that may cause the cognitive deficits as a result of sleep disturbances (Poe G *et al*, 2010).

Sleep loss causes decreased cognitive performance in simulated driving tests and induces sleepiness, fatigue and mood changes (Babson K *et al*, 2010). The effects of short-term sleep deprivation (days or weeks of disturbed sleep) can be remedied by simply sleeping properly for more than 2 nights, whereas long-term sleep deprivation that lasts months or even years can have more serious consequences to the sufferers' health. Perceived sleep deprivation can be more of a problem than actually being sleep deprived and can cause many unpleasant symptoms such as stress, anxiety and insomnia (Bei B *et al*, 2010). Research into human pathways is ongoing however emotional reactivity and stress are also impacted by sleep disruption (Lim J *et al*, 2010).

4.2. Mood

Depression and sleep deprivation are thought to be a bidirectional relationship. Sleep changes and depression can occur simultaneously (Buysse D *et al*, 2008) . Neuroimaging, neuropathological, and lesion analysis data suggest that most areas similar to sleep pathways are involved in the regulation of the evaluative, expressive, and experiential aspects of emotion (Drevets W *et al*, 1999). Sleep deprivation could interfere with mood, resulting in depression.

Melatonin is one hormone that affects stress and sleep responses. The rhythm of pineal melatonin production is controlled by the suprachiasmatic nucleus (Reiter R *et al*, 1992) and properly phased light exposure (Deacon S *et al*, 1994). Plasma melatonin is used as a marker of the phase of the human circadian timing system, and the use of extended wakefulness that examine the circadian clock without sleep (Duffy J *et al*, 2002).

Tryptophan is an amino acid that is vital to the production of serotonin and melatonin (Leu-Semenescu S *et al*, 2010). It is found in foods such as dairy, bananas and poultry. Serotonin affects mood and melatonin affects sleep. Melatonin, through its actions in the central nervous system, coordinates the circadian mechanism of sleep (Waldhauser F *et al*, 1990). The concentration of the amino acid tryptophan found in the blood and in the cerebrospinal fluid is lower in psychologically depressed patients than in controls (Yatham L *et al*, 2001). This is one cause of a decrease in plasma serotonin, a neurotransmitter that has frequently been implicated in depressive syndromes (Maes M *et al*, 1997) when it is decreased in the brain.

5-hydroxytryptamine (5-HT) is, a monoamine compound classically referred to as 'serotonin' due to early observations identifying it as a serum agent (*sero-*) affecting vascular tone (*-tonin*) (Paredes S *et al*, 2009). 5-HT is synthesized in two steps from the essential amino acid tryptophan. Patients with depression show reduced monoamine serotonin (5-HT) levels (Placidi G *et al*, 2001), abnormal serotonin receptor function (Drevets W *et al*, 1999) and abnormal serotonin transporter function (Caspi A *et al*, 2003). One way to increase serotonin in healthy individuals is by using an antidepressant (Robinson O *et al*, 2009).

Numerous drugs acting as serotonin agonists or serotonin reuptake inhibitors (SSRI's) decrease food intake, induce weight loss, and advance the behavioral satiety sequence in rodents (Tallett A *et al*, 2009). SSRI's improved sleep quality in subjects with major depressive disorder (MDD). This has been confirmed in female human use through clinical trials (Babson K *et al*, 2010, Germaine A *et al*, 2008, Hanlon C *et al*, 2009). SSRI's commonly used in South Africa such as Cipralex, Zoloft, Diatroxin and generics of those, are non-sedating and are taken in the morning (Murck H. *et al*, 2004, Weissman A. *et al*, 2006). This has been shown to increase attention, learning and memory, emotional reactivity, executive function and decision making and decrease emotional reactivity (Killgore W. 2010)

Apart from antidepressants, it is important that mothers have the support that they require emotionally (Murck H *et al*, 2004). A husband, parent or a staff member, night or day assistance can help a mother to catch up on rest that has been lost due to disturbed sleep (Chuah L *et al*, 2010). This will aid in increased attention, learning and memory, emotional

reactivity, executive function and decision making, helping mothers to cope with daily demands and lifting mood.

There is a circadian gene network in human tissues that express autonomous clocks. Disruption of these clock genes results in metabolic dysregulation. Interactions between metabolism and circadian rhythms at neural, molecular, and cellular levels have been shown. A major challenge remains in understanding the interplay between the brain and peripheral clocks and in determining how these interactions promote energy homeostasis across the sleep-wake cycle (Huang W *et al*, 2011).

4.3. Metabolic

Reduced sleep duration has been associated with larger body mass index (BMI) (Taheri S *et al*, 2004, Gangwisch J *et al*, 2005). Short term sleep restriction results in a number of abnormal physiologic changes including reduced leptin levels (Spiegel K *et al*, 2004), reduced glucose tolerance (Spiegel K *et al*, 1999), increased blood pressure (Tochikubo O *et al*, 1996), elevation in evening cortisol (Spiegel K *et al*, 1999), activation of the sympathetic nervous system (Kato M *et al*, 2000) and increased inflammatory markers (Meier-Ewert H *et al*, 2004). It has been suggested that sleep restriction produces changes in the secretory profiles of appetite-regulating hormones that change the signaling of hunger and appetite and promote increased weight gain and obesity (Spiegel K *et al*, 2003).

4.3.1 Hormonal control

Appetite is the desire to eat food, felt as hunger. Appetite regulates adequate energy intake to maintain metabolic needs and is controlled by hormones that signal between the digestive tract, adipose tissue and the brain. The balance of calories ingested is based on body mass, body fat percentage, gender and level of hormones, including leptin, ghrelin, insulin, glucagon and other digestive hormones (galanin and orexin), that send signals to the brain, manage appetite and food intake.

Maintaining a healthy body mass depends on balancing the number of calories you consume with the number of calories used. A calorie is defined as a unit of energy supplied by food. A calorie is a calorie, regardless of its source, for example carbohydrates, fats, sugars, or proteins. Body fat percentage is the amount of fat within the body excluding organs, muscles, bones, tendons, and water (Østbye T *et al*, 2009). Men and women carry different amounts of body fat percentage. Women have a higher percentage of body fat than men and store more fat in the gluteal-femoral region, where men store more fat in the abdominal area (Østbye T *et al*, 2009).

4.3.1.1. Leptin and Ghrelin

Leptin is a protein and a cytokine produced in and secreted by adipose tissue (Campfield L *et al*, 1995). The action of leptin on energy homeostasis is controlled by neuropeptide Y (NPY),

a peptide produced in the hypothalamus. Leptin reduces food intake and increases energy expenditure (Pelleymounter M *et al*, 1995). Levels of NPY reflect the amount of adipose tissue in the body based on afferent signals. Acting as a central appetite suppressant, NPY affects energy intake and expenditure, sympathetic and parasympathetic nervous systems, and thyroid hormones (Rosenbaum M *et al*, 1997). NPY directly controls the effects of leptin in the body. In rats, administration of leptin normalises insulin's actions on glucose, decreases hyperphagia, and prevents infertility (McGregor G *et al*, 1996). Leptin has appetite-suppressant effects in obese mice but its role in human obesity is not clear. Leptin-deficient mice display hyperphagia, insulin resistance, hyperinsulinemia, and infertility that are reversible by injecting leptin (Pelleymounter M *et al*, 1995). Leptin plasma levels are significantly suppressed and feeding increased after chronic sleep deprivation for 16 days (Everson C *et al*, 2004). Plasma leptin significantly decreased after 1-4 days of sleep deprivation. Other peptides involved in regulating the relationship between sleep deprivation and feeding include ghrelin.

Ghrelin is secreted in a pulsatile manner from cells in the lining of the fundus of the stomach and is regulated by the hypothalamus. It is increased after fasting. Ghrelin stimulates growth hormone secretion and increases food intake in rodents and humans by influencing mealtime-related appetite and hunger (levels rise before and fall after every meal) and is involved in long-term regulation of body weight (Kojima M *et al*, 1999).

Ghrelin is believed to play a key role in the compensatory changes in appetite and energy expenditure associated with weight loss. Ghrelin levels are high in subjects that lose weight by restricting calories, but suppressed in patients with weight loss from gastric bypass

surgery (Cummings D *et al*, 2002). Administration of ghrelin increases food intake and body weight in rats (Cummings D *et al*, 2001). Ghrelin and leptin appear to have inverse amplitude changes during fasting in rodents (Bagnasco M *et al*, 2002). Food deprivation increased the amount of leptin secretion causing stimulation of NPY by ghrelin to induce feeding (Bagnasco M *et al*, 2002).

Sleep deprivation increases plasma levels of ghrelin (increasing food intake) and plasma leptin levels decrease (Everson C *et al*, 2004). Bodosi *et al* (2004) found plasma ghrelin levels and food intake were increased in sleep deprived rats when compared to non-sleep-deprived controls. Other peptides regulating the relationship between sleep and food include galanin and orexin (hypocretin).

4.3.1.2. Galanin and Orexin

Galanin is a bioactive neuropeptide that is released during sleep deprivation. It is widely distributed throughout the nervous system and has diverse neuromodulatory effects (Murck H *et al*, 2004). Galanin is secreted by the pituitary gland known to stimulate food intake (Kyrkouli S *et al*, 1986).

Galanin mRNA is increased in the brains of sleep-deprived rats (Lu X *et al*, 2005). It has been shown that increased REM sleep deprivation causes an increase in plasma galanin in rats (Fujihara H *et al*, 2003).

Orexin neurons are specialised cells for sensing blood glucose in the hypothalamus (Burdakov D *et al*, 2006). Orexin is made in the hypothalamus and it stimulates food intake. In rats, orexin levels are increased by sleep deprivation and decreased with recovery sleep (Pedrazzoli M *et al*, 2004). These studies all suggest there is a link between sleep deprivation and increased food intake. There are no studies in humans and very few studies in rats (Fujihara H *et al*, 2003) on the effects of sleep deprivation on galanin and orexin. Orexin neurons have been shown to interact with glucose metabolism by inhibiting intestinal glucose absorption (Ducroc R *et al*, 2007).

4.3.1.3. Insulin and Glucagon

Glucose is an important signal molecule. Glucose levels in the bloodstream are sensed and controlled by hypothalamic circuits of neurons and neurochemicals that regulate appetite, energy expenditure and metabolism (Burdakov D *et al*, 2005). Metabolic fuel homeostasis is controlled by the balance between the anabolic hormone insulin and the catabolic hormone glucagon, both produced by the pancreatic islets of Langerhans.

Insulin secretion from islet β -cells is regulated by a number of factors, but the main stimulatory signal is an increase in blood glucose that occurs when ingesting carbohydrate-containing meals (Newgard C *et al*, 2001). Insulin regulates metabolic function by acting on liver, muscle, and fat. Insulin affects glucose metabolism by facilitating and increasing its transport into the cell; glycogen synthesis in liver, muscle, and adipose tissues; and glycolysis in fat and muscle tissues. It also promotes storage of triglycerides in fat cells, decreases lipolysis after meals, and increases protein metabolism. Plasma insulin levels are

generally in proportion to adipocyte concentration in the body; in adipocytes, insulin can regulate the expression of leptin to help reduce food intake (Rosenbaum M *et al*, 1997).

Impairment of insulin-secretory capacity and the loss of normal glucose-stimulated insulin secretion cause the transition from obese and insulin-resistant states to Type 2 Diabetes (Pfeifer M *et al*, 1981). Despite the central role of insulin secretion in the regulation of metabolic fuel homeostasis and the impact of the loss of normal β -cell function in the development of Type 2 Diabetes, the biochemical mechanisms involved in glucose-stimulated insulin secretion are not completely understood. Increased plasma glucose can result in weight gain (Vettor R *et al*, 2005). Weight problems can be assessed measuring various dimensions of the body.

4.3.2. Factors affecting anthropometry

Hormones, gender, age, ethnicity and sleep deprivation may influence body mass and therefore health through effects on appetite, physical activity and thermoregulation. Sleep duration at night, having an infant hospitalized in the first year and eating attitudes of mothers have also been considered (Siega-Riz A *et al*, 2010).

A survey by the American Cancer Society of 1.1 million subjects showed a U-shaped association between sleep duration (Ikehara S *et al*, 2009) and BMI in women with a minimum sleep duration of 7 hours (Figure 1.3). Sleep durations on the deprivation side of 3.5 to 4.5 hours and an excess of more than 8.5 hours per night were associated with a higher BMI (Kripke D *et al*, 2002). Figure 1.3 shows hours of sleep and the BMI of women.

Only the dip in the U-shape (6.5 to 7.5hours sleep) is considered optimal for the lowest BMI (Kripke D *et al*, 2002). In this study the mean age for women was 57 ± 11 yrs therefore the effect of age on sleep and BMI must be taken into consideration (Hasler G *et al*, 2004) and the same relationship may not hold for younger women. It has been shown that with increasing age, slow wave sleep during the night decreases, the ability to stay asleep at night lessens and the desire to sleep during the day decreases (Dijk D *et al*, 2010).

There are many more studies that confirm the U-shaped theory of sleep duration and mortality in men and women. There are many uncertainties surrounding the amount of hours within each section of the 'U' (Amagai Y *et al*, 2004, Gottlieb D *et al*, 2006, Heslop P *et al*, 2002). Chaput J *et al* (2009) researched 276 subjects over 6 years and showed that those with short (6 hours) and long (9 hours) sleeping times have a higher risk of developing Type 2 Diabetes. Women in Gothenburg were followed for 32 years to study the effect of sleep problems and diabetes incidence (Bjorkeland C *et al*, 2005).

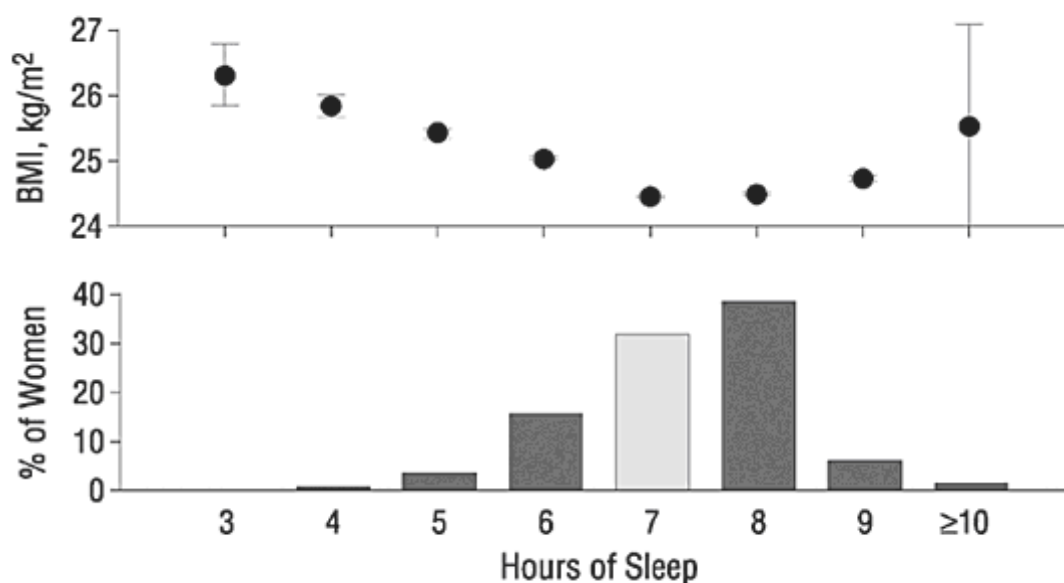


Figure 1.3. Mean body mass index (BMI) and reported hours of sleep for 636 095 women.

The 95% confidence intervals of the BMI are shown.

Sleep duration was found to be inversely correlated to BMI ($r=-0.06$) and WHR ($r=-0.08$). Centralised obesity was shown to be associated with sleep problems, and obesity is known to increase the risk of diabetes. Women sleeping 6 or less hours had a 0.63kg/m^2 greater mean BMI than those with longer sleep durations (Cournot M *et al*, 2004). Kohatsu N *et al* (2006) showed that of 990 employed adults in Iowa, BMI was 0.42 kg/m^2 greater for each hour reduction in sleep duration.

Some studies suggest that chronic lack of sleep affects hormonal release, glucose regulation and cardiovascular functioning. Hormones that are affected by chronic lack of sleep include insulin and growth hormone (Chaput J *et al*, 2007) that increase insulin resistance and result in decreased glucose tolerance. Physiological data suggests an increased sympathetic tone, increased cortisol concentrations and the appetite regulating hormones. Leptin levels

reduced and ghrelin increased resulting in an increased appetite, in response to sleep deprivation (Omisade A *et al*, 2010).

Experimental sleep restriction has been shown to impact glucose homeostasis by changing insulin sensitivity and increasing the risk of diabetes (Knutson K *et al*, 2008). Long term partial sleep restriction to <6.5 hours per night for at least 6 months (healthy volunteers of both sexes) have a glucose response to i.v glucose similar to that of subjects with habitual sleep times 7.5 to 8.5 hours per night but at the cost of a higher insulin secretion (Mander B *et al*, 2001). This implies that over an even longer period of time, when sleep deprivation becomes chronic, insulin resistance may develop.

The mechanisms regulating the association of short sleep time with Type 2 Diabetes and obesity are speculative (Cizza G *et al*, 2011). Van Cauter E *et al* (2008) suggest that there are three pathways that are responsible for weight gain and diabetes due to restricted sleep. They are the changes in the processing of glucose, the increased appetite and lethargy resulting in decreased energy expenditure.

Shorter sleep and sleep fragmentation have been shown to cause an increased BMI (Lauderdale DS *et al*, 2009) after being adjusted for all other variables. A review and analysis of published data from 2003 to 2009 showed that there is a consistent pattern of increased risk of developing Type 2 Diabetes, with short and long sleep durations and with qualitative disturbances of sleep (Cappuccio F *et al*, 2010). This study showed that subjects with <5-6

hours per night had a 28% risk of developing diabetes whereas those with difficulties maintaining their sleep had a risk of 84% (Cappuccio F *et al*, 2010).

There is mounting evidence that chronic partial sleep loss is a stress factor that would stimulate appetite, promote fat gain, impair glycaemic regulation, change body distribution of fat and may increase the risk of obesity and eventually Type 2 Diabetes (Yaggi H *et al*, 2006). In low socioeconomic societies there are studies showing increased weight gain during pregnancy and difficulty in losing the body mass post partum (Rothberg B *et al*, 2011).

4.3.3. Measuring body dimensions

It is important to know how much of the body mass is adipose tissue (fat mass) and how much is lean muscle mass as this can become a problem with metabolism and be related to adverse future events such as obesity (Peltz G *et al*, 2010).

Anthropometric measurements are defined as a set of noninvasive, quantitative techniques for determining an individual's body fat composition by measuring, recording, and analysing specific dimensions of the body, such as height and weight; skin-fold thickness; and bodily circumference at the waist, hip, and chest (National Institutes of Health, 1998). These measurements are used to assess nutritional status.

Body mass index (BMI) is the most commonly used measure of weight in relation to height. Body mass index is defined as the individual's body mass (in kg) divided by the square of his

or her height (in m) producing a unit of measure of kg/m^2 (Gallagher D *et al*, 1996). Adult BMI are classified as follows: Underweight is a score less than 18.5 and a normal weight is a score of 18.5 to 25.0. Overweight or 'pre-obese' has a score of 25–29.9, 30–34.9 as 'obese class I'; 35–39.9 as 'obese class II'; and over 40 as 'obese class III'. This classification was based on the risk of comorbidities, from the four categories 'increased', to 'moderate', 'severe' and 'very severe' (WHO, 1995).

Ethnic differences in body composition and the percentage of body fat associated with adverse health consequences mean that a single international definition of obesity is not appropriate (Wang Y. 2004). Differences in BMI is not a concern in this paper as all mothers were Caucasian. BMI is a reasonable measure of adiposity, although the relationship differs not only according to age, sex and ethnicity, but also degree of fatness. In a study done by Weerarathna T *et al* (2008), BMI had the strongest association with total and visceral fat mass among 106 women (30-54 yrs).

Waist circumference, hip circumference and waist to hip ratios (waist circumference divided by the hip circumference) have the advantage of being simple and low-cost measurements requiring only a tape measure. Waist and hip circumferences showed high correlations in women 30-54 yrs old with total and visceral fat mass (Weerarathna T *et al*, 2008) highlighting the importance of waist and hip circumference measurements as well as waist to hip ratios.

The waist circumference (wc) is the measurement in cms above the hip bones. Waist circumference is variable according to sex, age, ethnicity and country of origin. Asians have

values below 80cm (Ito H *et al*, 2003), Canadians at 80cm (Dobbelsteyn C *et al*, 2001), Mexicans at 85cm (Berber A *et al*, 2001), and Brazilians at 84cm for example. These values give an indication of the variability. It has been shown that waist circumference is an effective predictor of metabolic syndrome (Beydoun M *et al*, 2011).

Hip circumference is the measurement in cms of the area around the body below the hip bones. Normal hip circumference is about 102cms in Caucasian women. Hip circumference is independently and inversely associated with diabetes (Snijder M *et al*, 2004). After adjustment for age, body mass index and waist, a larger hip circumference was associated with a lower prevalence of undiagnosed diabetes. Waist to hip ratio, at a value of 0.80 is the cut-off point to estimate abdominal obesity in women (Dobbelsteyn C *et al*, 2001). But studies conducted in Brazil, found values were 0.83 for adult women 30 and 74 years of age, 0.84 for women from 30 to 49 years of age, and 0.88 for those older than 50 years (Pitanga F *et al*, 2005). The cut-off point found in Mexico was 0.85 (Berber A *et al*, 2001). Truncal and abdominal fat accumulation (android pattern) in women has increased due to changes related to eating habits and lifestyle that occurred in the past decades, increasing adverse health risks.

Triceps skinfold (TSF) is suggested as the best simple predictor of body density and percentage total body fat. Existing methods in low resource settings of predicting muscle mass are based on mid upper arm circumference (MUAC) measurements corrected for tricep skinfold fat. This is a simple and non-invasive method that is accurate in determining muscle wasting using a scale. It is measured using calibrated fat calipers. The upper arm length and the mid-upper arm circumference (MUAC) are measured from the mid-point

between the acromion (shoulder bone) and the olecranon process of the ulna (elbow) using a tape measure. These measurements are calculated and applied to a table as indicators of body fat (Shephard R. 1991). Factors influencing the bone, fat, and muscle composition of the upper arm include age, sex, nutritional status, fitness training level and race. All calculations are explained further in Chapter Two.

5. Postpartum

5.1. Post Partum Depression

Very few studies have examined depression prevalence and severity among women beyond 6 months to 1 year postpartum and the studies that do exist have small samples. Small R *et al* (1994) reported that 34% of a sample of 45 women who were found to be depressed at 8 to 9 months postpartum continued to experience depression at 2 years after delivery. Horowitz J *et al* (2004) discovered that 31% of a group of 62 women with elevated depression scores at 2 to 4 weeks postpartum continued to score in the depressed range at 2 years after delivery. Depression symptoms occur beyond the early postpartum weeks and there is a need to establish symptom prevalence rates later than 3 months after delivery with larger, more representative samples. Research regarding maternal demographic factors, such as income, have shown an increase in the risk for postpartum depression (Beeber S *et al*, 2003; Beeghly *et al*, 2003). Few studies have been conducted with definitive conclusions as to the relationship of age, education, parity, and race to the development of either mild or severe depression symptoms among women 3 to 6 months postpartum.

5.2. Antidepressants

There are four main classes of antidepressants on the market. The tricyclic antidepressants (TCA) block the reuptake of norepinephrine, dopamine and serotonin. TCA's can cause cardiac toxicity in overdose as well as a variety of adverse effects due to their multiple receptor impacts. Therefore tricyclics are being prescribed less (Wisner K *et al*, 2006). Selective serotonin-reuptake inhibitors (SSRI) primary mode of action is the selective inhibition of serotonergic reuptake. However different SSRIs have noradrenergic and dopaminergic reuptake inhibition, serotonin 2C, muscarinic and sigma 1 receptor antagonism. They also inhibit nitric oxide synthesis and various cytochrome P450 enzymes. SSRIs are widely prescribed antidepressants due to their safety in overdose (Weissman A *et al*, 2004). Serotonin and noradrenalin-reuptake inhibitors (SNRI) treat patients with the combined inhibition of the reuptake of serotonin and noradrenaline, and less potent inhibition of dopaminergic reuptake (Eberhard-Gran M *et al*, 2006). Noradrenergic and specific serotonergic antidepressants (NaSSA) work by blocking presynaptic alpha-2 adrenergic receptors that normally inhibit the release of the noradrenaline and serotonin (Weissman A *et al*, 2004). Norepinephrine and dopamine reuptake inhibitor (NDRI), are often used as a co-therapy with SSRIs or SNRIs. SSRI's have been shown to decrease sleep quality in some women, that may result in weight gain (Van Cauter E *et al*, 2008).

5.3. Antidepressants, Sleep and Anthropometry

Sleep deprivation is an important symptom of MDD and has the potential to worsen depressive symptoms. No current antidepressant treatment addresses the specific sleep disruptions of depression (Becker P. 2006). The relationship between obesity and depression is bidirectional and are associated with sleep disturbances. Higher BMI is associated with lighter and less deep sleep. Both sleep and body mass are factors that treatment can focus on to interrupt the complex, relationships with MDD and sleep (Dombrovski A *et al*, 2008).

Maternal depression has been well documented to adversely impact relationships, parenting practices, family functioning, and baby's development and general well-being (Mazure C *et al*, 2002). Some research suggests an association between sleep problems and depression. While the impact of less sleep on metabolism has been studied under experimental conditions, there is some inconclusive research on new mothers suffering with long-term sleep debt due to natural circumstances, such as a new baby waking up frequently during the night. There are no studies done in South Africa detailing the sleep habits of mothers and babies and any relationship between sleep and other variables. Due to its effects on health and well being sleep deprivation may increase a mother's risk of post natal depression, obesity and diabetes.

5.4. Oral Contraceptives

After a baby is born, and breastfeeding has ceased completely, the regular female cycle can begin again. This can occur as soon as 6-weeks postpartum. Assume that the start of the

menstrual period marks the start of the cycle. Follicle-stimulating hormone (FSH) is secreted by the pituitary gland. FSH is responsible for stimulating the development of an ovarian follicle and the egg it contains. The follicle secretes estrogen, so during development of the follicle, the estrogen levels in the blood rise. Estrogen has a number of effects; the glands lining the cervix secrete mucus and the uterine lining builds up. A day prior to ovulation the estrogen level in the blood peaks that triggers a change in the pituitary gland resulting in secretion of luteinizing hormone (LH). Ovulation occurs because the level of LH causes extrusion of the ovum from the follicle (Ecochard R *et al*, 2000). If the ovum is not fertilized by sperm over the next 24 hours, menstruation starts approximately two weeks later and the cycle repeats. If a woman is on an oral contraceptive her cycle becomes anovulatory.

Combined oral contraceptives have both a low estrogen and progestogen content. The contraceptive effects of estrogenic agents change ovulation, ovum transport, and implantation. The progestational agents act mainly by inhibiting ovulation and creating a hostile uterine environment. Biphasic and triphasic combined oral contraceptives are designed to deliver the hormones, throughout the menstrual cycle, in varying amounts that are similar to the natural physiologic quantities. The combined oral contraceptive, is the most effective method of birth control available with the exception of sterilisation. If the low-dose combined oral contraceptives are taken at approximately the same time each day, they have a theoretical failure rate of less than 0.5 per 100 women-years (Hannafor P. 2000).

Both estrogenic adverse effects (nausea, dizziness, irritability, weight gain, bloating) and progestogenic adverse effects (vaginal dryness, acne, hirsutism, weight gain, depression,

loss of libido) can occur in 50% of women, but these generally disappear after a few months of use (Grossman N. 2010). Low-dose, third generation combined oral contraceptives are associated with minimal risks in the absence of other risk factors and have many beneficial effects such as the prevention of ovarian and endometrial cancer, a decrease in pelvic inflammatory disease and ectopic pregnancies, and protection from anemia, primary dysmenorrhea, functional ovarian cysts, and benign breast disease as well as from the morbidity and mortality associated with pregnancy (Grossman N. 2010).

Oral contraceptives have also been shown to change normal sleep architecture in women (Baker F *et al*, 2001). Women taking oral contraceptives have more Stage 2 N-REM sleep in the active phase of their cycles and less slow wave sleep in the luteal phase of their cycle. These changes in sleep architecture are thought to be due to changes in progesterone. This is not yet proven due to the complex interactions of the hormones in the menstrual cycle and the different types of oral contraceptives that are prescribed. According to a review by Gallo MF *et al* (2004) oral contraceptives have shown no significant effect on body mass in many studies across race, age and body mass. Oral contraceptive users have a higher level of depression in some studies and positive mood changes in others (Almagor M *et al*, 1991). Depressed women under the age of 40 on combined oral contraception were significantly more likely to worsen their depression but if they were not depressed before consuming combined oral contraception there was no significant change in mood (Young E *et al*, 2007). The current literature has not yet confirmed a direct connection between oral contraceptive use and depression.

6. Conclusion

Sleep deprivation, whether by restricted hours of sleep or sleep fragmentation, has been shown to impair glucose tolerance and decrease insulin sensitivity (Knutson K *et al*, 2007). The compounded effect of increased plasma glucose and an increased appetite could result in an increased risk for diabetes and obesity. Sleep disruption may also impact on anxiety and depression. The current research suggests that disturbed sleep may cause abnormalities in emotional wellbeing and stress hormone release (Omisade A *et al*, 2010).

Sleep deprivation has become an increasingly public health concern for men and women, due to its detrimental effects on cognitive functioning over time, accidents and errors in the workplace, and alterations in metabolic and endocrine function of individuals (Banks S *et al*, 2007). Increasing evidence also suggests that sleep deprivation may increase mortality (Banks S *et al*, 2007, Hublin C *et al*, 2007). Pregnancy and the postpartum period are also times when women are at a heightened risk of depression (Mazure C *et al*, 2002). Some of the limitations of previous research in postpartum women and depression include small sample sizes (Patel S *et al*, 2004), only up to 3 months postpartum (Bloch M *et al*, 2006, Bowman M *et al*, 2009)) and lack of anthropometric variables included in the studies (Horowitz J *et al*, 2004, Jung C *et al*, 2011, Spiegel K *et al*, 2004).

This study assessed age, education, employment, familial history of diabetes and depression, anthropometric variables, depression and medication use in a small sample of South African mothers of infants between 5 and 13 months old. Gender influence on baby data such as: age at the date of interview, weight at birth, daytime naps and number of arousals during an 8hour period at night were observed. Differences between sleep

measurements were assessed by dividing the sample into various categories including the PSQI score, hours sleep at night, sleep efficiency and number of baby arousals at night. Hours of sleep at night and sleep efficiency are a part of the PSQI questionnaire and I was interested in finding out whether they could be used on their own. Sleep quality may also be affected by other confounding factors such as depression, antidepressant use and other medication such as oral contraceptives. Oral contraceptive use may also have an effect on anthropometrics.

The hypothesis for the study was that poor sleep (in all categories) would be associated with significantly increased body mass post-partum, increased occurrence of diabetes and the medication related to insulin control, and significantly increased postpartum depression.

7.Objectives

- To describe the sleep and anthropometry in a group of mothers with babies between 5-13 months old in Gauteng, South Africa
- To evaluate a number of different ways of measuring sleep
- To compare anthropometric variables between mothers with poor and good sleep
- To assess the factors other than baby's sleep influencing maternal sleep

CHAPTER TWO - Materials and Methods

2.1. Subjects

The population sampled was from the northern suburbs of Johannesburg and Pretoria in Gauteng. All the subjects were recruited from Moms and Babes workshops in Pretoria, Sandton and Fourways and from Well-Baby clinics in Morningside and Fourways.

Inclusion criteria were women currently living in Gauteng who had given birth to only one child (unipara). The mothers had to be between the ages of 25 and 40 years old living with only one child of between 5 and 13 months of age.

Ethics approval was obtained from the Human Research Ethics Committee of the University of the Witwatersrand (Ethics Clearance Number M070549).

2.2. Experimental Protocol

The study design conducted was a cross sectional questionnaire and anthropometric measurements of the mothers. Seventy five interview packs were distributed and thirty one were returned completed. The researcher attended the Well-Baby clinics and Moms and Babes workshops at the times that the babies (that fitted the inclusion criteria) were there. Mothers were assessed for inclusion and then given the information sheet to read (Appendix A). If they agreed to the study and signed the consent form (Appendix B) they were requested to complete the interview and the questionnaire. If they participated, the

researcher took the anthropometric measurements immediately and filled the anthropometric measurements into the interview (Appendix C). At the Well-Baby clinics, the mothers completed the questionnaire with the researcher and handed it back immediately. At the Moms and Babes workshops, the mothers were asked to complete their questionnaires at their convenience and return the completed forms at the next scheduled class.

Interview

The Interview (Appendix C) gathered information on demographics of the subjects, medical history during and after pregnancy, body mass before, during and after pregnancy and body mass and sleep patterns of the baby. It was completed by the mother with assistance from the researcher as required. Mothers were asked to recall information such as pre-pregnancy weight and the presence of glucose in their urine during pregnancy. The Pittsburgh Sleep Quality Index (Appendix E) and Becks Depression Inventory-II (Appendix D) were also completed by the mother and scored by the researcher. The Becks Depression Inventory II (BDI-II) is a standard questionnaire that asks 21 questions that are illustrative of the symptoms and attitudes of patients with depression (Appendix D). Each answer is scored on a scale value of 0 to 3. Once the score is totaled the results are interpreted as follows: 0–13: minimal depression; 14–19: mild depression; 20–28: moderate depression; and 29–63: severe depression (Beck A *et al*, 1996). Poor sleep and depressive symptoms seem to be linked but the exact, underlying mechanisms remain unknown.

The unipara mothers supplied information about their age, employment status, education, anthropometrics (historical mass recollections), family history of diabetes and depression (diseases and medications), current sleep patterns and quality (PSQI), medication use and depression (BDI-II). Data were recalled by the mothers about baby mass at birth, gender, number of night-time arousals, night assistance and general perceived sleep problems.

Anthropometric Measurements

Each mother underwent non-invasive anthropometric measurements on the day of survey (DOS) including height, weight, waist and hip circumference, mid-arm circumference and a tricep skin-fold-thickness (TSF) measurement. All measurements were done by the researcher. The mother's body mass was measured using a calibrated Scalerite Micro Slimline Bathroom Scale in kg (to one decimal place). The mother's height was measured using a portable, calibrated stadiometer. The mother's mass on the date of survey was subtracted from the pre-pregnancy mass to obtain the resultant mass change.

Body Mass Index (BMI) was calculated using the formula $\text{mass (kg)}/\text{height (m)}^2$. BMI was interpreted as <18 for undernutrition, 18.5-25 as normal, 25-29.9 overweight and 30+ as obese (Baker J *et al*, 2007).

A single calibrated measuring tape (Salooja Brothers Pvt Ltd, SA) was used to measure the mothers' waist in cm by taking the tape around the woman's back and closing it firmly around the umbilicus. The hip circumference was measured using the same measuring tape

around the woman's back and read at the level of the iliac crest (hip) bones. The measuring tape was held parallel to the floor for all measurements.

The waist hip ratio (WHR) was calculated by dividing the waist circumference (cm) by the hip circumference (cm).

The mid-arm circumference was measured by finding the halfway point between the acromion at the shoulder and the olecranon process of the ulna at the elbow. At the halfway point the tape measure was pulled around the mothers arm to get a reading of the mid-arm circumference in cm. The tricep skin fold thickness was measured at the same point by pulling the skin away from the muscle at the tricep and using a calibrated skin fold thickness caliper to measure in mm.

The Adjusted Mid-Arm (AMA) percentage was calculated using the mid-arm circumference and the skin fold thickness. The AMA estimates lean body mass and is used for calculating the mid upper arm muscle area in cm^2 using the equation:

$$\frac{[\text{midarm circumference(cm)} - (3,14 \times \text{tricep skinfold thickness (cm)})]^2}{12.56} - 6,5(\text{for females})$$

The formula corrects the upper arm area for fat and bone.

2.3. Data Analysis

Descriptive statistics (means, standard deviations and percentages) were used to describe the demographics of the sample of the population collected.

The mothers' data was then split into good and poor sleep categories using the following variables.

- A PSQI $\leq$ 5 was interpreted as good sleep whereas poor sleep was defined as PSQI $>$ 5 (Buysse D *et al*, 1989).
- Mother-reported baby arousals was defined as good sleep when woken up by the baby $\leq$ 1 time and poor sleep as woken by the baby $\geq$ 2 per night (Graham J *et al*, 2002).
- Mother-reported number of hours spent asleep where good sleep was defined as $\geq$ 7hours and poor sleep $<$ 7 hours (Banks S *et al*, 2010).
- Sleep efficiency where $\geq$ 80% was defined as good sleep and $<$ 80% poor sleep (Scheer F *et al*, 2009).

The continuous variables in this study were related to the purely categorical variables to decide on a way to categorise the continuous variables. 'Normal' sleepers were used as the control group, rather than mothers with children as there is no normal data for them.

Data were split along defined lines (as reported in the literature) for normal women of young age without young children (Luyster F *et al*, 2012) Data for number of hours of sleep and sleep efficiency were taken from the PSQI. Individual questions of all other data were divided into the appropriate categories. Data comparing poor and good sleepers in the four different ways indicated above were compared using unpaired students t-tests.

All data were tested for skewness and once found to be normally distributed, unpaired t-tests for independent variables at a confidence interval (CI) of 95% were used for analysis

The body mass of the mothers 6 months postpartum and 12 months post-partum (the effect of time) was compared between oral contraceptive users and non oral contraceptive users using a one-way repeated measures ANOVA. One mother with an intrauterine device (Merina™) that secretes low levels of estrogen and progestogen was excluded from analysis of the effect of oral contraceptives. It was assumed that none of the subjects were using oral contraceptives pre-pregnancy because it was before they fell pregnant or at 6 weeks post partum, where gynecologists normally prescribe contraceptives for new mothers.

Depression scores from the BDI-II were split into categories. Mothers with a score of 0-13 were classified as not depressed and scores 14 and above as having a high number of depressive symptoms and likely to be depressed (Jacksic N *et al*, 2013). All sleep variables were divided between the two depression categories and compared using unpaired students t-tests.

All the statistical analysis was done personally using Excel and Prism 5. P- values <0.05 were accepted as significant.

CHAPTER THREE - Results

3.1. Subject Demographics

3.1.1. Mothers Information

The mothers' mean(SD, range) age was 31.3 yrs(4.0, 25-40). They were all Caucasian and 81% of the mothers had tertiary education. At the time of the study 68% of the mothers were employed. The mothers mean(SD ,range) PSQI was 7.7(4.9, 0-20). Twelve mothers had a PSQI score under or equal to 5 (38%) and nineteen mothers (62%) had a PSQI greater than 5 indicating poor sleep. Two subjects had a PSQI score of 20 indicating severely bad sleep. From the PSQI, the mothers mean(SD, range) time spent in bed was 8.3 hrs(0.9,7-10) and the mean actual self-reported hours of sleep was 6.6 hrs(1.5, 4-9). Sleep efficiency for the group was calculated as 78.3%(14.4, 47-100).

The data were analysed as a continuous measure (Statistics for continuous variables in Appendix F) to illustrate the lack of significance in the anthropometric measures relating to all sleep categories. Only PSQI significantly correlated to the depression score

The anthropometric measurements for the mothers' body mass, height, waist circumference, hip circumference, adjusted mid-arm (AMA) percentage and recalled mass at pre-pregnancy and post-partum are shown in Table 3.1. Thirty of the thirty-one subjects were in the normal range for body mass index (BMI) whereas one subject was obese with a BMI of 52.1. The mean waist hip ratio (WHR) was below 0.85 and therefore within normal

limits for all mothers. Body mass loss from pre-pregnancy to the date of survey was on average ranged from a loss of 16kg to a gain of 20kgs with a median of -1.0kg. The mean change in body mass for the group was a loss of 1.3kg (8.0,-16-20)kg.

Table 3.1. Anthropometric data from unipara mothers

Anthropometric data	Mean (SD, Range)
Mass (kg)	62.7 (9.4, 51-95)
Height (cm)	165.3 (8.6, 135-180)
Body Mass Index (BMI)	23.3 (6.1,17.4-52.1)
Waist circumference (cm)	76.4 (7.5, 65-100)
Hip circumference (cm)	99.7 (7.7, 87-127)
Waist hip ratio (WHR)	0.8 (0.1, 0.7-0.8)
Midarm circumference (cm)	28.3 (3.9, 20.0-40)
Tricep skinfold thickness (cm)	1.7 (0.8, 4-40)
Adjusted mid-arm percentage(%)	61.4 (16.7, 30.3-117.8)
Mass on date of questionnaire (kg)	62.7 (9.4, 51-95)
All data represented as mean (SD, Range)	

There were no significant differences between the two groups with respect to employment status ($t=-0.235$, $p=0.408$), education ($t=0.536$, $p=0.298$). There was no significant impact of a family history of diabetes ($t=0.322$, $p=0.375$) or depression ($t=0.977$, $p=0.168$) using unpaired t-tests with df 29 on mothers in this study.

3.1.2. Family History of Diabetes

A family history of diabetes was found in 23% of the mothers and 16% of mothers had detectable glucose in their urine during pregnancy. There was no significant association between mothers with a family history of diabetes and mothers with glucose in the urine during pregnancy ($p=0.343$, $r=-0.02$, Pearson's correlation).

3.1.3. Family History of Depression

A family history of diagnosed depression was reported in 35% of the subjects. These eleven subjects had a mean BDI-II score of 14.8 (7.8, 5-28). Twenty subjects had no history of depression and a slightly lower mean BDI-II score of 13.3 (9.8, 3-40). There was no significant difference in the BDI score between those women with a family history of depression and those without ($t(29)=0.4555$, $p=0.33$, unpaired t-test).

3.1.4 Change in mothers' body mass from 6 months postpartum to 12 months postpartum

Mothers were split into two groups according to baby age 5-8 and 9-12 months to assess if there were any differences in mothers weight in the 6-12 months following birth. Mothers mean change in body mass in the former group was 1.3kg (8.2, -13 - 20) and 1.3kg (8, 16 - 15) in the latter. An unpaired t-test showed no significantly different body mass change in the two groups of mothers ($t(29)=1.7$, $p=0.489$) despite the age of the baby.

3.1.5. Baby Information

There were twenty male babies and eleven female babies and the mean age of the infants at the date of survey was 8.8 months (2.7,5-13). There were sixteen infants in the age group 5-8 months and fifteen in the 9-12 month group. The mean babies' birth mass was 3.1 kg (0.4, 2.1-3.9). There were no significant differences between the gender of the babies for age, weight or sleeping schedules as Table 3.2 highlights.

Table 3.2. Gender influence on baby data

	Male	Female	Df=29 t-value	p-value
Age (months)	9 (2.8, 5-13)	9 (2, 6-13)	0.169	0.433
Baby body mass (kg)	3 (0.4, 2-4)	3 (0, 2-4)	1.097	0.141
Daytime naps(hours)	2 (1.6, 0-6)	3 (2, 0-5)	-1.195	0.121
Baby Arousals (number)	2 (1.8, 0-6)	1 (1, 0-3)	1.596	0.061

All data mean (SD, Range) and comparison done using unpaired t-tests

The duration of daytime naps of all the babies are shown in the frequency distribution (Figure 3.1.) and range from 0 to 5.5 hours during the day.

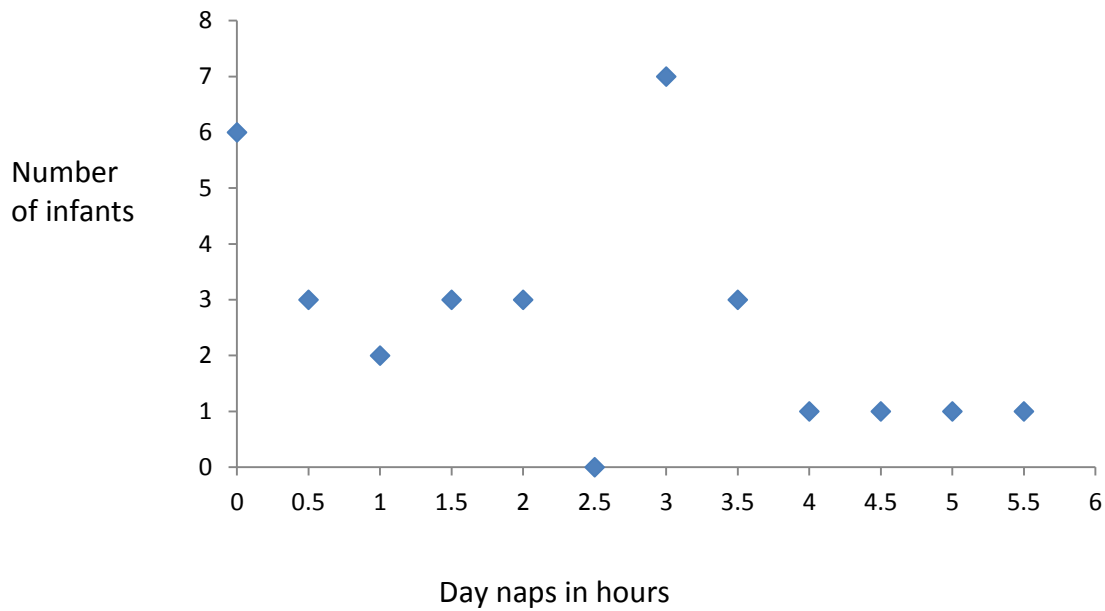


Figure 3.1. The frequency distribution for the duration of daytime naps of the 31 infants

The number of baby arousals in an 8 hr period at night ranged from 0 to 6 times per night (Figure 3.2). The mean number of baby arousals for the whole group was 1.6 times(1.6, 0-6range). Four of the mothers reported that their infant had a sleeping problem. These infants woke up a mean of 3.5 times(0.6, 3-4) which is significantly more often than the infants of mothers not reporting a sleeping problem 1.3 times(1.5, 0-6.) using and unpaired students t-test where $t(29)=-2.74$, $p=0.005$.

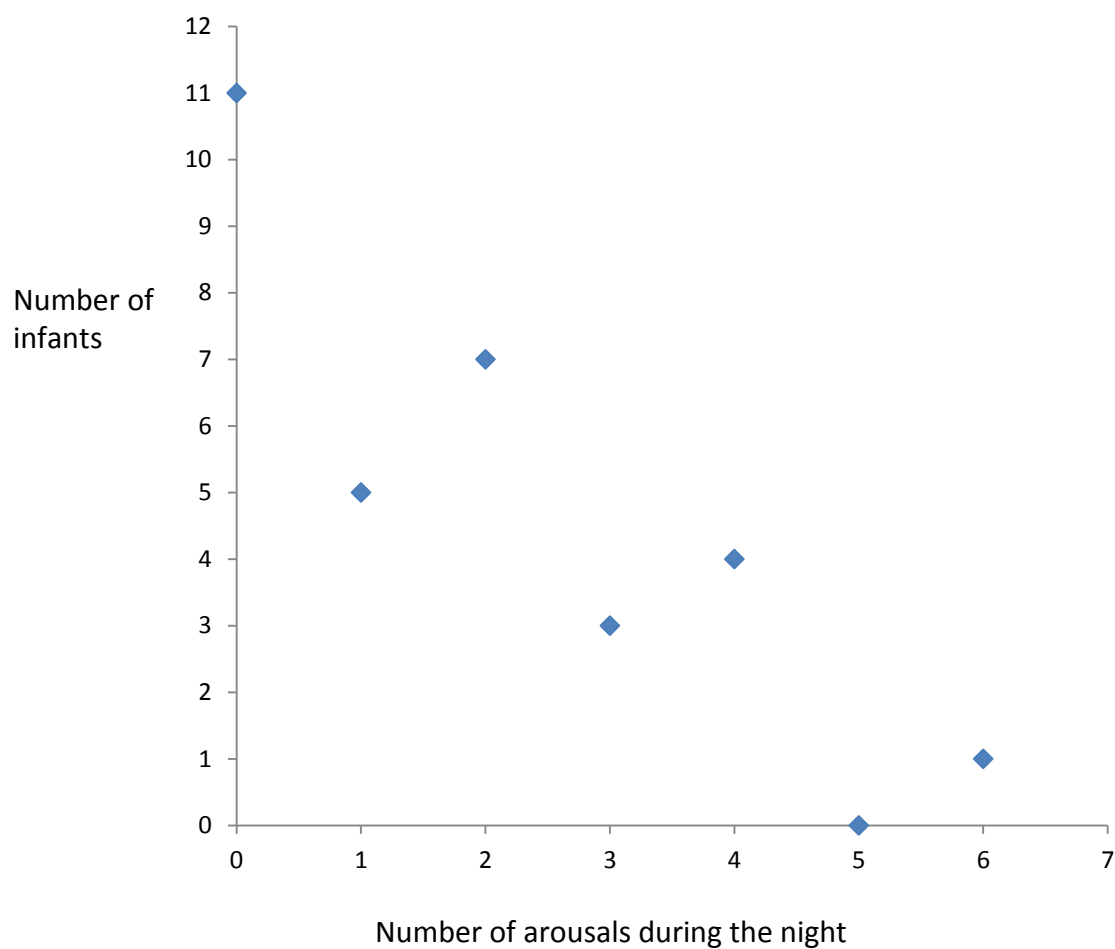


Figure 3.2. Frequency distribution for the number of arousals of 31 infants during an 8 hour period at night

3.1.6. Assistance with Baby

All the babies in this study were sleeping in their own cot in a separate room to the mothers. There were nineteen mothers with no support and twelve mothers with assistance, mostly by their husbands, mothers or night nurses. The mean BDI-II score for the unassisted group was 13.9(7.9, 4-28) and 13.7(10.9, 3-40) for the supported group. An unpaired t-test showed no significant difference in depression scores where $t(29) = 0.067$, $p = 0.4734$).

3.2. Quantifying Sleep Disruption

The mothers were categorised into groups determined by

- Pittsburg Sleep Quality Index (PSQI) score (≤ 5 and > 5)
- Number of self-reported actual hours of sleep at night (≥ 7 or < 7)
- Sleep Efficiency ($< 80\%$ and $\geq 80\%$)
- Number of baby arousals during an 8hr period at night (≤ 1 or ≥ 2)

The mean PSQI score for the whole group of mothers was 7.7 (4.9, 0-20). A PSQI above 5 is considered poor sleep (Gottlieb D *et al*, 2006). The mean number of times the baby woke up during the night according to mothers' recall was 1.6 times (1.6, 0-6). The average number

of hours of self-reported sleep was 6.6 hrs(1.5, 4-9). Sleep efficiency as a mean percentage was 78.3% (14.4,47-100).

The PSQI score significantly negatively correlated with hours of sleep and sleep efficiency of the mothers. Table 3.3 shows Pearsons correlation values. Hours of sleep and sleep efficiency were significantly correlated. The number of baby arousals was not significantly correlated with PSQI, sleep efficiency or hours of sleep at night. There are no significant differences between any category of sleep. PSQI ($p=0.423$), hours sleep ($p=0.132$) and sleep efficiency ($p=0.432$) using Fishers Exact Tests.

Table 3.3. Correlations between sleep measurements

	Baby arousals	Hours of sleep	Sleep efficiency
PSQI (score)	$r=0.28$ $p=0.1256$	$r=-0.82$ $p<0.0001$	$r=-0.84$ $p<0.0001$
Baby arousals (times)		$r=-0.27$ $p=0.1418$	$r=-0.24$ $p=0.1934$
Hours of sleep (hours)			$r=0.89$ $p<0.0001$

3.3. Sleep Quality and Anthropometric Measures

Differences in mothers' age and anthropometric differences were analysed using poor and good sleep categories for PSQI, hours of sleep at night, sleep efficiency and number of baby arousals.

3.3.1. Pittsburg Sleep Quality Index (PSQI)

There were no significant differences between the mothers with a PSQI $\leq$ 5 (good quality sleep) and the mothers with PSQI $>$ 5 (poor quality sleep) with respect to age or any anthropometric data (Table 3.4.). There were also no significant differences between the two groups with respect to employment status ($t=-1.338$, $p=0.09$), education ($t=-0.571$, $p=0.286$) and family history of diabetes ($t=-0.248$, $p=0.403$) or depression ($t=-0.953$, $p=0.174$) using unpaired t-Tests with df at 29 for all.

Table 3.4. PSQI scores comparing mothers age and anthropometrics between poor and good sleepers

	PSQI $\leq$ 5	PSQI $>$ 5	Df=29 t-value	p-value
n	12	19		
Mother's age (yrs)	29.9 (3.7, 25-37)	32.2(4, 26-40)	0.559	0.06
Body Mass Index (BMI)	22.9 (3, 19.5-29)	23.6 (7.5, 17.4-52.1)	-0.0272	0.39
Waist hip ratio (WHR)	0.76 (0.0, 0.7-0.8)	0.77 (0.0, 0.7-0.81)	-0.584	0.281
Adjusted mid-arm percentage(%)	61.5 (15.12,)	61.3(17.9)	0.29	0.9674
Change in mass from pre-pregnancy to date of questionnaire	0.9 (8.5, -16-15)	1.6 (7.8, -13-20)	0.559	0.8149

All data mean (SD,Range) and comparison done using unpaired t-tests

3.3.2. Hours of Sleep at Night

Those mothers sleeping less than 7hours were classified as short sleepers whereas subjects sleeping more than 7hours were defined as long sleepers. There were no significant differences between the long sleeping group (>7hours) and the short sleeping ($\leq$ 7hours) group with respect to age or any anthropometric data (Table 3.5.). There were no significant differences between the two groups with respect to employment status ($t=-0.352$, $p=0.364$), education ($t=-0.495$, $p=0.312$) and family history with respect to diabetes ($t=0.664$, $p=0.256$) and depression ($t=0.352$, $p=0.364$) using unpaired t-tests with df of 29.

Table 3.5. Mothers' age and anthropometrics between poor and good sleepers using hours of sleep

	≥ 7 hours sleep	< 7 hours sleep	Df=29 t-value	p-value
n	15	16		
Mother's age (yrs)	30.6 (3.7, 25-37)	31.9 (4.1, 26-40)	-1.375	0.0899
Body Mass Index (BMI)	24.7 (8.1, 18.9-27.5)	22 (3.1, 17.4-52.1)	-0.232	0.409
Waist hip ratio (WHR)	0.75 (0.04, 0.68- 0.8)	0.77 (0.03, 0.7-0.83)	-1.223	0.116
Adjusted mid-arm percentage(%)	64.6 (20.1, 15-90)	58.4 (12.5, 5-95)	0.516	0.305
Change in mass from pre-pregnancy to date of questionnaire	0.4 (9.6, -16-15)	2.2 (6.3,-13-20)	0.122	0.452

All data shown as mean (SD, Range) and comparison done using unpaired t-tests

3.3.3. Sleep Efficiency

There were no significant differences between the efficient sleepers ($\geq 80\%$ sleep efficiency) and the inefficient sleep ($< 80\%$ sleep efficiency) group with respect to age, or any anthropometric data (Table 3.6.). There were no significant differences between the two groups with respect to employment status ($t=1.251$, $p=0.11$), education ($t=1.197$, $p=0.12$) and family history with respect to diabetes ($t=-0.511$, $p=0.306$) and depression ($t=0.235$, $p=0.408$) using unpaired t-tests with df of 29..

Table 3.6. Mothers' age and anthropometrics between poor and good sleepers using sleep efficiency

	$\geq 80\%$ sleep efficiency	$< 80\%$ sleep efficiency	Df=29 T value	p-value
n	16	15		
Mother's age (yrs)	30.6 (0.4, 25-37)	32.1 (4.2, 26-40)	-1.06	0.148
Body Mass Index				
(BMI) units	22.3 (2.9, 17.5-27.5)	24.5 (8.3, 17.4-52.1)	-1.003	0.162
Waist hip ratio				
(WHR)	0.78 (0.04, 0.68-0.81)	0.77 (0.04, 0.7-0.83)	-0.459	0.325
Adjusted mid-arm				
percentage(%)	54.4 (31.6, 5-95)	65.7 (29.5, 5-95)	-1.028	0.156
Change in mass from pre-pregnancy to date of questionnaire (kg)	-3.2 (8.0, -16-15)	0.7 (7.6, -12-20)	-1.405	0.085

All data shown as mean (SD, Range) and comparison done using unpaired t-tests

3.3.4 Baby Arousals

There were no significant differences between the age or anthropometrics of mothers' whose babies woke ≤ 1 times at night and those whose babies woke ≥ 2 per night shown in Table 3.7.. There were no significant differences between the two groups with respect to employment status ($t=-0.235$, $p=0.408$), education ($t=0.536$, $p=0.298$). There was no significant impact of a family history of diabetes ($t=0.322$, $p=0.375$) or depression ($t=0.977$, $p=0.168$) using unpaired t-tests with degrees of freedom of 29 on mothers in this study.

Table 3.7. Mothers' age and anthropometrics between poor and good sleepers using the number of baby arousals at night.

	≤ 1 baby arousals	>2 baby arousals	Df= 29 t-value	p-value
n	16	15		
Mother's age (yrs)	31.3 (4.1, 25-40)	31.3 (3.9, 26-37)	-0.058	0.477
Body Mass Index (BMI)	24.3 (8.1, 17.5-52.1)	22.3 (2.9, 17.4-29)	0.906	0.186
Waist hip ratio (WHR)	0.76 (0.03, 0.68-0.81)	0.77 (0.04, 0.7-0.83)	-0.865	0.197
Adjusted mid-arm percentage(%)	61.9 (18.7, 5-95)	60.8 (14.8, 5-95)	-0.028	0.489
Change in mass from pre- pregnancy to date of questionnaire	0.1 (9.1, -13-20)	2.6 (6.5, -16-7)	0.874	0.195

All data shown as mean (SD, Range) and comparison done using unpaired t-tests

The data were analysed as a continuous measure to illustrate the lack of significance within the data relating to age, education, employment and all measures of anthropometry. Only PSQI significantly correlated to the depression score. Table 3.8 illustrates the correlations between different sleep measures and BMI, WHR and BDI-II.

Table 3.8.. Correlations between sleep measurements and mothers' anthropometric and depression data

	BMI	WHR	BDI-II
PSQI (score)	$r=0.03$ $p=0.144$	$r=-0.03$ $p=0.135$	$r=-0.74$ $p<0.0001$
Baby arousals (times)	$r=-0.15$ $p=0.134$	$r=0.21$ $p=0.142$	$r=-0.15$ $p=0.183$
Hours of sleep (hours)	$r=0.09$ $p=0.144$	$r=-0.15$ $p=0.134$	$r=-0.48$ $p=0.174$
Sleep efficiency (%)	$r=-0.595$ $p=0.182$	$r=-0.074$ $p=0.162$	$r=-0.595$ $p=0.143$

3.4. Depression and Sleep

The Becks Depression Inventory II score was 13.8 (9.1, 3-40) for the total sample. There were nineteen subjects with BDI-II scores ≤ 13 (minimally depressed) and twelve subjects with scores 14 to 40. Four of the mothers had scores indicating severe depression (28-40). PSQI scores, sleep efficiency and hours of sleep were significantly higher in the group with the higher BDI-II scores compared to the mothers with the lower BDI-II scores (Table 3.9). The number of baby arousals were not significantly different between the non-depressed and the depressed groups of mothers.

Table 3.9. Mothers' Becks Depression-II (BDI-II) score between poor and good sleepers using all the sleep measurements.

	BDI-II score 0-13	BDI-II score 14-40	Df=29 t-value	p-value
Pittsburgh Sleep Quality Index	5.4 (3, 0-11)	11.3 (5.3, 4-20)	-3.931	0.0002
Baby Arousals (number)	1.3 (1.4, 0-4)	2.1 (1.9, 0-6)	-1.294	0.103
Hours of sleep (hr)	7 (1.4, 4.5-9)	5.9 (1.4, 4-8)	2.138	0.021
Sleep Efficiency (%)	83.2 (11.7, 62.5-100)	70.5 (15.1, 47.2-92.9)	2.637	0.007

All data mean (SD, Range) and comparison done using unpaired t-tests

3.5. Antidepressants

Antidepressant (ATD) medication was used by six of the subjects. The subjects using ATD's were significantly more depressed ($t=-2.861$, $p=0.0039$) than the mothers not using ATD's. There were six mothers using SSRI's. BDI-II scores in mothers using ATD's 22.3 (10.5, 13-40) are significantly higher than mothers not using ATD's 11.8 (7.6, 3-29). Mothers using antidepressants had significantly increased PSQI scores. Self reported hours of sleep in bed and sleep efficiency were not significantly different between the two groups (Table 3.10).

Table 3.10. The affect of antidepressant use on mothers' sleep quality

	Using antidepressants	Not using antidepressants	Df=29 t-value	p-value
Pittsburgh Sleep Quality Index	11.5 (5.3, 7-20)	6.8 (4.5, 0-20)	-2.273	0.015
Baby Arousals (number)	0.5 (0.8, 0-2)	1.3 (1.4, 0-6)	1.952	0.030
Hours of sleep (hr)	6.0 (1.2, 4.5-7.5)	6.7 (1.5, 4-9)	1.015	0.159
Sleep Efficiency (%)	71.1 (11.9, 52.9-83.3)	80.0 (14.6, 47.1-100)	1.392	0.0872

All data mean (SD, Range) and comparison done using unpaired t-tests

3.6. Oral Contraceptives and Body Mass

Twelve mothers were on monophasic, low-dose combined oral contraceptives (OC). There were no significant differences found when comparing pre-pregnancy body mass with post partum body mass whether between mothers who were taking oral contraceptives and those who were not (Table 3.11).

Table 3.11. Oral contraceptives effect on body mass, 6 months postpartum

	Using oral contraceptives	No oral contraceptives	Df=29 t-value	p-value
Pre-pregnancy body mass (kg)	63.4 (8.8, 52-80)	64.3 (9.6, 50-81)	0.239	0.406
6 weeks post partum body mass (kg)	63.7 (5.6, 56-77)	76.9 (10.8, 56-100)	0.958	0.173
6 – 12 months post partum body mass (kg)	60.3 (7.5, 51-78)	64.0 (10.7, 52-95)	0.927	0.181

All data shown as mean (SD, Range) and comparison done using unpaired t-tests

3.7. Oral Contraceptives and Sleep

Mothers not on OC had a significantly lower PSQI score indicating poor quality sleep when using oral contraceptives (Table 3.12.). Whilst there was no significant difference in the hours of slept by women taking oral contraceptives, they had significantly better sleep efficiency.

Table 3.12. The affect of oral contraceptive use on sleep quality

	Using oral contraceptives	No oral contraceptives	Df=29 t-value	p-value
PSQI	10.9 (5.4, 0-11)	5.7 (3.4, 3-20)	-2.929	0.003
Baby Arousals (number)	2.1 (1.9, 0-6)	1.2 (1.4, 0-4)	-1.615	0.059
Hours of sleep (hr)	6 (1.5, 4-8)	6.9 (1.4, 4.5-9)	1.687	0.051
Sleep Efficiency (%)	71.7 (15.9, 47-94)	82.2 (12.2, 62.5-100)	1.844	0.038

All data shown as mean (SD, Range) and comparison done using unpaired t-tests

CHAPTER FOUR – Discussion

This sample of mothers' showed that poor sleep (in all categories) results in no significant difference to body mass post-partum. There is no increased occurrence of diabetes and the medication related to insulin control irrespective of family history. A family history of depression does not significantly increase the risk of postpartum depression.. Antidepressant users are significantly poorer sleepers and significantly more depressed than good sleepers in this study. The nature of the relationship between poor sleep, depression and oral contraceptive use is still unclear as there is little available research done on this topic (to my knowledge). Oral contraceptive have been shown to significantly decrease slow wave sleep, in only one study (Baker F *et al*, 2001) to my knowledge.

Age, anthropometry and sleep in subjects

The mothers' mean age was older than the highest average in the world which is New Zealand at 29.9 yrs (Mayberry L *et al*, 2007). Increasing age has been shown to significantly decrease sleep quality (Hakan A. 2011) and there is increased association with depression (Dombrovski A *et al*, 2008).

There was no significant difference in the body mass of mothers' in this study between 6 months compared to 12 months after birth. These mothers were mostly back to their pre-pregnancy weights very quickly after birth. The difference in body mass of mothers' of 5-8 month olds was not significantly different to mothers' of 9-12 month olds. I expected that with time, there would have been an effect on mothers' body mass, for example, that the

mothers' not sleeping would be gaining weight or not losing it as quickly as the mothers' that were sleeping well.

A family history of diabetes had no significant effect on mothers' urine glucose while they were pregnant in this study in accordance with some of the literature (Mitchell B *et al*, 1995). A family history of diabetes has been shown to increase the risk of diabetes in a dose-response manner (Kim C *et al*, 2009). The risk is higher when both parents are affected than when only one is (Meigs J *et al*, 2000, Harris M *et al*, 1998). Maternal diabetes may (Karter A *et al*, 1999) or may not (Mitchell B *et al*, 1995) cause greater risk of diabetes in offspring than paternal diabetes.

Baby Gender

The gender of the baby did not have a significant effect on the amount of sleep the baby or the mother was getting. Two studies done by Foreman SW *et al* (2008) showed male preterm infants required significantly more care than female preterm infants, other studies suggest that gender does not play a role in sleep in infants (Richardson H. 2010) as was the case in my study.

Baby Sleep

Baby day naps in this study showed a wide range of up to six infants that didn't nap during the day, to one infant napping for up to 5.5 hours during the day. After 6 months of age the amount of sleep depends on the personality of the baby and the training the care-giver has

provided rather than the sex of the baby (Bernal J. 1973). Some factors identified as negatively impacting infant sleep include TV viewing (Thompson D *et al*, 2005) and past history of maternal depression (Bayer J *et al*, 2007).

Baby age did not affect whether the child slept through the night or not. Once the baby's stomach has the capacity to fill enough to sustain the child throughout the night (from about 4 months) there is no difference in the baby's ability to fall asleep and stay asleep. A baby will cycle through its sleep stages and if it knows how to soothe itself back to sleep it will not wake its caregiver (Franco P *et al*, 2010). By the time the child is 8 to 9 months of age, 70% of parents report that their infant sleeps through the night regularly (Anders T. 1992).

The range of baby arousals in an 8 hour period at night was from 0 times to 6 times per night. It is not only the babies that disturb the mothers since there were no significant differences in baby arousals in any of the sleep disrupted or the sleeping groups of mothers'. The number of baby arousals at night had no significant impact on the PSQI, sleep hours or sleep efficiency of mothers' sleep but they did determine whether mothers thought that their baby had a sleep problem or not.

Measuring sleep

The number of hours of sleep and sleep efficiency were used as independent variables to test the viability of using them instead of the PSQI test. It is expected that as the PSQI score increases, the number of hours of sleep decreases and the sleep efficiency decreases. A

diagnostic clinician could ask the patient for hours of sleep and sleep efficiency information instead of administering a whole PSQI which is time consuming and arduous to score.

Anthropometric Measures and Sleep Quality

The quantity and quality of sleep did not significantly alter the anthropometrics of South African Caucasian mothers in this study. I expected increased body mass in the mothers with poor sleep . The unusual anthropometry i.e. low BMI, WHR and AMA observed in this sample of mothers could be due to their employment, high level of education, affluence and perhaps a societal expectation of their body image. Higher education has been associated with the selection of thinner body shapes as healthiest. BMI and education have both been positively related to body dissatisfaction (Diamond D *et al*, 2009).

When mothers were split into poor and good sleepers using PSQI, hours of sleep, sleep efficiency and number of baby arousals there were no significant differences in the age or anthropometric data. Poor sleep has been linked to weight loss (Tamakoshi A *et al*, 2004, Jung C *et al*, 2011), no change in body mass (Dorheim S *et al*, 2009), or weight gain (Gottlieb D *et al*, 2006, Heslop P *et al*, 2002, Anderson S *et al*, 2010, Locard E *et al*, 1992, Watson N *et al*, 2010). A review on studies done on short sleep duration and weight gain also had inconclusive results (Patel S *et al*, 2008).

Other factors affecting maternal sleep

A family history of depression did not predict the occurrence of depression in the mothers that were sampled. Familial depression has been predictive of postpartum depression in some studies (Johnstone S *et al*, 2001), but other studies show no relationship between a woman's family history of depression and her own likelihood of developing postpartum depression (Bloch M *et al*, 2006, Dennis C *et al*, 2004).

Family support has been shown to lower depression and aid in the recovery from depression (Bloch M *et al*, 2006, Dennis C *et al*, 2004). This study showed no significant differences in the depression of assisted and unassisted mothers in contrast to the literature. This suggests that the depressed mothers' were not depressed due to excessive baby arousals.

The nineteen mothers falling into the depressed category had significantly increased PSQI scores, and significantly lower hours of sleep and sleep efficiency percentage indicating significantly poor sleep compared to non-depressed mothers. Depression was significantly associated with poor sleep quality in this study, as well as other research. (Germaine A *et al*, 2008, Babson K *et al*, 2010). Poor sleep quality could increase the risk of depression (Babson K *et al*, 2010, Tadavarty R *et al*, 2011) although causality in the relationship between poor sleep and depression has not been confirmed.

The most common severe mood disorder affecting women during the first year after childbirth is postpartum depression (Gavin N *et al*, 2005). There is a clear relationship between the number of stressful life events and depression (Caspi A *et al*, 2003). The postpartum period is characterised by pain, physical exertion, and psychological stress

(Dennis C *et al*, 2004). There were 39% of women in this study with postpartum depression 6-12 months after giving birth. This is consistent with the literature from low- and middle-income countries showing a prevalence of depression of 35-40% (Hanlon C *et al*, 2009). Depression has been linked to socioeconomic status where higher paid, first time mothers were less prone to post partum depression (Goyal D *et al*, 2010) in contrast to what was found in this study. This could be due to stress from the constant threat of crime in Gauteng, South Africa.

Postpartum depression has dangerous implications for a new mother's health and well-being and for the health and development of her infant. The disorder may interfere with maternal role development and mother infant bonding and may increase physical risk in mother and child (Beck A *et al*, 1996). The effects on children may include behavioral, developmental, socio-emotional, and cognitive delay (Logsdon M *et al*, 2006). Women with higher levels of psychological distress, depression or other diagnosis, might judge their sleep to be poorer, that may confound stress and frustration creating a potentially vicious cycle (Corwin E *et al*, 2010).

Depression has been shown to cause chronic fatigue, increased physical aches and pains and a lowered immune system (Bonnet M *et al*, 1995). Increased stress hormones suppress the immune system further. Depressed subjects spend an excessive amount of time in REM sleep and not slow wave sleep where repair within the body occurs (Duffy J *et al*, 2002). There could be a long-term hormonal imbalance due to the pregnancy perhaps needing more than a year to rebalance as in the case of post-partum depression (Bowman M *et al*, 2009). There is evidence that depression can continue for more than 2 years

postpartum in unipara women (Small R *et al*, 2004). Depression has been shown to result in a change in serotonin levels that require treatment with SSRIs (Paredes S *et al*, 2009).

Antidepressants should first act on regulating sleep and then correct the depression (Maes M *et al*, 1997). The six mothers on antidepressants reported significantly less baby arousals. The antidepressants used by these mothers were not sedating allowing mothers to respond to baby arousals, so that was not the only reason for their poor sleep. Those mothers' on antidepressants were more likely to be depressed and depression can be associated with wakefulness.

Antidepressant users had a significantly higher mean PSQI score indicating poor sleep which is typical of depression and is also linked to the fact that their BDI-II scores were higher than the mothers not taking antidepressants. There was however no significant differences in hours of sleep or sleep efficiency. This shows the importance of using the full PSQI in diagnosing sleep problems.

In previous studies, oral contraceptive use had no significant impact on weight (Berenson A *et al*, 2009, Kattapong R *et al*, 1995) and this was confirmed in this study.

Oral contraceptive use was significantly associated with poor sleep in these mothers. Mothers using oral contraceptives had significantly higher PSQI scores and lower hours of sleep and sleep efficiency. Oral contraceptives have been shown to significantly decrease slow wave sleep, (Baker F *et al* 2001) that decreases restorative sleep, therefore negatively affecting sleep quality (Webley G *et al*, 1986, Kattapong R *et al*, 1995).

Mothers on oral contraception were significantly more depressed than those not using oral contraceptive. This study showed that contraceptive use was significantly associated with decreased sleep and increased depression. Their poor sleep and frequent baby arousals are confounding factors of the depression rather than the use of oral contraceptives, however use of oral contraceptives has been associated with higher depression levels (Oinonen K *et al*, 2001).

The small sample size is a limitation of this study. A control group of 31 women matched for age, race, employment and education would have been useful to isolate the effect of having a 6-12 month old baby.

What are the anthropometric and depression scores of women with children sleeping poorly for longer than 6-12 months. Does the effect of more sleep deprivation over a longer period of time, confound negative anthropometric measurements and depression ?

There was no clinical biochemistry performed to quantify the metabolic variables affecting the women in the study. Testing plasma insulin and glucose levels before and after a glucose load would assist in determining diabetic tendencies. Including obese women would diversify the anthropometry of the study

The questionnaire did not ask the length of treatment of medication in either antidepressants or contraceptives. Antidepressants could have been consumed prior to and

throughout the pregnancy, however, these subjects could not have been on oral contraception for more than 6 months.

The sample in this study was not indicative of the South African population in general but more of a first world environment. Perhaps a study of sleeping versus non-sleeping mothers and a control group would need to be performed locally and internationally in poorer and wealthier socio-economic classes.

This study focuses on exploring South African women more than 6 months post-partum to assess their age, education, employment status, sleeping habits, their baby's sleep and the effects of long-term, sustained, partial sleep deprivation on their body mass and various other anthropometric measurements. Different assessments of sleep measures are discussed and compared to one another to ascertain which measures of sleep are important to record. Depression is assessed and compared to quality of sleep and medication use.

There has not been this kind of study in South Africa (or to my knowledge, the world) before.

CHAPTER FIVE - Conclusions

The recovery of a mother from the birth process and the postpartum experience is a dynamic time. There are many variables that affect a new mother.

In this small study, age, education, employment, familial diabetes or familial depression did not significantly effect sleep quality. Anthropometric measures including changes in body mass over time, BMI, WHR, AMA at the date of survey were not significantly affected by sleep quality.

A bidirectional relationship between depression and sleep was found; where depressed subjects had significantly higher PSQI scores and sleep deprived subjects had significantly higher BDI-II scores.

Antidepressant users were significantly more depressed than non-users, although they had been treated for depression implying that the medication did not seem to be effective.

Oral contraceptive use had no effect on body mass in this sample of the population. Those mothers' taking oral contraceptives had significantly decreased subjective sleep quality.

More research is required into the extent of sleep deprivation and the clinical, social, and behavioral risk factors associated with sleep deprivation among postpartum women after 6 months.

REFERENCES

- Adair, R., Bauchner, H. Sleep problems in childhood. *Curr Probl Pediatr* 23, 147-170. 1993.
- AlDabal, L., BaHammam, A. Metabolic, endocrines and immune consequences of sleep deprivation. *The Open Resp Med J* 5, 31-43. 2011
- Almagor, M. Ben-Porath, Y. Mood changes during the menstrual cycle and their relation to the use of oral contraceptive. *J Psychosom Res* 35, 721-728. 1991.
- Anderson, S., Whitaker, R. Household routines and obesity in US preschool-aged children. *Pediatrics* 125, 420-428. 2010.
- Anuntaseree, W., Mo-suwan, L., Vasiknanonte, P., Kuasirikul, S., Ma-a-lee, A., Choprapawan, C. Night waking in Thai infants at 3 months of age: association between parental practices and infant sleep. *Sleep Med* 9, 564-571. 2008.
- Atalay H. Comorbidity of insomnia detected by the Pittsburgh Sleep Quality Index with anxiety, depression and personality disorders. *Isr J Psych Relat Sci* 48, 54-62. 2011.
- Ayas, N., White, D., Manson, J., Stampfer, M., Speizer, F., Malhotra, A., Hu, F. A prospective study of sleep duration and coronary heart disease in women. *Arch Intern Med* 163, 205-9. 2003.
- Babson, K., Trainor, C., Feldner, M., Blumenthal, H. A test of the effects of acute sleep deprivation on general and specific self-reported anxiety and depressive symptoms: an experimental extension. *J Behav Ther Exp Psychiatry* 41, 297-303. 2010.
- Bagnasco, M., Kalra, P., Kalra, S. Ghrelin and leptin pulse discharge in fed and fasted rats. *Endocrinology* 143,726-729. 2002.

Baker, F., Selsick, H., Driver, H., Taylor, S., Mitchell, D. Different nocturnal body temperatures and sleep with forced-air warming in men and in women taking hormonal contraceptives. *J Sleep Res* 7, 175-181. 1998.

Baker, F., Mitchell, D., Driver, H. Oral Contraceptives alter sleep and raise body temperature in young women. *Pflugers Arch* 442, 729-737. 2001.

Baker, F., Waner, J., Vieira, E., Taylor, S., Driver, H., Mitchell, D. Sleep and 24 hour body temperatures: a comparison in young men, naturally cycling women and women taking hormonal contraceptives. *J Physiol* 1, 565-574. 2001.

Baker, J., Michaelsen, K., Sorensen, T., Rasmussen, K. High pre-pregnant body mass index is associated with early termination of full and any breastfeeding in Danish women. *Am J Clin Nutr* 86, 404-411. 2007.

Balkin, T., Rupp, T., Picchioni, D., Wesensten, N.. Sleep loss and sleepiness. *Chest* 134,653-660.2008.

Banks, S., Dinges, D. Behavioural and physiological consequences of sleep restriction. *J Clin Sleep Med* 3, 519-528. 2007.

Bayer, J., Hiscock, H., Hampton, A., Wake, M. Sleep problems in young infants and maternal mental and physical health. *J Paed and Child Health* 43, 66-73. 2007.

Beck, A., Steer, R., Ball, R., Ranieri, W. Comparison of Beck Depression Inventories -IA and -II in psychiatric outpatients. *J Pers Assess* 67, 588-597. 1996.

Becker, P. Insomnia: prevalence, impact, pathogenesis, differential diagnosis, and evaluation. (Review) *Psychiatr Clin North Am* 29,855-870. 2006.

Beeber, L., Miles, M. Maternal mental health and parenting in poverty. *Ann Rev Nurs Res* 21, 303-331. 2003.

Beeghly, M., Olson, K., Weinberg, M., Pierre, S., Downey, N., Tronick, E. Prevalence, stability and sociodemographic correlates of depressive symptoms in black mothers during the first 18 months postpartum. *Matern Child Health J* 7, 157-168. 2003.

Bei, B. Milgrom, J., Ericksen, J., Trinder, J. Subjective perception of sleep, but not its objective quality, is associated with immediate postpartum mood disturbances in healthy women. *Sleep* 33, 531-538. 2010.

Berber, A., Gómez-Santos, R., Fanghänel, G., Sánchez-Reyes, L. Anthropometric indexes in the prediction of Type 2 Diabetes mellitus, hypertension and dyslipidaemia in a Mexican population. *Int J Obes* 25, 1794-9. 2001.

Berenson, A.B., Rahman, M. Changes in weight, total fat, percent body fat, and central-to-peripheral fat ratio associated with injectable and oral contraceptive use. *Am J Obstet Gynecol* 200, 329.e1-8. 2009.

Bernal, J. Night waking in infants during the first 14 months. *Dev Med Child Neurol* 15, 362-372. 1973.

Beydoun, M.A., Kuczmarski, M.T., Wang, Y., Mason, M.A., Evans, M.K., Zonderman, A.B. Receiver-operating characteristics of adiposity for metabolic syndrome: the Healthy Aging in Neighborhoods of Diversity across the Life Span (HANDLS) study. *Pub Health Nutr* 14, 77-92. 2011.

Bliwise, D., King, A., Harris, R., Haskell, W. Prevalence of self-reported poor sleep in a healthy population aged 50-65. *Soc Sci Med* 34, 49-55. 1992.

Bloch, M., Rotenberg, N., Koren, D., Klein, E. Risk factors for early postpartum depressive symptoms. *Gen Hosp Psych* 28, 3-8. 2006.

- Bodosi, B., Gardi, J., Hajdu, I., Szentirmai, E., Obal, F. Jr., Krueger, J.M. Rhythms of ghrelin, leptin, and sleep in rats: effects of the normal diurnal cycle, restricted feeding, and sleep deprivation. *Am J Physiol Regul Integr Comp Physiol* 287, 1071-1079. 2004.
- Bonnet, M., Arand, D. We are chronically sleep deprived. *Sleep* 18, 908-911. 1995.
- Bowman, M., Neale, A. On postpartum depression, hormonal problems, and practice management for medical home implementation. *J Am Board Fam Med* 22, 465-467. 2009.
- Bozoky, I., Corwin, E. Fatigue as a Predictor of Postpartum Depression. *J Obstet Gynecol Neonatal Nurs* 31, 436-443. 2002.
- Burdakov, D., Gerasimenko, O., Verkhatsky, A. Physiological changes in glucose differentially modulate the excitability of hypothalamic melanin-concentrating hormone and orexin neurons in situ. *J Neurosci* 25, 2429-2433. 2005.
- Burdakov, D., Jensen, L., Alexopoulos, H., Williams, R., Fearon, I., O'Kelly, I., Gerasimenko, O., Fugger, L., Verkhatsky, A. Tandem-pore K⁺ channels mediate inhibition of orexin neurons by glucose. *Neuron* 50, 711-722. 2006.
- Burnham, M., Goodlin-Jones, B., Gaylor, E., Anders, T. Nighttime sleep-wake patterns and self-soothing from birth to one year of age: a longitudinal intervention study. *J Child Psychol Psychiatry* 43, 713-25. 2002.
- Buyse, D., Hall, M., Strollo, P., Kamarck, T., Owens, J., Lee, L., Reis, S., Matthews, K. Relationships between the Pittsburgh Sleep Quality Index (PSQI), Epworth Sleepiness Scale (ESS) and clinical /polysomnographic measures in a community sample. *J Clin Sleep Med* 4, 563-571. 2008.
- Buyse, D., Reynolds, C., Monk, T., Berman, S., Kupfer, D. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res* 28, 193-213. 1989.

Cain, S., Dennison, C., Zeitzer, J., Guzik, A., Khalsa, S., Santhi, N., Schoen, M., Czeisler, C., Duffy, J. Sex differences in phase angle of entrainment and melatonin amplitude in humans. *J Biol Rhythms* 25, 288-96. 2010.

Campfield, L., Smith, F., Guisez, Y., Devos, R., Burn, P. Recombinant mouse OB protein: evidence for a peripheral signal linking adiposity and central neural networks. *Science* 269, 546-549. 1995.

Cappuccio, F., Strazzullo, P., D'Elia, L., Miller, M. Quantity and quality of sleep and incidence of Type 2 Diabetes: a systemic review and meta-analysis, *Diab Care* 33, 414-420. 2010.

Caspi, A., Sugden, K., Moffitt, T., Taylor, A., Craig, I., Harrington, H., McClay, J., Mill, J., Martin, J., Braithwaite, A., Poulton, R. Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene. *Science* 301, 386-389. 2003.

Chaput, J.P., Despres, J.P., Bouchard, C., Astrup, A., Tremblay, A. Sleep duration as a risk factor for the development of Type 2 Diabetes or impaired glucose tolerance: analyses of the Quebec Family Study. *Sleep Med* 10, 919-924. 2009.

Chuah, L., Dolcos, F., Chen, A., Zheng, H., Parrimal, S. Chee, M. Sleep Deprivation and Interference by Emotional Distracters. *Sleep* 33, 1305-1313. 2010

Cizza, G., Requena, M., Galli, G., De Jonge, L. Chronic sleep deprivation and seasonality: Implications for the obesity epidemic. *J Endocrinol Invest* 34,793-800.2011.

Corwin, E., Kohen, R., Jarrett, M., Stafford, B. The Heritability of Postpartum Depression. *Biol Res Nurs* 12, 73-83. 2010.

Cournot, M., Ruidavets, J., Marquie, J., Esquirol, Y., Baracat, B., Ferrieres, J. Environmental factors associated with body mass index in a population of Southern France. *Eur J Cardiovasc Prev Rehabil* 11, 291-297. 2004.

Cummings, D., Purnell, J., Frayo, R., Schmidova, K., Wisse, B., Weigle, D. A preprandial rise in plasma ghrelin levels suggests a role in meal initiation in humans. *Diab* 50, 1714-1719. 2001.

Cummings, D., Weigle, D., Frayo, S. Plasma ghrelin levels after diet-induced weight loss or gastric bypass surgery. *N Engl J Med* 346,1623-1630. 2002.

Dahl, R. The development and disorders of sleep. *Adv Pediatr* 45, 73-90. 1998.

Deacon, S., Arendt, J. Posture influences melatonin concentrations in plasma and saliva in humans. *Neurosci Letters* 167, 191-194. 1994.

Dement, W., Kleitman, N. The relation of eye movements during sleep to dream activity: an objective method for the study of dreaming. *J Exp Psychol* 53, 339-46. 1957.

Dennis, C., Ross, L. The clinical utility of maternal self-reported personal and familial psychiatric history in identifying women at risk for postpartum depression. *Acta Obstetrica & Gynecologica Scandinavica* 85, 1179-1185. 2006.

Diamond, D., Baylin, A.,Plazas, M.,Villamor, E. Correlates of Obesity and Body Image in Colombian Women. *J Womens Health (Larchmt)* 18, 1145-1151. 2009

Dijk, D., Groeger, J., Stanley, N., Deacon, S. Age-Related Reduction in Daytime Sleep Propensity and Nocturnal Slow Wave Sleep. *Sleep* 33, 211-223. 2010.

Dobbelsteyn, C., Joffres, M., MacLean, D., Flowerdew, G. A comparative evaluation of waist circumference, waist-to-hip ratio and body mass index as indicators of cardiovascular risk factors. The Canadian Heart Health Surveys. *Int J Obes* 25, 652-661. 2001.

Dombrovski, A., Cyranowski, J., Mulsant, B., Houck, P., Buysse, D., Andreescu, C., Thase, M., Mallinger, A., Frank, E. Which symptoms predict recurrence of depression in women treated with maintenance interpersonal psychotherapy? *Depress Anxiety* 25, 1060-1066. 2008.

Dorheim, S., Bondevik, G., Eberhard-Gran, M., Bjorvatn, B. Sleep and Depression in Postpartum Women: A Population-Based Study. *Sleep* 32, 847-855. 2009.

Drevets, W., Frank, E., Price, J., Kupfer, D., Holt, D., Greer, P., Huang, Y., Gautier, C., Mathis, C. Pet imaging of serotonin 1A receptor binding in depression. *Biol Psychiatry* 46, 1375-1387. 1999.

Ducroc, R., Voisin, T., Firar, A., Laburthe, M. Orexins control intestinal glucose transport by distinct neuronal, endocrine and direct epithelial pathways. *Diab* 56, 2494-2500. 2007.

Duffy, J., Cain, S., Chang, A., Phillips, A., Munch, M., Gronfier, C., Wyatt, J., Dijk, D., Wright, K., Czeisler, C. Sex difference in the near-24-hour intrinsic period of the human circadian timing system. *PNAS* 108, 15602-15608. 2011.

Duffy, J., Dijk, D. Getting through to circadian oscillators: why use constant routines? *J Biol Rhythms* 17, 4-13. 2002.

Dzaja, A., Wehrle, R., Lancel, M., Pollmacher, T. Elevated estradiol plasma levels in women with restless legs during pregnancy. *Sleep* 32, 169-174. 2009.

Eberhard-Gran, M., Eskild, A., Opjordsmoen, S. Use of psychotropic medications in treating mood disorders during lactation. Practical recommendations. *CNS Drugs* 20, 187-198. 2006.

Ecochard, R., Gougeon, A. Side of ovulation and cycle characteristics in normally fertile women. *Hum Reprod* 15, 752-755. 2000.

Eley, T., Stevenson, J. Exploring the covariation between anxiety and depression symptoms: a genetic analysis of the effects of age and sex. *J Child Psychol Psychiatry* 40, 1273-1282. 1999.

Everson, C., Crowley, W. Reductions in circulating anabolic hormones induced by sustained sleep deprivation in rats. *Am J Physiol Endocrinol Metab* 286, 1060-70. 2004.

Fombonne, E., Wostear, G., Cooper, V., Harrington, R., Rutter, M. The Maudsley long-term follow-up of child and adolescent depression. 1. Psychiatric outcomes in adulthood. *Br J Psychiatry* 179, 210-217. 2001.

Foreman, S., Thomas, K., Blackburn, S. Individual and gender differences matter in preterm infant state development. *J Obstet Gynecol Neonatal Nurs* 37, 657-665. 2008.

Franco, P., Kato, I., Richardson, H., Yang, J., Montemitro, E., Horne, R. Arousal from sleep mechanisms in infants. *Sleep Med* 11, 603-614. 2010.

Frederick, T., Frederichs, R., Clark, V. Personal health habits and symptoms of depression at the community level. *Prevent Med* 17, 173-182. 1988.

Friedman, E., Love, G., Rosenkranz, M. Urry, H., Davidson, R., Singer, B., Ryff, C. Socioeconomic status predicts objective and subjective sleep quality in aging women. *Psychoso Med* 69, 682-691. 2007.

Fujihara, H., Serino, R., Ueta, Y., Sei, H., Morita, Y. Six-hour selective REM sleep deprivation increases the expression of the galanin gene in the hypothalamus of rats. *Brain Res Mol Brain Res* 119, 152-9. 2003.

Gallagher, D., Visser, M., Sepulveda, D., Pierson, R., Harris, T. How useful is body mass index for comparison of body fatness across age, sex, and ethnic groups? *Am J Epidemiol* 143, 228-39. 1996.

Gallo, M., Grimes, D., Schultz, K., Helmerhorst, F. Combination estrogen-progestin contraceptives and body weight: systemic review of randomized controlled trials. *Obstet Gynecol* 103, 359-373. 2004.

Gangwisch, J.E., Malaspina, D., Boden-Albala, B., Heymsfield, S.B. Inadequate sleep as a risk factor for obesity: analyses of the NHANES I. *Sleep* 28, 1289-96. 2005.

Garn, S., Leonard, W., Hawthorne, V. Three limitations of the body mass index. *Amer J of Clin Nutr* 44, 996-997. 1986.

Gavin, N., Gaynes, B., Lohr, K., Meltzer-Brody, S., Gartlehner, G., Swinson, T. Perinatal depression: A systematic review of prevalence and incidence. *Obs and Gyn* 106, 1071-1083. 2005.

Gay, C., Lee, K., Lee, S. Sleep Patterns and Fatigue in New Mothers and Fathers. *Biol Res Nurse* 5, 311-318. 2004.

Germain, A., Kupfer, D. Circadian rhythm disturbances in depression. *Hum Psychopharmacol* 23, 571-585. 2008.

Gottlieb, D., Redline, S., Nieto, F., Baldwin, C., Newman, A., Resnick, H., Punjabi, N. Association of usual sleep duration with hypertension: the Sleep Heart Health Study. *Sleep* 29, 1009-1014. 2006.

Goyal, D., Gay, C., Lee, K. How much does low socioeconomic status increase the risk of prenatal and postpartum depressive symptoms in first time mothers? *Wom Health Iss* 20, 96-104. 2010.

Graham, J., Lobel, M., DeLuca, R. Anger after childbirth: An overlooked reaction to postpartum stressors. *Psych of Wom Quart* 26, 222-233. 2002.

Grigg-Damberger, M., Gozal, D., Marcus, C., Quan, S., Rosen, C., Chervin, R., Wise, M., Picchietti, D., Sheldon, S., Iber, C. The visual scoring of sleep and arousal in infants and children. *J Clin Sleep Med* 3, 201-240. 2007.

Grossman, B. Managing adverse effects of hormonal contraceptives. *Am Fam Physician* 82, 1499-506. 2010.

Hakan, A. Comorbidity of Insomnia Detected by the Pittsburgh Sleep Quality Index with Anxiety, Depression and Personality Disorders. *Isr J Psychiatry Relat Sci* 48, 54-59. 2011.

Hanlon, C., Medhin, G., Alem, A. Impact of antenatal common mental disorders upon perinatal outcomes in Ethiopia: the P-MaMiE population-based cohort study. *Trop Med Int Health* 14, 156-66. 2009.

Hannaford, P. Cardiovascular events associated with different combined oral contraceptives: a review of current data. *Drug Safety* 22, 361-71. 2000.

Harris, M., Flegal, K., Cowie, C. Prevalence of diabetes, impaired fasting glucose, and impaired glucose tolerance in U.S. adults: the Third National Health and Nutrition Examination Survey. *Diabetes Care* 21, 518. 1998.

Harvey, A. A cognitive model of insomnia. *Behav Res Ther* 40, 869-93. 2002.

Harvey, A., Sharpley, A., Ree, M., Stinson, K., Clark, D. An open trial of cognitive therapy for chronic insomnia. *Behav Res Ther* 45, 2491-501. 2007.

Hasler, G., Buysse, D., Klaghofer, R., Gamma, A., Ajdacic, V., Eich, D., Rossler, W., Angst, J. The association between short sleep duration and obesity in young adults: a 13-year prospective study. *Sleep* 27, 661-666. 2004.

Heslop, P., Smith, G., Metcalfe, C., Macleod, J., Hart, C. Sleep duration and mortality: The effect of short or long sleep duration on cardiovascular and all-cause mortality in working men and women. *Sleep Med* 3, 305-314. 2002.

Hoevenaar-Blom, M., Spijkerman, A., Kromhout, D., van den Berg, J., Verschuren, M. Sleep duration and sleep quality in relation to 12-year cardiovascular disease incidence: The MORGEN Study. *Sleep* 34, 1487-1492. 2011.

Horowitz, J., Goodman, J. A longitudinal study of maternal depression symptoms. *Res and Theory for Nurs Prac: An Int J* 18, 149-163. 2004.

- Huang, W., Ramsey, K., Marcheva, B., Bass, J. Circadian rhythms, sleep and metabolism. *J Clin Invest* 121, 2133-2141. 2011.
- Hublin, C., Partinen, M., Koskenvuo, M., Kaprio, J. Sleep and mortality: a population-based 22-year follow up study. *Sleep* 30 (10), 1245-53. 2007
- Ikehara, S., Iso, H., Date, C., Kikuchi, S., Watanabe, Y., Wada, Y., Inaba, Y., Tamakoshi, A. Association of sleep duration with mortality from cardiovascular disease and other causes for Japanese men and women: JACC Study Group. *Sleep* 32, 295-301. 2009.
- International Paediatric Work Group on Arousals. The scoring of arousals in healthy term infants (between the ages of 1 and 6 months). *J Sleep Res* 14, 37-41. 2005.
- Ishizuka, Y., Pollak, C., Shirakawa, S. Sleep spindle frequency changes during the menstrual cycle. *J of Sleep Res* 3, 26-29. 1994.
- Ito, H., Nakasuga, K., Ohshima, A., Maruyama, T., Kaki, Y., Harada, M. Detection of cardiovascular risk factors by indices of obesity obtained from anthropometry and dual-energy X-ray absorptiometry in Japanese individuals. *Int J Obes* 27, 232-237. 2003.
- Jaksic, N., Ivezić, E., Jokic-Begik, N., Sranyi, Z., Stojanovic-Spehar, S. Factorial and Diagnostic Validity of the Becks Depression Inventory II (BDI-II) in Croatian Primary Healthcare. *J Clin Psychol Med Settings* epub ahead of print April 3, 2013
- Janssen, I. Influence of age on the relation between waist circumference and cardiometabolic risk markers. *Nutr Metab Cardiovasc Dis* 19, 163-169. 2009.
- Johnstone, S., Boyce, P., Hickey, A., Morris-Yatees, A., Harris, M. Obstetric risk factors for postnatal depression in urban and rural community samples. *Aus and NZ J of Psych* 35, 69-74. 2001.

- Jung, C., Melanson, E., Frydendall, E., Perreault, L., Eckel, R., Wright, K. Energy expenditure during sleep, sleep deprivation and sleep following sleep deprivation in adult humans. *J Physiol* 589, 235-244. 2011.
- Karter, A., Rowell, S., Ackerson, L. Excess maternal transmission of Type 2 Diabetes. *Diabetes Care* 22, 938-943. 1999.
- Kato, M., Phillips, B., Sigurdsson, G., Narkiewicz, K., Pesek, C., Somers, V. Effects of sleep deprivation on neural circulatory control. *Hypertension* 35, 1173-5. 2000.
- Kattapong, R., Fogg, L., Eastman, C. Effects of sex, menstrual cycle phase, and oral contraceptive use on circadian temperature rhythms. *Chronobiol Int* 12, 257-66. 1995.
- Killgore, W. Effects of sleep deprivation on cognition. *Prog in Brain Res.* 185, 105-129. 2010.
- Kim, C., Liu, T., Valdez, R., Beckles, G. Does frank diabetes in first-degree relatives of a pregnant woman affect the likelihood of her developing gestational diabetes mellitus or nongestational diabetes? *Am J Obstet Gynecol* 201, 576. 2009.
- Klein, D., Schatzberg, A., McCullough, J., Dowling, F., Goodman, D., Howland, R., Markowitz, J., Smith, C., Thase, M., Rush, A., LaVange, L., Harrison, W., Keller, M. Age of onset in chronic major depression: relation to demographic and clinical variables, family history, and treatment response. *J Affect Disord* 55, 149-157. 1999.
- Knab, B., Engel, R. Perception of waking and sleeping: possible implications for the evaluation of insomnia. *Sleep* 11, 265-272. 1988.
- Knutson, K., Spiegel, K., Penev, P., Van Cauter, E. The metabolic consequences of sleep deprivation. *Sleep Med Rev* 11, 163-178. 2007.
- Knutson, K., Van Cauter, E. Associations between sleep loss and increased risk of obesity and diabetes. *Ann NY Acad Sci* 1129, 287-304. 2008.

Knutson, K., Van Cauter, E., Rathouz, P., DeLeire, T., Lauderdale, D. Trends in the prevalence of short sleepers in the USA: 1975-2006. *Sleep* 33, 37-45. 2010.

Koban, M., Stewart, C. Effects of age on recovery of body weight following REM sleep deprivation of rats. *Physiol Behav* 87, 1-6. 2006.

Kohatsu, N., Tsai, R., Young, T., Vangilder, R., Burmeister, L., Stromquist, A., Merchant, J. Sleep duration and body mass index in a rural population. *Arch Intern Med* 166, 1701-1705. 2006.

Kojima, M., Hosoda, H., Matsuo, H., Nakazato, M., Matsuo, H., Kangawa, K. Ghrelin is a growth-hormonereleasing acylated peptide from the stomach. *Nature* 402, 656-660. 1999.

Kripke, D., Garfinkel, L., Wingard, D., Klauber, M., Marler, M. Mortality associated with sleep duration and insomnia. *Arch Gen Psych* 59, 131-136. 2002.

Kripke, D., Simons, R., Garfinkel, L., Hammond, E. Short and long sleep and sleeping pills: is increased mortality associated? *Arch Gen Psychiat* 36, 103-16. 1979.

Kyrkouli, S., Stanley, B., Leibowitz, S. Galanin: stimulation of feeding induced by medial hypothalamic injection of this novel peptide. *Eur J Pharmacol* 122, 159-60. 1986.

Lampl, M., Johnson, M. Infant growth in length follows prolonged sleep and increased naps. *Sleep* 34, 641-650. 2011

Lauderdale, D., Knutson, K., Rathouz, P., Yan, L., Hulley, S., Liu, K. Cross-sectional and longitudinal associations between objectively measured sleep duration and body mass index: the CARDIA Sleep Study. *Am J Epidemiol* 170, 805-813. 2009.

Léger, D., Bayon, V. Societal costs of insomnia. *Sleep Med Rev.* 14, 379-389. 2010.

Leu-Semenescu, S., Arnulf, I., Decaix, C., Moussa, F., Clot, F., Boniol, C., Touitou, Y., Levy, R., Vidailheit, M., Roze, E. Sleep and rhythm consequence of a genetically induced loss of serotonin. *Sleep* 33, 307-314. 2010.

- Lieb, R., Isensee, B., Hofler, M., Wittchen, H. Parental depression and depression in offspring: evidence for familial characteristics and subtypes? *J Psychiatr Res* 36, 237-246. 2002.
- Lim ,J., Dinges, D. A meta-analysis of the impact of short-term sleep deprivation on cognitive variables. *Psychol Bull.* 136, 375-389. 2010.
- Lin, A, Koltai, P. Sleep endoscopy in the evaluation of pediatric obstructive sleep apnea. *Int J Ped* 19, 576719-576725. 2012
- Locard, E., Mamelie, N., Billette, A., Miginiac, M., Munoz, F., Rey, S. Risk factors of obesity in a five year old population. Parental versus environmental factors. *Int J of Obe and Rel Met Dis* 16, 721-729. 1992.
- Logsdon, M., Wisner, K., Pinto-Foltz, M. The impact of postpartum depression on mothering. *J of Obs, Gyn, and Neonatal Nurs: JOGNN* 35, 652-658. 2006.
- Lu, X., Lundström, L., Langel, U., Bartfai, T. Distribution and differential regulation of galanin receptor subtypes in rat brain: effects of seizure activity. *Neuropeptides* 39, 147-52. 2005.
- Luckhaupt, S., SangWoo, T., Calvert, G. The prevalence of short sleep duration by industry and occupation in the national health interview survey. *Sleep* 33, 149-59. 2010.
- Lugaresi, E., Cirignotta, F., Zucconi, M., Mondini, S., Lenzi, P., Coccagna, G. Good and poor sleepers: an epidemiological survey of the San Marino population. In *Guilleminault C, Lugaresi E eds. Sleep/wake disorders: natural history, epidemiology and long-term evolution.* New York: Raven Press 13-28. 1983
- Luyster, F.,Strollo, P.,Walsh, J. Sleep: A health Imperative. *Sleep* 35, 727-734. 2012

Maes, M., Bosmans, E., De Jongh, R. Increased serum IL-6 and IL-1 receptor antagonist concentrations in major depression and treatment resistant depression. *Cytokine* 9, 853-8. 1997.

Manber, R., Bootzin, R. Sleep and the menstrual cycle. *Health Psychology* 16, 209-214. 1997.

Manconi, M., Ulfberg, J., Berger, K., Ghorayeb, I., Wesström, J., Fulda, S., Allen, R., Pollmächer, T. When gender matters: restless legs syndrome. Report of the "RLS and woman" workshop endorsed by the European RLS Study Group. *Sleep Med Rev* 16, 297-307. 2012.

Mander, B., Colecchia, E., Spiegel, K., Kim, R., Sannar, E., Van Cauter, E. Short sleep: A risk factor for insulin resistance and obesity. *Diabetes* 50, A45. 2001.

Mayberry, L., Horowitz, J., Declercq, E. Depression symptom prevalence and demographic risk factors among U.S. women during the first 2 years postpartum. *J Obstet Gynecol Neonatal Nurs* 36, 542-549. 2007.

Mazure, C., Keita, G., Blehar, M. Summit on women and depression: Proceedings and Recommendations. 2002 Available from: <http://www.apa.org/pi/wpo/women&depression.pdf>

McGregor, G., Desaga, J., Ehlenz, K. Radioimmunologic measurement of leptin in plasma of obese and diabetic human subjects. *Endocrinology* 137, 1501-04. 1996.

Meigs, J., Cupples, A., Wilson, P. Parental transmission of Type 2 Diabetes: the Framingham Offspring Study. *Diabetes* 49, 2201-2207. 2000.

Meier-Ewert, H., Ridker, P., Rifai, N. Effect of sleep loss on C-reactive protein, an inflammatory marker of cardiovascular risk. *J Am Coll Cardiol* 43, 678-83. 2004.

Miller, V., Duckles, S. Vascular actions of estrogens: Functional implications. *Pharmacol Rev* 60, 210-241. 2008.

- Milligan, R., Lenz, E., Parks, P., Pugh, L., Kitzman, H. Postpartum fatigue: clarifying a concept. *Sch Inq Nurs Pract* 10, 279-291. 1996.
- Mindell, J., Owens, J., Carskadon, M. Developmental features of sleep. *Child Adolesc Psychiatr Clin N Am* 8, 695-725. 1995.
- Mindell, J. Empirically supported treatments in pediatric psychology: bedtime refusal and night wakings in young children. *J Pediatr Psychol* 24, 465-481. 1999.
- Mitchell, B., Kammerer, C., Reinhart, L., Stern, M., Maccluer, J. Is there an excess in maternal transmission of NIDDM? *Diabetologia* 38, 314-7. 1995.
- Moline, M., Broch, L., Zak, R. Sleep in women across the life cycle from adulthood through menopause. *Med Clin N Am* 88, 705-736. 2004.
- Monasta, L., Batty, G., Cattaneo, A. Early-life determinants of overweight and obesity: a review of systematic reviews. *Obes Rev* 11, 695-708. 2010.
- Mong, J., Baker, F., Mahoney, M., Paul, K., Schwartz, M., Semba, K., Silver, R. Sleep, rhythms and the endocrine brain: Influence of sex and gonadal hormones. *J Neurosci* 31, 16107-16116. 2011
- Montemitro, E., Franco, P., Scaillet, S., Kato, I., Groswasser, J., Villa, M.P., Kahn, A., Sastre, J.P., Ecochard, R., Thiriez, G., Lin, J. Maturation of spontaneous arousals in healthy infants. *Sleep* 31, 47-55. 2008.
- Murck, H., Held, K., Ziegenbein, M., Künzel, H., Holsboer, F., Steiger, A. Intravenous administration of the neuropeptide galanin has fast antidepressant efficacy and affects the sleep EEG. *Psychoneuroendocrinology* 29, 1205-11. 2004.
- Newgard, C., Matschinsky, F. Substrate control of insulin release. In *Handbook of Physiology*, Jefferson J eds, UK: Oxford Univ Press 125-152. 2001.

Nguyen, A., Baltzan, M., Small, D. Clinical reproducibility of the Epworth Sleepiness Scale. *J Clin Sleep Med* 2, 170-4. 2006.

Oinonen, K., Mazmanian, D. To what extent do oral contraceptives influence mood and affect? *J of Affect Disord* 40, 229-240. 2002.

Omisade, A., Buxton, O., Rusak, B. Impact of acute sleep restriction on cortisol and leptin levels in young women. *Physiol Behav* 99, 651-656. 2010.

Østbye, T., Krause, K., Lovelady, C., Morey, M., Bastian, L., Peterson, B., Swamy, G., Brouwer, R., McBride, C. Active Mothers Postpartum: A Randomized Controlled Weight-Loss Intervention Trial. *Am J Prev Med* 37, 173-180. 2009.

Patel, S., Ayas, N., Malhotra, M., White, D., Schernhammer, E., Speizer, F., Stampfer, M., Hu, F. A prospective study of sleep duration and mortality risk in women. *Sleep* 27, 440-4. 2004.

Patel, S., Hu, F. Short sleep duration and weight gain: a systematic review. *Obes* 16, 643-653. 2008.

Paredes, S., Barriga, C., Reiter, R., Rodríguez, A. Assessment of the Potential Role of Tryptophan as the Precursor of Serotonin and Melatonin for the Aged Sleep-wake Cycle and Immune Function: *Streptopelia risoria* as a Model. *Int J Tryptophan Res* 2, 23-36. 2009.

Pavlova, M., Sheikh, L. Sleep in women. *Semin Neurol* 31, 397-403. 2011.

Pearl, P. Childhood sleep disorders: diagnostic and therapeutic approaches. *Curr Neurol Neurosci Rep* 2, 150-157. 2002.

Pedrazzoli, M., Bedito, M. Rapid eye movement sleep deprivation-induced down-regulation of beta-adrenergic receptors in the rat brainstem and hippocampus. *Pharmacol Biochem Behav* 79, 31-6. 2004.

Pelleymounter, M., Cullen, M., Baker, M. Effects of the obese gene product on body weight regulation in ob/ob mice. *Science* 269, 540-543. 1995.

- Pfeifer, M., Halter, J., Porte, D. Jr. Insulin secretion in diabetes mellitus. *Am J Med* 70, 579-588. 1981.
- Pitanga, F., Lessa, I. Anthropometric indicators of obesity as a screening tool for coronary heart risk in adults in the city of Salvador (Bahia). *Arq Bras Cardiol* 85, 26-31. 2005.
- Poe, G., Walsh, S., Bjorness, T. Cognitive neuroscience of sleep. *Prog in Brain Res* 185,1-19. 2010.
- Placidi, G., Oquendo, M., Malone, K., Huang, Y., Ellis, S., Mann, J. Aggressivity, suicide attempts, and depression: relationship to cerebrospinal fluid monoamine metabolite levels. *Biol Psychiatry* 50, 783-791. 2001.
- Reddy, A., O'Neill, J. Healthy clocks, healthy body, healthy mind. *Trends Cell Biol* 20, 36-44. 2010.
- Reijneveld, S., Wal, M., Van Der Brugman, E., Sing, R., Verloove-Vanhorick, S. Infant crying and abuse. *Lancet* 364, 1340-1342. 2004.
- Reiter, R., Richardson, B. Some perturbations that disturb the circadian melatonin rhythm. *Chronobiol Intl* 9, 314-21. 1992.
- Richardson, H., Walker, A., Horne, R. Sleeping Like a Baby—Does Gender Influence Infant Arousability? *Sleep* 33, 1055-1060. 2010.
- Robinson, O., Sahakian, B. Acute tryptophan depletion evokes negative mood in healthy females who have previously experienced concurrent negative mood and tryptophan depletion. *Psychopharmacol* 205, 227-235. 2009.
- Roman, V., Van der Borght, K., Leemburg, S., Van der Zee, E., Meerlo, P. Sleep restriction by forced activity reduces hippocampal cell proliferation. *Brain Res* 1065, 53-9. 2005.

Romanov, K., Varjonen, J., Kaprio, J., Koskenvuo, M. Life events and depressiveness-the effect of adjustment for psychosocial factors, somatic health and genetic liability. *Acta Psychiatr Scand* 107, 25-33. 2003.

Rosenbaum, M., Leibel, R., Hirsch, J. Obesity. *N Engl J Med* 2, 396-407. 1997.

Rothberg, B., Magriples, U., Kershaw, T., Rising, S., Ickovics, J. Gestational weight gain and post-partum weight loss among young, low-income, ethnic minority women. *Am J Obstet Gynecol* 204, 52.e1-52.e11. 2011.

Sabel, K., Lundqvist-Persson, C., Bona, E., Petzold, M., and Strandvik, B. Fatty acid patterns early after premature birth, simultaneously analysed in mothers' food, breast milk and serum phospholipids of mothers and infants. *Lipids Health Dis* 8, 20. 2009.

Sadeh, A., Tikotzky, L., Scher, A. Parenting and infant sleep. *Sleep Med Reviews* 14, 89-96. 2010.

Scheer, F., Hilton, M., Mantzoros, C., Shea, S. Adverse metabolic and cardiovascular consequences of circadian misalignment. *Proc Natl Acad Sci USA* 106, 4453-4458. 2009.

Schwartz, J., Roth, T. Neurophysiology of Sleep and Wakefulness: Basic Science and Clinical Implications. *Curr Neuropsychopharmacol* 6, 367-378. 2008.

Sekine, M., Yamagami, T., Hamanishi, S. Parental obesity, lifestyle factors and obesity in preschool children: results of the Toyama Birth Cohort Study. *J of Epidemiol* 12, 33-39. 2002.

Shephard, R. Epidemiological indices, anthropometric and cadaver estimates of body composition. Body composition in biological anthropology. Cambridge Studies in Biological and Evolutionary Anthropology. *Cambridge University Press* 6, 24-25. 1991.

Siege-Riz, A., Herring, A., Carrier, K., Evenson, K., Dole, N., Deierlein, A. Sociodemographic, perinatal, behavioural, and psychosocial predictors of weight retention at 3 and 12 months postpartum. *Obes* 18, 1996-2003.2010.

Small, R., Brown, S., Lumley, J., Ashbury, J. Missing voices: What women say and do about depression after childbirth. *J of Rep and Infant Psych* 12, 89-103. 1994.

Snijder, M., Zimmet, P., Visser, M., Dekker, J., Seidell, J., Shaw, J. Independent and opposite associations of waist and hip circumferences with diabetes, hypertension and dyslipidemia: the AusDiab Study. *Int J Obes Relat Metab Disord* 28, 402-409. 2004.

Spiegel, K., Leproult, R., L'Hermite-Baleriaux, M., Copinschi, G., Penev, P.D., Van Cauter, E. Leptin levels are dependent on sleep duration: relationships with sympathovagal balance, carbohydrate regulation, cortisol and thyrotropin. *J Clin Endocrinol Metab* 89, 5762-71. 2004.

Spiegel, K., Leproult, R., Tasali, E., Penev, P., Van Cauter, E. Sleep curtailment results in decreased leptin levels and increased hunger and appetite. *Sleep* 26, A174. 2003.

Spiegel, K., Leproult, R., Van Cauter, E. Impact of sleep debt on metabolic and endocrine function. *Lancet* 354, 1435-9. 1999.

Spruyt, K., Aitken, R., So, K., Charlton, M., Adamson, T., Horne, R. Relationship between sleep/wake patterns, temperament and overall development in term infants over the first year of life. *Early Hum Dev* 84, 289-296. 2008.

Stuebe, A., Mantzoros, C., Kleinman, K., Gillman, M., Rifas-Shiman, S., Gunderson, E., Rich-Edwards, J. Duration of lactation and maternal adipokines at 3 years postpartum. *Diab* 60, 1277-1285. 2011.

- Sullivan, P., Neale, M., Kendler, K. Genetic epidemiology of major depression: review and metaanalysis. *Am J Psychiatry* 157, 1552-1562. 2000.
- Tadavarty, R., Rajput, P., Wong, J., Kumar, U., Sastry, B. Sleep-deprivation induces changes in GABA (B) and mGlu receptor expression and has consequences for synaptic long-term depression. *PLoS One* 6, e24933. 2011.
- Taheri, S., Lin, L., Austin, D., Young, T., Mignot, E. Short sleep duration is associated with reduced leptin, elevated ghrelin and increased body mass. *PLoS Med*, 1:e62. 2004.
- Tallett, A., Blundell, J., Rodgers, R. Sibutramine-induced anorexia: potent, dose-dependent and behaviourally-selective profile in male rats. *Behav Brain Res* 198, 359-65. 2009.
- Tamakoshi, A., Ohno, Y. Self-reported sleep duration as a predictor of all-cause mortality: results from the JACC. JACC Study Group. *Sleep* 27, 51-54. 2004.
- Taveras, E., Rifas-Shiman, S., Oken, E., Gunderson, E., Gillman, M. Short sleep duration in infancy and risk of childhood overweight. *Archives of Pediatrics and Adolescent Medicine* 162, 305-311. 2008.
- Taveras, E., Rifas-Shiman, S., Oken, E., Rich-Edwards, J., Gunderson, E., Stuebe, A., Mantzoros, C. Association of maternal short sleep duration with adiposity and cardiometabolic status at 3 years postpartum. *Obes* 19, 171-178. 2011.
- Thompson, D., Christakis, D. The association between television viewing and irregular sleep schedules among children less than 3 years of age. *Pediatrics* 116, 851-856. 2005.
- Tian, Z., Ye, T., Zhang, X. Sleep duration and hyperglycemia among obese and nonobese children aged 3 to 6 years. *Arch of Ped and Adol Med* 164, 46-52. 2010.
- Tikotzky, L., De Marcas, G., Har-Toov, J., Dollberg, S., Bar-Haim, S., Sadeh, A. Sleep and physical growth in infants during the first 6 months. *J of Sleep Res* 19, 103-110. 2010.

- Tizard, J., Tizard, B. Social development of two-year-old children. *In The Origins of Human Social Relations*. Schaffer H. eds. London: Academic Press. 1972.
- Tochikubo, O., Ikeda, A., Miyajima, E., Ishii, M. Effects of insufficient sleep on blood pressure monitored by a new multibiomedical recorder. *Hypertension* 27, 1318-24. 1996.
- Touchette, E., Petit, D., Tremblay, R., Montplaisir, J. Risk factors and consequences of early childhood dyssomnias: new perspectives. *Sleep Med Rev* 13, 355-61. 2009.
- Van Cauter, E., Knutson, K. Sleep and the epidemic of obesity in children and adults. *Eur J Endocrinol* 159, S59-66. 2008.
- Van Cauter, E., Spiegel, K., Tasali, E., Leproult, R. Metabolic consequences of sleep and sleep loss. *Sleep Med* 9 Suppl 1, S23-8. 2008.
- Van Dongen, H., Maislin, G., Mullington, J., Dinges, D. The cumulative cost of additional wakefulness: dose-response effects on neurobehavioral functions and sleep physiology from chronic sleep restriction and total sleep deprivation. *Sleep* 26, 117-126. 2003.
- Vettor, R., Milan, G., Rossato, M., Federspil, G. Review article: adipocytokines and insulin resistance. *Aliment Pharmacol Ther* 22, 3-10. 2005.
- Von Kries, R., Toschke, A., Wurmser, H., Sauerwald, T., Koletzko, B. Reduced risk for overweight and obesity in 5-and 6-y-old children by duration of sleep—a cross-sectional study. *Int J of Obesity and Related Met Disorders* 26, 710-716. 2002.
- Waldhauser, F., Saletu, B., Trinchard-Lugan, I. Sleep laboratory investigations on hypnotic properties of melatonin. *Psychopharmacology (Berl)* 100, 222-6. 1990.
- Wang, Y. Epidemiology of childhood obesity - Methodological aspects and guidelines: What is new? *Int J of Obesity* 28, S21-S28. 2004.

Watson, N., Buchwald, D., Vitiello, M., Noonan, C., Goldberg, J. A twin study of sleep duration and body mass index. *J Clin Sleep Med* 6, 11-17. 2010.

Webley, G., Liedenberger, F. The circadian pattern of melatonin and its positive relationship with progesterone in women. *J Clin Endocrin Metab* 63, 323-8. 1986.

Website reference as of 25 Dec 2012:

http://healyourselfathome.com/HOW/NEWTARTS/7_REST/stages_of_sleep.aspx

Weerarathna, T., Lekamwasam, S., Rodrigo, M. Prediction of total and visceral fat contents using anthropometric measures of adiposity in women. *Ceylon Med J* 53, 128-32. 2008.

Weissman, M., Wickramaratne, P., Nomura, Y., Warner, V., Verdelli, H., Pilowsky, D., Grillon, C., Bruder, G. Families at high and low risk for depression: a 3- generation study. *Arch Gen Psychiatry* 63, 29-36. 2005.

Weissman, A., Levy, B., Hartz, A. Pooled analysis of antidepressant levels in lactating mothers, breast milk, and nursing infants. *Am J Psychiatry* 161, 1066-1078. 2004.

Wisner, K., Hanusa, B., Perel, J. Postpartum depression. A randomized trial of sertraline versus nortriptyline. *J Clin Psychopharmacol* 26, 353-360. 2006.

Yaggi, H., Araujo, A., McKinlay, J. Sleep duration as a risk factor for the development of Type 2 Diabetes. *Diabetes Care* 29, 657-61. 2006.

Yatham, L., Liddle, P., Shiah, I. Effects of rapid tryptophan depletion on brain 5-HT(2) receptors: a PET study. *Br J Psychiatry* 178, 448-53. 2001.

Young, E., Kornstein, S., Harvey, A., Wisniewski, S., Barkin, J., Fava, M., Trivedi, M., Rush, A. Influences of hormone based contraception on depressive symptoms in premenopausal women with major depression. *Psychoneuroendocrinology* 32, 843-853. 2007.

Yu, L., Buysse, D., Germain, A., Moul, D., Stover, A., Dodds, N., Johnston, K., Pilkonis, P.
Development of short forms from the PROMIS™ sleep disturbance and Sleep-Related
Impairment item banks. *Behav Sleep Med* 10, 6-24, 2011.

APPENDICES

APPENDIX A - Subject Information Sheet

APPENDIX B - Consent Form

APPENDIX C1 - Subject Interview Page 1

APPENDIX C2 - Subject Interview Page 2

APPENDIX D - Becks Depression Inventory II

APPENDIX E - Pittsburg Sleep Quality Index

APPENDIX A - Subject Information Sheet



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"The impact of long-term, partial sleep deprivation in unipara mothers"

Subject Information Sheet

Dear New Mom

My name is Terri Marks and I am currently registered for Masters in Science at the University of the Witwatersrand. I am working with Dr Alison Bentley and Dr Kennedy Erlwanger on a research project entitled: "The Impact of Long-Term, Partial Sleep Deprivation in Mothers". There is mounting evidence that disturbed sleep may cause abnormalities in metabolism, namely the ability of the body to handle glucose efficiently, stress hormone release and body weight. One of the causes of disturbed sleep is a baby waking up frequently during the night, especially when this has been persisting for more than 6 months. There is not much research done on this topic. The more we understand about how our bodies react to a normal stress such as sleep deprivation due to a new baby, the better prepared we can be to tackle problems such as returning to work after maternity leave.

We would like to please invite you to participate in this study conducted through the Wits-Dial-A-Bed Sleep Laboratory at Wits Medical School. The study involves collecting data on mothers by filling in a questionnaire consisting of an interview, a Becks Depression Inventory and a Pittsburgh Sleep Quality Index and some basic body measurements. The questionnaire takes about 10 minutes to complete. The interview will ask about some of your family history, your pregnancy history and your child's sleeping patterns. The body measurements will include, weight, height, waist, hip and arm circumference and skin fold thickness taken from the mid-upper arm.

Important Information

- Participation in this study is voluntary and you are allowed to withdraw at any time without consequence
- If you choose to withdraw, your decision will not impact any further treatment by members of the Sleep Laboratory
- Your personal details will be given a code number to ensure total anonymity and confidentiality, only I will have access to your individual results. All the data from the study is published as group results.
- I have obtained approval for my study from the Committee for Research on Human Subjects of the University of the Witwatersrand

If you would like to participate in the study please could you confirm this by signing the consent form overleaf. Thank you for your time and interest in this project.

If you have any concerns or questions, please feel free to contact me at any time.

Terri Marks

Cell: 0727055610

terri.marks@gmail.com

APPENDIX B - Consent Form



UNIVERSITY OF THE WITWATERSRAND

SCHOOL OF PHYSIOLOGY



CONSENT TO TAKE PART IN A RESEARCH PROJECT

Subject Number: 2007/07/21_10

1. I, being 18 years or older, consent to participating in a research project entitled: "The Impact of Long-Term, Partial Sleep Deprivation in Unipara Mothers".
2. I understand that the interview forms part of a research project, and may not provide any direct benefit to me.
3. The procedures have been explained to me and I understand and appreciate their purpose, any risks involved, and the extent of my involvement. I have read and understand the attached information sheet.
4. I understand that all records will be coded and kept confidential to ensure anonymity and that the study has been sanctioned by the Committee for Research on Human Subjects, University of the Witwatersrand, Johannesburg.
5. I understand that my participation is voluntary, and that I am free to withdraw from the project at any time without prejudice.

Subject signature.....

Date.....

I have fully described the interview process and have explained the purpose. I have asked whether or not any questions have arisen regarding the interview and have answered these questions to the best of my ability.

Investigator signature.....

Date.....



"The impact of long-term, partial sleep deprivation on mother and child"

Subject Interview

Mother's Information

Date _____



Contact Details

Name _____

Tel (cell) _____

Postal Address (inc code) _____

Physical Address _____

Employer _____

Subject Number: _____

2007/07/21_10

Analysis

PSQI Score _____

BDI Score _____

Race _____

Height _____

Current Weight _____

BMI _____

Waist Circumference _____

Hip Circumference _____

Waist-Hip Ratio _____

Tricep skin fold _____

Mid-arm circumference _____

AMA score _____

History

Family history of diabetes _____

Family history of depression _____

Prepregnancy weight _____

Post partum weight (6 week check-up) _____

Pregnancy complications (eg. Gestational diabetes)? _____

Record of urine glucose during pregnancy _____

Are you on an oral contraceptive? Yes \ No

If so which one? _____

Are you on other medications? Yes / No

If so, what? _____

<u>Child's name</u>	<u>Date of Birth</u>	<u>Sex</u>	<u>Weight at birth</u>

Current perceived sleep schedule:

Daytime			Nighttime		
Time	Duration	Description	Time	Duration	Description

Sleeping arrangements (eg. Bed sharing, pram, etc) _____

Assistance (e.g. mother, night nurse, family) _____

Do you think your child has a sleep problem? Yes \ No

Why? _____

Name: _____ Subject Number: 2007/07/21_10 _____ Marital Status: _____ Age: _____ Sex: _____
Occupation: _____ Education: _____

1. Sadness

- 0 I do not feel sad.
1 I feel sad much of the time.
2 I am sad all the time.
3 I am so sad or unhappy that I can't stand it.

2. Pessimism

- 0 I am not discouraged about my future.
- 1 I feel more discouraged about my future than I used to be.
- 2 I do not expect things to work out for me.
- 3 I feel my future is hopeless and will only get worse.

3. Past Failure

- 0 I do not feel like a failure.
1 I have failed more than I should have.
2 As I look back, I see a lot of failures.
3 I feel I am a total failure as a person.

4. Loss of Pleasure

- 0 I get as much pleasure as I ever did from the things I enjoy.
- 1 I don't enjoy things as much as I used to.
- 2 I get very little pleasure from the things I used to enjoy.
- 3 I can't get any pleasure from the things I used to enjoy.

5. Guilty Feelings

- 0 I don't feel particularly guilty.
1 I feel guilty over many things I have done or
should have done.
2 I feel quite guilty most of the time.
3 I feel guilty all of the time.

6. Punishment Feelings

- 0 I don't feel I am being punished.
1 I feel I may be punished.
2 I expect to be punished.
3 I feel I am being punished.

7. Self-Dislike

- 0 I feel the same about myself as ever.
1 I have lost confidence in myself.
2 I am disappointed in myself.
3 I dislike myself.

8. Self-Criticalness

- 0 I don't criticize or blame myself more than usual.
1 I am more critical of myself than I used to be.
2 I criticize myself for all of my faults.
3 I blame myself for everything bad that happens.

9. Suicidal Thoughts or Wishes

- 0 I don't have any thoughts of killing myself.
1 I have thoughts of killing myself, but I would
not carry them out.
2 I would like to kill myself.
3 I would kill myself if I had the chance.

10. Crying

- 0 I don't cry anymore than I used to.
1 I cry more than I used to.
2 I cry over every little thing.
3 I feel like crying, but I can't.

Subtotal Page 1

Continued on Back

11. Agitation

- 0 I am no more restless or wound up than usual.
- 1 I feel more restless or wound up than usual.
- 2 I am so restless or agitated that it's hard to stay still.
- 3 I am so restless or agitated that I have to keep moving or doing something.

12. Loss of Interest

- 0 I have not lost interest in other people or activities.
- 1 I am less interested in other people or things than before.
- 2 I have lost most of my interest in other people or things.
- 3 It's hard to get interested in anything.

13. Indecisiveness

- 0 I make decisions about as well as ever.
- 1 I find it more difficult to make decisions than usual.
- 2 I have much greater difficulty in making decisions than I used to.
- 3 I have trouble making any decisions.

14. Worthlessness

- 0 I do not feel I am worthless.
- 1 I don't consider myself as worthwhile and useful as I used to.
- 2 I feel more worthless as compared to other people.
- 3 I feel utterly worthless.

15. Loss of Energy

- 0 I have as much energy as ever.
- 1 I have less energy than I used to have.
- 2 I don't have enough energy to do very much.
- 3 I don't have enough energy to do anything.

16. Changes in Sleeping Pattern

- 0 I have not experienced any change in my sleeping pattern.
- 1a I sleep somewhat more than usual.
- 1b I sleep somewhat less than usual.
- 2a I sleep a lot more than usual.
- 2b I sleep a lot less than usual.
- 3a I sleep most of the day.
- 3b I wake up 1-2 hours early and can't get back to sleep.

17. Irritability

- 0 I am no more irritable than usual.
- 1 I am more irritable than usual.
- 2 I am much more irritable than usual.
- 3 I am irritable all the time.

18. Changes in Appetite

- 0 I have not experienced any change in my appetite.
- 1a My appetite is somewhat less than usual.
- 1b My appetite is somewhat greater than usual.
- 2a My appetite is much less than before.
- 2b My appetite is much greater than usual.
- 3a I have no appetite at all.
- 3b I crave food all the time.

19. Concentration Difficulty

- 0 I can concentrate as well as ever.
- 1 I can't concentrate as well as usual.
- 2 It's hard to keep my mind on anything for very long.
- 3 I find I can't concentrate on anything.

20. Tiredness or Fatigue

- 0 I am no more tired or fatigued than usual.
- 1 I get more tired or fatigued more easily than usual.
- 2 I am too tired or fatigued to do a lot of the things I used to do.
- 3 I am too tired or fatigued to do most of the things I used to do.

21. Loss of Interest in Sex

- 0 I have not noticed any recent change in my interest in sex.
- 1 I am less interested in sex than I used to be.
- 2 I am much less interested in sex now.
- 3 I have lost interest in sex completely.

Subtotal Page 2

Subtotal Page 1

Total Score

The Pittsburgh Sleep Quality Index (PSQI)

Instructions: The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions. During the past month,

1. When have you usually gone to bed? _____
2. How long (in minutes) has it taken you to fall asleep each night? _____
3. When have you usually gotten up in the morning? _____
4. How many hours of actual sleep do you get at night? (This may be different than the number of hours you spend in bed) _____

5. During the past month, how often have you had trouble sleeping because you...	Not during the past month (0)	Less than once a week (1)	Once or twice a week (2)	Three or more times a week (3)
a. Cannot get to sleep within 30 minutes				
b. Wake up in the middle of the night or early morning				
c. Have to get up to use the bathroom				
d. Cannot breathe comfortably				
e. Cough or snore loudly				
f. Feel too cold				
g. Feel too hot				
h. Have bad dreams				
i. Have pain				
j. Other reason(s), please describe, including how often you have had trouble sleeping because of this reason(s):				
6. During the past month, how often have you taken medicine (prescribed or "over the counter") to help you sleep?				
7. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?				
8. During the past month, how much of a problem has it been for you to keep up enthusiasm to get things done?				
	Very good (0)	Fairly good (1)	Fairly bad (2)	Very bad (3)
9. During the past month, how would you rate your sleep quality overall?				

Component 1	#9 Score	C1
Component 2	#2 Score (≤ 15 min=0; 16-30 min=1; 31-60 min=2, >60 min=3) + #5a Score (if sum is equal 0=0; 1-2=1; 3-4=2; 5-6=3)	C2
Component 3	#4 Score ($>7=0$; 6-7=1; 5-6=2; $<5=3$)	C3
Component 4	(total # of hours asleep)/(total # of hours in bed) x 100 >85%=0, 75%-84%=1, 65%-74%=2, <65%=3	C4
Component 5	Sum of Scores #5b to #5j (0=0; 1-9=1; 10-18=2; 19-27=3)	C5
Component 6	#6 Score	C6
Component 7	#7 Score + #8 Score (0=0; 1-2=1; 3-4=2; 5-6=3)	C7

Add the seven component scores together _____ **Global PSQI Score** _____

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