The relationship between chronic sleep restriction, poor sleep quality and obesity in adults

Christopher Magee
University of Wollongong
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THE RELATIONSHIP BETWEEN CHRONIC SLEEP RESTRICTION, POOR SLEEP QUALITY AND OBESITY IN ADULTS

A THESIS SUBMITTED IN FULFILMENT OF THE REQUIREMENTS FOR THE AWARD OF THE DEGREE

DOCTOR OF PHILOSOPHY

FROM

UNIVERSITY OF WOLLONGONG

BY

CHRISTOPHER MAGEE, B. PSYC (HONS)

SCHOOL OF PSYCHOLOGY

2008
CERTIFICATION

I, Christopher Magee, declare that this thesis, submitted in fulfilment of the requirements for the award of Doctor of Philosophy, in the School of Psychology, University of Wollongong, is wholly my own work unless otherwise referenced or acknowledged. The document has not been submitted for qualifications at any other academic institution.

Christopher Magee

14 April 2008
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My supervisors, Dr Peter Caputi, Professor Don Iverson and Professor Xu-Feng Huang for their invaluable time, support and enthusiasm for this project.

Dr Nancy Humpel and Dr Stephen Palmisano for their advice and generous support along the way.

Sister Sheena McGhee for her technical advice and calming influence during the sleep restriction study.

All of the Research Assistants who assisted me in the overnight supervision of participants, allowing me nights of precious sleep.

All of the research participants from the University of Wollongong, Bluescope Steel and the University of the Third Age who participated in these studies. I am especially indebted to the 12 individuals who volunteered to participate in the sleep restriction study, which lasted a week and made them feel tired and hungry.

Finally, to my family and friends for always keeping things in perspective.
This thesis consists of two literature reviews followed by three empirical chapters that examined the relationship between chronic sleep restriction and obesity. Chapter 2 reviewed available research data and presented a theoretical model linking chronic sleep restriction to obesity. This model hypothesises that chronic sleep restriction contributes to obesity by altering energy regulatory hormones such as ghrelin and leptin. It was also argued that factors such as poor mental health, medication use and long work hours contribute to chronic sleep restriction at a population level, and could have implications for improving sleep. This model provides a sound theoretical framework, which was used to guide the subsequent empirical chapters. In chapter 3, the key methodological limitations of previous studies examining the relationship between chronic sleep restriction and obesity were outlined. Methodological recommendations for future research were then provided to facilitate a more complete understanding of how chronic sleep restriction and obesity are linked in the general population.

Chapter 4 tested a path model linking chronic sleep restriction to obesity in 325 adults aged 18 to 87 years, based on the theoretical framework provided in chapter 2 and the methodological recommendations listed in chapter 3. The results indicated that short sleep durations and age were associated with obesity, whilst age, uncomfortable sleep environments, irregular sleep/wake cycles and poor mental health were associated with short sleep durations. However, the results also identified potential environmental, behavioural and psychological determinants of chronic sleep restriction that could be targeted in the future treatment and prevention of obesity.
Chapter 5 examined the relationship between three dimensions of sleep quality as assessed by the Pittsburgh Sleep Quality Index and obesity in 262 adults aged 18 to 35 years. Short sleep durations and increased levels of daytime dysfunction (e.g., sleepiness) were associated with obesity, whilst irregular bedtimes, noisy environments, discomfort and depression were the major factors associated with poor sleep quality. These factors could play a role in obesity interventions that target sleeping patterns and need to be further investigated.

Finally, chapter 6 examined the effects of two nights of sleep restriction on energy expenditure and neuroendocrine hormones involved in energy balance regulation in ten healthy male adults. The results indicated that sleep restriction led to an increase in ghrelin and a reduction in PYY, which corresponded with increased hunger and reduced satiety. The results also suggested that energy expenditure declined with sleep restriction. These results suggest that sleep restriction could contribute to obesity by altering energy expenditure and the hormonal regulation of food intake.

The findings from this thesis therefore suggest that chronic sleep restriction contributes to the development of obesity by altering key pathways identified in chapter 2. The identification of possible determinants of chronic sleep restriction has potential applications for the treatment and prevention of obesity. For example, the factors identified in chapters 4 and 5 could be targeted as a way to promote healthy sleep durations, and could be effective in improving the efficacy of existing interventions for obesity.
PUBLICATIONS FROM THE THESIS

Published Manuscripts


Manuscripts under Review


Magee, C.A., Huang, X., Iverson, D.C., & Caputi, P. (Submitted). The Link between
Chronic Sleep Restriction and Obesity: A review of the underlying causal mechanisms. *Journal of Behavioral Medicine.*

**THE WORK PRESENTED IN THIS THESIS HAS UNDERGONE PEER REVIEW FROM THE FOLLOWING JOURNALS:**

- Preventive Medicine (2007)
- Public Health (2007)
- Sleep (2007)
- Sleep Medicine (2007)
DEFINITION OF KEY TERMS

BASAL METABOLIC RATE: The energy that is expended when an individual is at rest. It is recommended that this measurement is taken in the morning between 6:00am and 9:00am after a minimum of nine hours fasting (Levine, 2005). In this thesis, basal metabolic rate was estimated using indirect calorimetry, which is also defined in this list.

CHRONIC SLEEP RESTRICTION: Habitual sleep durations that are less than seven hours but more than four hours per night (Dinges, Rogers, & Baynard, 2005). This is in contrast to total sleep restriction or total sleep deprivation, which refers to a complete absence of sleep over a period of at least 24 hours.

INDIRECT CALORIMETRY: A method to estimate energy expenditure which involves the measurement of oxygen consumption and carbon dioxide production (Levine, 2005). In the present thesis, a whole room calorimeter was used to obtain these measurements.

OVERWEIGHT AND OBESITY: An excess of body fat (particularly visceral fat) that is associated with increased mortality risk. Most relevant literature has defined overweight by a body mass index (BMI) between 25.0 and 29.9, with obesity defined by a BMI of 30.0 and over. In this thesis, overweight and obesity are defined on the basis of a combination of BMI and waist circumference (WC) using cut-offs suggested by the World Health Organization (WHO, 2000).

PARTIAL LEAST SQUARES: A statistical method of estimating parameters in path models. This method of estimation is used in a technique that allows the complex
relationships between multiple variables to be examined simultaneously in a path model. This technique is the non-parametric equivalent to structural equation modelling and is suitable for exploratory data analyses (Chin, 1998).

**Sleep Quality:** A construct that encompasses multiple aspects of sleep such as subjective sleep satisfaction, sleep disturbances, sleep disorders, excessive daytime sleepiness and sleep duration. In this thesis, subjective sleep quality was assessed using the Pittsburgh Sleep Quality Index.
# Table of Contents

Certification......................................................................................................................... 2
Acknowledgements............................................................................................................. 3
Abstract............................................................................................................................. 4
Publications from thesis .................................................................................................... 6
Definition of Key Terms...................................................................................................... 8
Table of Contents............................................................................................................... 10
List of Tables..................................................................................................................... 13
List of Figures.................................................................................................................... 14

**Chapter 1: Introduction and Aims**

1.1 Introduction.................................................................................................................. 16
1.2 Aims............................................................................................................................ 18

**Chapter 2: The Link Between Chronic Sleep Restriction and Obesity: A Review of the Underlying Causal Mechanisms**

2.1 Introduction.................................................................................................................. 21
2.2 Causes of Chronic Sleep Restriction........................................................................... 26
2.3 Pathways linking Chronic Sleep Restriction to Obesity............................................. 32
2.4 A Model Linking Chronic Sleep Restriction to Obesity.............................................. 35
2.5 Conclusions................................................................................................................ 39

**Chapter 3: A Link Between Chronic Sleep Restriction and Obesity: Methodological Considerations**

3.1 Introduction.................................................................................................................. 42
3.2 Measurement of Obesity........................................................................................... 46
3.3 Measurement of Sleep............................................................................................... 50
3.4 Consideration of Confounding Variables.................................................................... 54
**List of Tables**

**Table 2.1** Summary of the main studies that have examined the link between chronic sleep restriction and obesity in adults ................................................................. 24

**Table 2.2** Factors associated with chronic sleep restriction in adults .................. 27

**Table 3.1** Summary of major studies examining the link between sleep and obesity in adults......................................................................................................................... 43

**Table 3.2** Current techniques used to estimate body fat and body composition...... 48

**Table 3.3** Current techniques used to estimate components of sleep quality........ 52

**Table 3.4** Major factors that could confound the relationship between sleep and obesity...................................................................................................................... 55

**Table 4.1** Summary of sample characteristics (N = 325) .................................... 62

**Table 4.2** Path coefficients and t statistics for the models predicting BMI, WC and obesity .................................................................................................................. 69

**Table 5.1** Criteria used to classify individuals by obesity-related health risk ....... 77

**Table 5.2** Factors entered into the univariate model predicting obesity ............ 82

**Table 5.3** Factors entered into the univariate models predicting duration/efficiency and perceived sleep quality...................................................................................... 83

**Table 5.4** Factors entered into the univariate model predicting daytime dysfunction......................................................................................................................... 83

**Table 6.1** Comparison of hormone levels and appetite ratings between the baseline and sleep restriction conditions (N = 10) and between the sleep restriction and recovery conditions (N = 9)................................................................................. 95
LIST OF FIGURES

Figure 2.1 Trends in the prevalence of chronic sleep restriction and obesity, 1965 – 2005 ........................................................... 22

Figure 2.2 The association between chronic sleep restriction and selected risk factors 1965 to 2005 .......................................................... 29

Figure 2.3 Schematic representation of the pathways linking chronic sleep restriction to obesity ......................................................... 37

Figure 4.1 Path model linking sleep duration and quality with obesity ............. 68

Figure 5.1 a. Factor loadings for three-factor model proposed by Cole et al. (2006); b Factor loadings for a two-factor model ............................................... 81

Figure 6.1 Changes in EE across the baseline and sleep restriction nights between 2130h and 0730h ........................................................... 93

Figure 6.2 a) the relative change in EE between 2130h and 0130h on the first night of sleep restriction; b) the absolute change in EE between 2130h and 0130h on the second night of sleep restriction ..................................................... 94
CHAPTER 1

INTRODUCTION AND AIMS
1.1 Introduction

Obesity has increased at an alarming rate in recent decades, and has been declared a global health epidemic by the World Health Organization (WHO, 2000). In Australia, the prevalence of obesity in adults has more than doubled in the past 20 years and it is currently estimated that 39% of Australian adults are overweight and a further 21% are obese (Cameron et al., 2003). Similar trends have been observed in other developed countries such as the US, Canada and the UK (Flegal, Carroll, Kuczynski, & Johnson, 1998; Health Canada, 2003; Hirani, 2003; Ogden et al., 2006). These trends are concerning because obesity contributes to elevated morbidity and mortality by increasing the risk of conditions such as diabetes, cardiovascular disease, stroke, osteoarthritis and metabolic-related cancers (Kopelman, 2000; WHO, 2000). Obesity also poses a considerable economic burden, with a total financial cost of approximately $3.8 billion a year in Australia (Access Economics, 2006). Therefore, there is an urgent need to investigate the causes of the present obesity epidemic so that effective treatment and prevention interventions can be developed.

Although obesity has a number of potential physiological, genetic and environmental causes, the present obesity epidemic is generally attributed to environmental factors. In particular, it is generally agreed that increasingly high fat diets (e.g. fast food) and reduced physical activity levels caused by motorised transport, a reduction in physically demanding jobs and increased television viewing, are driving the present epidemic (Hill & Peters, 1998; Hill, Wyatt, Reed, & Peters, 2003; WHO, 2000). As a consequence, most interventions for obesity primarily target diet and physical activity levels. It is therefore concerning that such interventions have not been effective in the long-term treatment and prevention of obesity at a population level (Anderson, Konz, Frederick, & Wood, 2001; Wing & Phelan, 2005). This emphasises
the need to investigate other factors that are also contributing to the present obesity epidemic (Keith et al., 2006).

In the past decade, a number of studies have identified chronic sleep restriction (habitual sleep durations that are less than seven hours a night) as a potential risk factor for obesity. These findings are significant because epidemiological data indicate that the prevalence of chronic sleep restriction has increased from 14% to 40% in the past 40 years (Kripke, Simons, Garfinkel, & Hammond, 1979; National Sleep Foundation, 2005), which corresponds with the rise in obesity observed over the same period of time (Flegal et al., 1998; Ogden et al., 2006). Several cross-sectional studies have also found that chronic sleep restriction is associated with obesity in adults (Bjorvatn et al., 2007; Fogelholm et al., 2007; Gangwisch, Malaspina, Boden-Albala, & Heysmsfield, 2005; Hasler et al., 2004; Ko et al., 2007; Singh, Drake, Roehrs, Hudgel, & Roth, 2005; Taheri, Lin, Austin, Young, & Mignot, 2004; Vioque, Torres, & Quiles, 2000), and long-term prospective studies indicate that chronic sleep restriction predicts increased BMI over several years (Gangwisch et al., 2005; Hasler et al., 2004; López-García et al., 2008; Patel, Malhotra, White, Gottlieb, & Hu, 2006).

These data suggest that chronic sleep restriction could contribute to the development of obesity and may have important implications for the treatment and prevention of obesity. For example, the efficacy of current obesity interventions could be improved by targeting chronic sleep restriction in addition to diet and physical activity. However, this is an under-developed area of research and many aspects of the relationship between chronic sleep restriction and obesity remain unclear. For example, epidemiological studies have been plagued by methodological limitations and inconsistencies that confound current understanding of the relationship between chronic sleep restriction and obesity. Furthermore, the physiological mechanisms linking
chronic sleep restriction to obesity, and the causes of chronic sleep restriction, have not been determined. Therefore, there is a need for extensive research to address these issues in order to clarify how chronic sleep restriction and obesity are related as this could have implications for combating the present obesity epidemic.

1.2 Aims

This thesis investigated the relationship between chronic sleep restriction and obesity by addressing the following aims:

1. Review available research data and identify the physiological and psychosocial pathways linking chronic sleep restriction to obesity (see chapter 2).

2. Identify the major methodological limitations of previous studies that have examined the relationship between chronic sleep restriction and obesity and provided recommendations for future research (see chapter 3).

3. Based on the outcomes from aims 1, and 2, conduct a cross-sectional study examining the relationship between chronic sleep restriction and obesity in adults (see chapters 4 and 5). This addressed the following three research questions:

   a. Are short sleep durations associated with obesity after controlling for possible confounding variables?

   b. Is poor sleep quality independently associated with obesity after controlling for possible confounding variables?

   c. What are the major environmental, psychological, lifestyle and behavioural factors associated with chronic sleep restriction?

4. Examine the effects of sleep restriction on the physiological pathways identified in aim 1 (see chapter 6). This involved investigating the effects of two nights of
sleep restriction on:

a. Neuroendocrine and metabolic hormones such as leptin, ghrelin, insulin, GLP-1, PYY and adiponectin,
b. Ratings of hunger and appetite,
c. Energy expenditure (basal metabolic rate and resting metabolic rate).

This thesis adopted an innovative approach to explore the relationship between chronic sleep restriction and obesity. In particular, Chapter 2 provides an important theoretical framework that was used to guide the subsequent empirical chapters. Chapter 3 outlined the methodological limitations of previous studies which are then directly addressed in chapters 4 and 5. Finally, chapter 6 investigated the effect of sleep restriction on the pathways identified in chapter 2. This novel approach involved investigating the effects of sleep restriction on energy expenditure and the hormonal regulation of body weight. The findings provide important insights into the pathways linking chronic sleep restriction to obesity, which could be targeted and/or manipulated in therapeutic settings to aid the treatment and prevention of obesity.
CHAPTER 2

LITERATURE REVIEW 1

The Link between Chronic Sleep Restriction and Obesity: A review of the underlying causal mechanisms

MANUSCRIPT SUBMITTED TO THE JOURNAL OF BEHAVIORAL MEDICINE IN NOVEMBER 2007

2.1 Introduction

Obesity has increased alarmingly in recent decades (see Figure 2.1) (WHO, 2000), and it is currently estimated that 65% of adults in the US, Australia and the UK are overweight or obese, and 25% to 30% are obese (Cameron et al., 2003; Hirani, 2003; Ogden et al., 2006). These trends are concerning since obesity increases the risk of conditions such as diabetes, cardiovascular disease, stroke, osteoarthritis, some metabolic-related cancers and consequently mortality (Kopelman, 2000; WHO, 2000). Obesity has a multi-factorial aetiology that includes a range of genetic, metabolic, environmental, behavioural and social/cultural factors (Stein & Colditz, 2004; Spiegelman & Flier, 2001). However, because the increase in obesity has been rapid, the present epidemic is generally attributed to dramatic environmental changes that have occurred in recent decades (Hill et al., 2003; WHO, 2000). For example, researchers generally cite high fat diets and increasingly sedentary lifestyles as the main causes of the present epidemic (WHO, 2000). Unfortunately, interventions targeting these factors have only been minimally effective in combating obesity at a population level (Anderson et al., 2001). This suggests that these interventions are not adequately developed and/or fully implemented.

Another possibility is that factors other than diet and physical activity are also contributing to obesity at a population level, but are not being addressed in existing obesity interventions. For example, there is growing evidence that chronic sleep restriction may contribute to the development of obesity and this requires further investigation. In this thesis, chronic sleep restriction is defined as habitual sleep durations that are less than seven hours per night but more than four hours a night (Dinges Rogers, & Baynard, 2005). This definition encompasses any factor (voluntary or involuntary) that prevents the individual from obtaining healthy sleep, which based
on epidemiological evidence refers to sleep durations between seven and eight hours a night (Kripke et al., 2002).

Data from the Cancer Prevention Study and the National Sleep Foundation suggest that the prevalence of chronic sleep restriction has increased from 14% to 40% in the past four decades (Kripke et al., 1979, 2002; National Sleep Foundation, 2005). Figure 2.1 demonstrates that the increase in chronic sleep restriction corresponds closely with the rise in obesity, which raises the intriguing possibility that these epidemics are related. As shown in Table 2.1, several studies have reported that chronic sleep restriction is associated with increased body mass indices (BMI) in adults (Bjorvatn et al., 2007; Gangwisch et al., 2005; Hasler et al., 2004; Ko et al., 2007; Kohatsu et al., 2006; Patel et al., 2004; Shigeta, Shigeta, Nakazawa, Nakamura, & Yoshikawa, 2001; Singh et al., 2005; Taheri et al., 2004; Vioque et al., 2000; Vorona et al., 2005). Four long-term prospective studies in adults have shown that chronic sleep restriction predicts small increases in BMI.
over periods of 2-16 years (Gangwisch et al., 2005; Hasler et al., 2004; López-Garcia et al., 2008; Patel et al., 2006). The odds ratios observed in the above studies are small (e.g. odds ratios 1.29 – 1.75) and indicate limited impact (Bjorvatn et al., 2007; Singh et al., 2005). However, this is not surprising because most studies have been cross-sectional, and sleep and obesity are highly complex and influenced by a multitude of factors. However, preliminary data from laboratory-based studies suggest that chronic sleep restriction could contribute to the development of obesity by altering levels of hormones (e.g. leptin and ghrelin) that are critical in weight regulation (Guilleminault et al., 2003; Spiegel, et al., 1999, 2004a, b).

Available data therefore raise the intriguing possibility that chronic sleep restriction contributes to obesity; these findings may have implications for obesity treatment and prevention. However, this area of research is only in its formative stages and many aspects of the relationship between chronic sleep restriction and obesity remain unclear. For example, recent reviews have summarised some potential neuroendocrine and metabolic pathways linking sleep restriction to diabetes (not discussed in the current thesis) and obesity (Knutson, Spiegel, Penev, & Van Cauter, 2007; Spiegel, Knutson, Leproult, Tasali, & Van Cauter, 2005; Taheri, 2006; Van Cauter et al., 2007) but many other pathways remain to be examined. Furthermore, a major gap in the literature is that the causes of chronic sleep restriction have not been adequately addressed. For example, some studies merely attribute chronic sleep restriction to behavioural sleep curtailment that ‘seems’ to have developed over recent decades (Knutson et al., 2007; Spiegel et al., 2005; Van Cauter et al., 2007). However, the causes of chronic sleep restriction are likely to be complex and encompass voluntary and involuntary factors; these require
Table 2.1. Summary of the main studies that have examined the link between chronic sleep restriction and obesity in adults.

<table>
<thead>
<tr>
<th>Study</th>
<th>Sample Size</th>
<th>Age</th>
<th>Design</th>
<th>Measuresa</th>
<th>Key Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vioque et al. (2000)</td>
<td>1772</td>
<td>≥15</td>
<td>Cross-sectional</td>
<td>BMI, sleep duration, physical activity, TV</td>
<td>Short sleep (≤ 6 hours), TV viewing, and physical activity at work associated with obesity</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>viewing, smoking, education, marital status</td>
<td></td>
</tr>
<tr>
<td>Shigeta et al. (2001)</td>
<td>453</td>
<td>53b</td>
<td>Cross-sectional</td>
<td>BMI, sleep duration</td>
<td>Short sleep (≤ 6 hours) associated with higher BMI</td>
</tr>
<tr>
<td>Kripke et al. (2002)</td>
<td>1116936</td>
<td>30-102</td>
<td>Prospective (6 years)</td>
<td>BMI, sleep duration, smoking, medical history,</td>
<td>Short sleep associated with higher BMI. Relationship linear in males and U shaped in females</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>education, occupation, marital status</td>
<td></td>
</tr>
<tr>
<td>Hasler et al. (2004)</td>
<td>496</td>
<td>19c</td>
<td>Prospective single-age cohort (13 years)</td>
<td>BMI, sleep duration, family history of obesity, physical activity</td>
<td>Short sleep predicted non significant increase in BMI over 13 years</td>
</tr>
<tr>
<td>Patel et al. (2004)</td>
<td>82969</td>
<td>30-55</td>
<td>Prospective (14 years)</td>
<td>BMI, sleep duration, physical activity, smoking, alcohol, depression</td>
<td>Women with long (≥ 9 hours) and short (≤ 5 hours) sleep had higher BMI</td>
</tr>
<tr>
<td>Taheri et al. (2004)</td>
<td>1024</td>
<td>30-60</td>
<td>Cross-sectional</td>
<td>BMI, sleep duration, smoking, alcohol, age, gender, sleep disordered breathing, some neuroendocrine hormones</td>
<td>U shaped relationship between short sleep and BMI. Short sleep also associated with reduced leptin and increased ghrelin</td>
</tr>
<tr>
<td>Gangwisch et al. (2005)</td>
<td>9588</td>
<td>25-74</td>
<td>Cross-sectional</td>
<td>BMI, sleep duration, alcohol, smoking, gender, physical activity, age, depression, fatigue,</td>
<td>Independent association between short sleep (≤ 7 hours) and obesity. Short sleep predicted non</td>
</tr>
</tbody>
</table>

a = measured in adults

b = Body Mass Index

c = cross-sectional

d = sleep duration

24
<table>
<thead>
<tr>
<th>Study</th>
<th>Sample Size</th>
<th>Age Range</th>
<th>Study Design</th>
<th>Confounders</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vorona et al. (2000)</td>
<td>1001</td>
<td>18-91</td>
<td>Cross-sectional</td>
<td>BMI, sleep duration, napping, sleep/wake times, medical problems, caffeine, smoking, alcohol, weight loss products</td>
<td>Significant increase in BMI over 20 years</td>
</tr>
<tr>
<td>Singh et al. (2005)</td>
<td>3158</td>
<td>18-65</td>
<td>Cross-sectional</td>
<td>BMI, sleep duration, age, gender, social economic status, snoring, hypertension, arthritis, alcohol, medical conditions (e.g. heart disease, stroke)</td>
<td>Short sleep durations ≤ 6 hours associated with increased BMI after controlling for confounding variables</td>
</tr>
<tr>
<td>Kohatsu et al. (2006)</td>
<td>990</td>
<td>48b</td>
<td>Cross-sectional</td>
<td>BMI, sleep duration, physical activity, depression, alcohol, snoring</td>
<td>Short sleep (≤ 6 hours) associated with obesity after confounding variables were controlled</td>
</tr>
<tr>
<td>Bjorvatn, et al. (2007)</td>
<td>8860</td>
<td>40-45</td>
<td>Cross-sectional</td>
<td>BMI, sleep duration, gender, smoking, total cholesterol, HDL-cholesterol, triglycerides</td>
<td>U shaped relationship between sleep duration and BMI</td>
</tr>
<tr>
<td>Ko et al. (2007)</td>
<td>4793</td>
<td>19-83</td>
<td>Cross-sectional</td>
<td>BMI, WC, sleep duration, smoking, alcohol, cholesterol, blood pressure, age</td>
<td>Obesity associated with reduced sleep and longer working hours</td>
</tr>
</tbody>
</table>

*Unless specified all BMI and sleep duration based on self-reported data; *average age of sample; *age at baseline; *BMI and/or sleep duration were measured.
identification as they could form the basis of interventions that target chronic sleep restriction to combat obesity.

Therefore, this chapter examines the potential causal mechanisms linking chronic sleep restriction to obesity. This examination includes identifying the key factors that contribute to chronic sleep restriction as well as the physiological mechanisms through which chronic sleep restriction could promote the development of obesity. On the basis of available data, the pathways that I believe are most important in linking chronic sleep restriction to obesity are summarised in a schematic representation. The scope of this chapter is limited to chronic sleep restriction and not acute (i.e. total) sleep deprivation, as the latter is not common in humans and is not relevant to obesity. Furthermore, although there is some evidence that longer sleep durations are associated with obesity, the implications of this link are not clear and will not be addressed in the present chapter (Kripke et al., 2002; Patel et al., 2004; Taheri et al., 2004; Vorona et al., 2005).

### 2.2 Causes of Chronic Sleep Restriction

There are a multitude of factors that could contribute to chronic sleep restriction. Table 2.2 summarises some of the major factors that have been associated with chronic sleep restriction in adults. It is not possible to infer causation solely from these data, since most have been derived from cross-sectional studies. However, large scale epidemiological studies conducted in the US over the past 40 years provide data on population trends for some of the risk factors for chronic sleep restriction that are listed in Table 2.2 (see Figure 2.2). These trends provide an indication of factors that could be contributing to the rise in chronic sleep restriction at a population level and hence
### Table 2.2 Factors associated with chronic sleep restriction in adults.

<table>
<thead>
<tr>
<th>Factor</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Certain Chronic Medical Conditions</strong></td>
<td>Includes arthritis, asthma, back pain, diabetes, obesity, stroke and some cancers (Ancoli-Israel, 2005; National Sleep Foundation, 2005)</td>
</tr>
<tr>
<td><strong>Psychological Disorders</strong></td>
<td>Includes stress, depression anxiety, schizophrenia etc, which are often comorbid with sleep disorders (Benca, 2005)</td>
</tr>
<tr>
<td><strong>Sleep Disorders</strong></td>
<td>Insomnia, OSA, restless leg syndrome and circadian rhythm disorders contribute to chronic sleep restriction (APA, 2000; Dauvilliers et al., 2005)</td>
</tr>
<tr>
<td><strong>Prescription Medications</strong></td>
<td>Includes anti-depressants (e.g. SSRIs), anti-hypertensives, statins, methylphenidate etc. It is difficult to separate the effects of the medication from the respective medical condition (Obermeyer &amp; Benca, 1996; Schweitzer, 2005)</td>
</tr>
<tr>
<td><strong>Psychoactive Substances</strong></td>
<td>Includes alcohol and caffeine consumption, nicotine and recreational drug use b (e.g. cocaine, amphetamines, opiates, cannabis) (Gillin et al., 2005; Obermeyer &amp; Benca, 1996; Riedel et al., 2004)</td>
</tr>
<tr>
<td><strong>Working hours</strong></td>
<td>Increased working hours (e.g. 49 hours or more a week) leave less time for sleep (Akerstedt, 2003; Liu &amp; Tanaka, 2002)</td>
</tr>
<tr>
<td><strong>Shift Work</strong></td>
<td>Work hours not within standard working hours (e.g. 9am to 5pm); common amongst police officers, fire-fighters, doctors, nurses, truck and taxi drivers, pilots etc (Akerstedt, 2003)</td>
</tr>
<tr>
<td><strong>Lifestyle Factors</strong></td>
<td>Includes physical inactivity, television viewing, computer use and/or video games (Driver &amp; Taylor, 2000)</td>
</tr>
<tr>
<td><strong>Environmental Factors</strong></td>
<td>Extreme ambient temperatures (i.e. too cold or too hot), excessive noise and light, and general discomfort (Heller, 2005; Morin et al., 1999)</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td>Sleep durations decline with age (Foley et al., 2004)</td>
</tr>
<tr>
<td><strong>Genetics</strong></td>
<td>Sleep duration is influenced by genetics. Certain sleep disorders (e.g. OSA) have a strong genetic basis (Dauvilliers et al., 2005)</td>
</tr>
</tbody>
</table>

a All factors are positively associated with chronic sleep restriction unless specified
warrant further investigation. The data presented in Figure 2.2 are taken from large epidemiological studies of US adults that include longitudinal studies (e.g. obesity data are derived from the National Health and Nutrition Examination Surveys) and studies that have used similar methods to assess particular variables at different time points (e.g. sleep data are derived from the Cancer Prevention Study and the National Sleep Foundation).

Figure 2.2 demonstrates that the prevalence of mental health problems (anxiety, mood disorders and insomnia), prescription medication use and long work hours have increased in recent decades and appear to correspond with the rise in chronic sleep restriction. As indicated in Table 2.2, there is a strong relationship between mental health and sleep problems, possibly due to shared monoaminergic pathways, which underlie the regulation of sleep and mood (Benca, 2005; Riemann, Berger, & Voderholzer, 2001). The cause(s) of the rise in mental health problems could be due to increased public awareness, changes in the diagnostic criteria, as well as lifestyle and societal changes (e.g. increased work hours). Since the relationship between chronic sleep restriction and poor mental health is likely to be bi-directional (Benca, 2005), further research needs to clarify the extent to which mental health problems contribute to chronic sleep restriction. The increase in prescription medication use (anti-depressant and anti-hypertensive medications) could also contribute to chronic sleep restriction. Most common anti-depressant drugs such as MOAIs (monoamine oxidase inhibitors) and SSRIs (selective serotonin reuptake inhibitors) lead to a reduction in sleep duration by acting on monoaminergic and adrenergic systems that are involved in the regulation of sleep (Obermeyer & Benca, 1996; Schweitzer, 2005). There is also some evidence that anti-hypertensive medications such as beta antagonists and alpha₂ antagonists reduce sleep durations in hypertensive individuals (Obermeyer & Benca...
Figure 2.2 The association between chronic sleep restriction and selected risk factors 1965 to 2005. Data obtained from the Bureau of Labor Statistics (BLS, 2004), Center for Disease Control (CDC, 2006), Behavioral Risk Factor Surveillance System (CDC, 2007), National Comorbidity Survey (Kessler et al., 1994, 2005), Cancer Prevention Study (Kripke et al., 1979, 2002) and the National Sleep Foundation (National Sleep Foundation, 2005).
1996; Schweitzer, 2005). Figure 2.2 also indicates that the use of non-steroidal anti-inflammatory drugs (e.g. Ibuprofen) that are used for pain relief have also increased in recent decades, and are associated with reduced sleep duration possibly through their effects on temperature and melatonin levels (a hormone involved in sleep regulation) (Murphy Myers, & Badia, 1996; Schweitzer, 2005).

The rise in work hours over the past few decades could also be contributing to the rise in chronic sleep restriction. For example, between 1976 and 2002 the average working week in the US increased from 38.1 to 41.8 hours, and the proportion of individuals working a long work week (49 hours and over) doubled (Grosch, Caruso, Rosa, & Sauter, 2006; Rones, Ilg, & Gardner, 1997). These changes could contribute directly to chronic sleep restriction simply by providing less time for sleep. Furthermore, longer working hours could also indirectly contribute to chronic sleep restriction by interfering with family life, limiting relaxation time and contributing to mental health problems (Grosch et al., 2006).

The rise in obesity might also account for some of the increase in chronic sleep restriction over the past 40 years. Although the present chapter focuses primarily on the mechanisms through which chronic sleep restriction leads to obesity, the relationship may be bi-directional. This is since obesity-related conditions such as obstructive sleep apnoea (OSA) and diabetes have the potential to contribute to chronic sleep restriction by disrupting night-time sleep (Young, Peppard, & Taheri, 2005). Therefore, the rise in chronic sleep restriction may be partially accounted for by the rise in obesity, and vice-versa.

Figure 2.2 also demonstrates that the prevalence of chronic medical conditions such as diabetes and hypertension have increased in recent decades and could feasibly contribute to chronic sleep restriction through hormonal changes or increased pain.
which can interfere with sleep. It should be noted that the relationship between chronic medical conditions and chronic sleep restriction may also be bi-directional (Gangwisch et al., 2006; Spiegel et al., 2005; Van Reeth et al., 2000). Chronic alcohol consumption has also become more common in recent decades and may contribute to the rise in chronic sleep restriction by acting on hypothalamic pathways implicated in the regulation of sleep (Roehrs & Roth, 2001). Finally, the proportion of adults aged 45 years and over has increased gradually since 1980 (Figure 2.2). Since increased age is associated with a reduction in sleep duration, it is feasible that an aging population could also be contributing to the rise in chronic sleep restriction (Foley et al., 2004). However, there is a need for research to further investigate the effects of these factors on chronic sleep restriction.

Other factors that are typically associated with chronic sleep restriction at an individual level (e.g., cigarette smoking, shift work, leisure time physical activity) do not correspond with the rise in chronic sleep restriction at a population level (see Figure 2.2). For example, shift-work is a major cause of chronic sleep restriction at an individual level (Akerstedt, 2003; Akerstedt et al., 2003) but the prevalence of shift-work has recently declined (BLS, 2004). Cigarette smoking is associated with reductions in sleep duration (Riedel, Durren, Lichstein, Taylor, & Bush, 2004) but has declined progressively since the 1960s. Physical inactivity is also associated with reduced sleep duration at an individual level (Driver & Taylor, 2000), but there is evidence that the prevalence of physical inactivity has declined since 1990. Therefore, although these factors contribute to chronic sleep restriction at an individual level, epidemiological data indicate that they may not be contributing to the rise in chronic sleep restriction at a population level. A final consideration is that although genetic factors also influence sleep duration (Dauvilliers, Maret, & Tafti, 1979) the gene pool
could not have changed in the space of a couple of decades. Therefore, it is unlikely that genetics has played a role in the rise in chronic sleep restriction in recent decades.

Therefore, on the basis of available data, it is hypothesised that mental health problems, prescription medication use and increased work hours are the major contributors to the rise in chronic sleep restriction that has occurred at a population level. Other factors such as obesity, an aging population, chronic alcohol consumption, diabetes and hypertension may also account for the rise in chronic sleep restriction. Furthermore, the consequences of increased urbanisation (e.g. increased environmental noise, pollution and lighting) could also play a role in the rise in chronic sleep restriction (Rajaratnam & Arendt, 2001), but these effects are difficult to quantify and relevant data are currently unavailable.

The above factors could be targeted as a way to combat chronic sleep restriction at a population level, and this may have important implications for obesity. However, the relative contribution of these factors to chronic sleep restriction is likely to differ depending on the individual and the specific factor, and the potential to modify these factors in therapeutic settings is also likely to vary. For example, mental health problems and sleep disorders would require interventions from specialist practitioners. Work hours could be targeted by behavioural interventions that do not necessarily aim to reduce work hours as this could result in adverse consequences (e.g. a loss of income), but rather attempt to minimise the impact of work hours on sleep patterns.

### 2.3 Pathways linking Chronic Sleep Restriction to Obesity

Chronic sleep restriction could lead to obesity by altering the pathways implicated in the regulation of energy balance. It is well documented that in order for body weight to remain constant, there needs to be a balance between energy intake
(diet) and energy expenditure (basal metabolic rate, physical activity and thermogenesis). This energy balance is maintained by a complex regulatory system involving hypothalamic pathways and energy signalling hormones arising primarily from the gastrointestinal tract and adipose (fat) tissue (Schwartz, Woods, Porte, Seeley, & Baskin, 2000; Wisse, Kim, & Schwartz, 2007). The adipose tissue hormones, leptin and adiponectin, the pancreatic hormone insulin, and the gastrointestinal hormones ghrelin, peptide YY3-36 (PYY) and glucagon-like peptide-1 (GLP-1) are critical energy signalling hormones implicated in energy balance and provide an ongoing indication of body energy stores (i.e. the amount of energy intake relative to energy expenditure) (Huang, Xin, McLennan, & Storlien, 2004; Schwartz et al., 2000; Wisse et al., 2007). The hypothalamus integrates these signals and acts to regulate energy intake and expenditure to ensure that body weight is maintained within a narrow range (Schwartz et al., 2000; Spiegelman & Flier, 2001; Wisse et al., 2007). However, obesity can develop from a chronic positive energy balance (i.e. intake exceeds expenditure), as this disrupts the pathways involved in energy regulation (Spiegelman & Flier, 2001; Wisse et al., 2007).

Emerging data from laboratory based studies indicate that sleep restriction could alter energy balance by disrupting the release of important hormones. For example, Spiegel et al. (1999, 2000, 2004a) found that six consecutive nights of sleep restriction (four hours a night) led to a 30% reduction in the insulin response to glucose, increased evening cortisol levels, increased sympathetic nervous system (SNS) activity, increased growth hormone (GH) secretion, a 26% reduction in thyroid stimulating hormone (TSH) and a 19% reduction in leptin. Guilleminault et al. (2003) found that seven nights of sleep restriction (five hours a night) led to a reduction in leptin rhythm amplitude. Finally, Spiegel et al. (2004b) found that sleep restriction (four hours a
night) over a period of just two nights led to an 18% reduction in leptin and a 28% increase in ghrelin. The increase in the leptin-to-ghrelin ratio corresponded with a 24% increase in hunger and a 23% increase in appetite that was mainly for energy dense foods. These results are consistent with Taheri et al. (2004) who found an association between short sleep durations, increased BMI, elevated ghrelin and reduced leptin levels in a cross-sectional study of 1024 adults.

The direction of the changes in leptin, ghrelin, insulin, GH, TSH and cortisol suggest that sleep restriction could alter the regulation of energy balance in a manner that is predictive of obesity. For example, elevated ghrelin levels act on hypothalamic pathways to stimulate food intake (Wren, Seal, Cohen, Byrnes, Frost, et al., 2001; Zigman & Elmquist, 2003); elevated ghrelin levels therefore suggest that sleep restriction could lead to an increase in food intake. Among its many functions, insulin signals the hypothalamus to reduce energy intake and increase energy expenditure (Huang et al., 2004; Lin, Thomas, Storlien, & Huang, 2000; Porte Baskin, & Schwartz, 2002). Leptin is released in proportion to adipose tissue amount and is a critical component of long term energy regulation as it acts on hypothalamic circuits to reduce energy intake and increase energy expenditure (Porte et al., 2002). Hence, the reductions in leptin and insulin suggest that sleep restriction could lead to obesity by promoting increased food intake and reduced energy expenditure.

The elevated evening cortisol levels suggest that sleep restriction alters the function of the hypothalamic-pituitary-adrenal gland axis which is involved in mood regulation (Leproult, Copinschi, Buxton, & Van Cauter, 1997; Spiegel et al., 2004a; Van Reeth et al., 2000). This also suggests another possible pathway linking sleep restriction to obesity, since elevated cortisol levels have also been shown to promote increased food intake and the accumulation of visceral fat (Björntorp, 2001).
normally functions to stimulate basal metabolic rate; the reduction in TSH therefore suggests that sleep restriction could lead to a reduction in basal metabolic rate which could contribute to obesity (Ganong, 2003). It is unclear whether the reductions in GH have implications for the development of obesity, since GH is associated with a negative energy balance and acts to reduce adipose tissue mass (Richelsen, 1997). However, the effects of sleep restriction on GH have not been examined elsewhere in humans and may warrant further investigation.

It is hypothesised that sleep restriction alters the hormones listed above by influencing common hypothalamic pathways. For example, sleep restriction has been shown to activate the SNS, which inhibits vagal nerve activity and may account for the rise in ghrelin (Heath, Jones, Frayn, & Roberston, 2004; Williams, Grill, Cummings, & Kaplan, 2003). Increased SNS activity also provides negative feedback to adipose tissue and inhibits the release of leptin and possibly insulin (Rayner & Trayhurn, 2001; Spiegel et al., 2005). It is also possible that suprachiasmatic nucleus (SCN) could be disrupted by sleep restriction and also account for the alterations in some of these hormones. The SCN is located in the anterior hypothalamus and regulates the circadian rhythms of several physiological systems, including sleep and hormones involved in energy balance (Saper, Scammell, & Lu, 2005). Therefore the changes in hormones such as leptin, cortisol, TSH and GH with sleep restriction could in part reflect disruptions in SCN functioning.

2.4 A Model Linking Chronic Sleep Restriction to Obesity

On the basis of the data reviewed in this chapter, a schematic representation of the potential psychosocial and physiological pathways linking chronic sleep restriction to altered energy balance and obesity is presented in Figure 2.3. This representation
hypothesizes that mental illness, long work hours and prescription medication use are major contributors to the rise in chronic sleep restriction that has occurred at a population level in recent decades. Other factors such as diabetes, hypertension and chronic alcohol consumption may also be factors that contribute to chronic sleep restriction. Public health programs could address these factors in addition to diet and exercise as a way to more effectively combat obesity. However, the causes of chronic sleep restriction need to be clarified in further studies.

It is also hypothesised that chronic sleep restriction alters the regulation of energy balance by disrupting the release of hormones such as leptin, ghrelin, cortisol and insulin, which are implicated in obesity (see Figure 2.3). However, there are other pathways implicated in energy regulation that could also link sleep restriction to altered energy balance, but have not yet been investigated. For example, PYY3-36 is released from the digestive tract with food consumption and acts on hypothalamic circuits to inhibit further food intake (Bloom, Wynne, & Chaudhri, 2005; Huang et al., 2003). Administration of PYY (total) in rodents has been shown to alter sleep/wake states, possibly via pathways in the amygdala, hypothalamus and/or brainstem (Akanmua, Ukponmwan, Katayama, & Honda, 2006; Corp, Melville, Greenberg, Gibbs, & Smith, 1990; Rahardjo, Huang, Tan, & Deng, 2007). PYY therefore appears to be implicated in both sleep and energy regulation, indicating that the effect of sleep restriction on PYY warrants investigation.

Furthermore, adiponectin is released from adipose tissue in proportion to fat amount and acts on hypothalamic circuits to increase energy expenditure (Beltowiski, 2003; Wolf, 2003). Obese individuals suffering from OSA have lower adiponectin levels compared to obese individuals without OSA (Masserini, Morpurgo, Baldessari, Bossi, Beck-Peccoz, et al., 2006). OSA is a complex disorder, but it is possible that the
Figure 2.3 Schematic representation of the pathways linking chronic sleep restriction to obesity. This figure presents the possible causes of chronic sleep restriction at a population level. The hypothetical neuroendocrine, metabolic and neural pathways implicated in energy regulation and obesity are also presented. Arrows on the left indicate the known effects of sleep restriction on each respective pathway: ↑, increased with sleep restriction; ↓, decreased with sleep restriction; ?, pathway has not yet been examined in relation to sleep restriction. The normal physiological function of each pathway in relation to energy balance is then presented on the right of each hormone: +, pathway normally contributes to a positive energy balance; -, pathway normally contributes to negative energy balance.
lower levels of adiponectin in these individuals could be partially due to the effects of chronic sleep restriction. Therefore, the possible influence of sleep restriction on adiponectin also warrants investigation.

Neural pathways involving orexin could also link chronic sleep restriction and obesity. Orexin containing neurons are located primarily in the lateral and posterior hypothalamus and have widespread projections throughout the central nervous system (Mieda & Yanagisawa, 2002). Orexin has been implicated in the regulation of sleep/wake states as well as feeding behaviour (Mieda & Yanagisawa, 2002). For example, orexin plays a role in maintaining wakefulness, and disruptions in the orexin system have been associated with narcolepsy (Kok, Meinders, Overeem, Lammers, Roelfsema, et al., 2002; Mieda & Yanagisawa, 2002).Injecting orexin in rodents stimulates feeding and could lead to obesity (Mieda & Yanagisawa, 2002). It is therefore possible that sleep restriction could lead to altered energy regulation via the orexin system.

Furthermore, although this chapter has primarily focused on the possible pathways linking chronic sleep restriction to obesity, it is acknowledged that the relationship is bi-directional (see Figure 2.3). This relationship is proposed because obesity can also contribute to chronic sleep restriction via OSA, depression, stress and lowered physical activity (Björntorp, 2001; Hill et al., 2003).

Interventions could ultimately be developed that target chronic sleep restriction as a way to improve the treatment and prevention of obesity. However, there is first a need for more studies to examine the mechanisms linking chronic sleep restriction to obesity. This research is particularly pertinent since the physiological pathways linking chronic sleep restriction to altered energy balance and obesity have only been examined in regards to short-term sleep restriction. Therefore, existing studies have not yet
demonstrated that chronic sleep restriction leads to actual weight gain and visceral fat accumulation via these pathways.

The pathways outlined in Figure 2.3 will guide future research attempting to determine the physiological and psychosocial pathways linking chronic sleep restriction to obesity. These pathways will be difficult to examine in laboratory studies, but could be investigated by long-term prospective studies that examine the associations between changes in sleeping patterns, body composition and key hormones identified in Figure 2.3. Furthermore, future studies should also examine the effect of interventions targeting the causes of chronic sleep restriction in the treatment of obesity as this has not yet been examined in the literature. These studies will provide clearer insights into the mechanisms linking chronic sleep restriction to obesity which will have implications for combating the obesity epidemic.

2.5 Conclusions

This chapter has reviewed available data suggesting the chronic sleep restriction is a novel risk factor for obesity. The potential psychosocial and physiological pathways linking chronic sleep restriction to obesity have been examined. It is hypothesised that chronic sleep restriction could be the result of a combination of factors including increased work demands, a rise in mental health problems and increased use of prescription medications that restrict sleep. Furthermore, it is hypothesised that chronic sleep restriction contributes to obesity by altering levels of hormones that are important in regulation energy balance. These pathways are summarised in a schematic representation linking chronic sleep restriction to obesity, and can be used to guide future research in behavioural medicine. This area of research is significant given that the obesity epidemic continues to grow and poses a number of
major health, social and economic problems (Kopelman, 2000); targeting the amount we sleep could be an important step in combating this health problem.
CHAPTER 3

LITERATURE REVIEW 2

A Link between Chronic Sleep Restriction and Obesity: Methodological Considerations

MANUSCRIPT PUBLISHED IN PUBLIC HEALTH

3.1 Introduction

The prevalence of obesity has increased dramatically over the past 30 years and is currently considered a global epidemic (Hedley et al., 2004; WHO, 2000). Unhealthy diets and insufficient exercise are cited as the main causes of this epidemic (WHO, 2000) but public health programs that target these factors have only been moderately effective in combating obesity (Anderson et al., 2001). Part of the reason for this outcome could be that other factors also contribute to obesity, but have not been incorporated into existing obesity interventions (Keith et al., 2006). One possible overlooked cause of obesity is chronic sleep restriction (sleep durations less than seven hours a night), which has become more common in the past 30 years (Kripke et al., 1979, 2002) and is associated with increased body mass indices (BMI) in children (Taheri, 2006) and adults (see Table 3.1). It appears that the association between chronic sleep restriction and increased BMI is evident up until middle age, but is absent in adults aged over 40 or 49 years (Gangwisch et al., 2005; Hasler et al., 2004). The relationship between sleep durations and BMI could be also U-shaped, but the implications that long sleep has for obesity are unclear and will not be discussed in this chapter.

Unfortunately, causal inferences cannot be drawn from these cross-sectional studies. It is possible that obesity simply leads to chronic sleep restriction via sleep disordered breathing (e.g. OSA) (Young et al., 2005). However, Gangwisch et al. (2005) found that chronic sleep restriction predicted higher BMI ten years after baseline. In a single age cohort of adults, Hasler et al (2004) found that reductions in sleep durations predicted increases in BMI, although this effect did not reach statistical significance. Preliminary data from small scale laboratory studies in young adults also indicate that sleep restriction over several days alters energy regulatory hormones (e.g.
<table>
<thead>
<tr>
<th>Author and Year</th>
<th>Obesity Measures</th>
<th>Sleep Measures</th>
<th>Confounding variables *</th>
<th>Major Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vioque et al.</td>
<td>BMI (measured)</td>
<td>Self-reported sleep duration (hours of sleep per day)</td>
<td>None</td>
<td>Sleep duration inversely associated with BMI</td>
</tr>
<tr>
<td>(2000)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Shigeta et al.</td>
<td>BMI (measured)</td>
<td>Self-reported sleep duration and bedtime</td>
<td>None</td>
<td>Short sleep (6 hours or less) associated with increased BMI</td>
</tr>
<tr>
<td>(2001)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kripke et al.</td>
<td>BMI (calculated from self-reported</td>
<td>Self-reported sleep duration: ‘On the average, how</td>
<td>None</td>
<td>U shape associated between sleep duration and BMI in females. Inverse association</td>
</tr>
<tr>
<td></td>
<td>height and weight)</td>
<td>many hours do you sleep each night?’</td>
<td></td>
<td>between sleep duration and BMI in males</td>
</tr>
<tr>
<td>(2002)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hasler et al.</td>
<td>BMI (calculated from self-reported</td>
<td>Interview regarding bedtimes, wake times, sleep</td>
<td>Gender, education,</td>
<td>Short sleep predicted a non-significant increase in BMI over a 13 year period</td>
</tr>
<tr>
<td>(2004)</td>
<td>height and weight)</td>
<td>latency, sleep behaviours and sleep disorder</td>
<td>physical activity,</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>symptoms</td>
<td>smoking, binge eating,</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>childhood depression,</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>family history of obesity</td>
<td></td>
</tr>
<tr>
<td>Patel et al.</td>
<td>BMI (calculated from self-reported</td>
<td>Self-reported sleep duration: ‘How many hours of</td>
<td>None</td>
<td>U shape association between sleep durations and BMI in women</td>
</tr>
<tr>
<td>(2004)</td>
<td>height and weight)</td>
<td>sleep do you get in a 24 hours period?’</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>BMI (calculated from self reported height and weight)</td>
<td>Self-reported sleep duration</td>
<td>Age, gender, loud snoring, hypertension, diabetes, arthritis and alcohol consumption</td>
<td>Short sleep durations associated with increased BMI after controlling for confounding variables</td>
</tr>
<tr>
<td>---------------</td>
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</tr>
<tr>
<td>Singh et al.</td>
<td>BMI (calculated from self reported height and weight)</td>
<td>Self-reported sleep duration</td>
<td>Age, gender, loud snoring, hypertension, diabetes, arthritis and alcohol consumption</td>
<td>Short sleep durations associated with increased BMI after controlling for confounding variables</td>
</tr>
<tr>
<td>(2005)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gangwisch et al.</td>
<td>BMI (measured at baseline, self-reported at follow up)</td>
<td>Self-reported: Sleep duration - ‘How many hours of sleep do you usually get a night (or when you usually sleep)?’ Night-time awakenings Daytime sleepiness</td>
<td>Depression, physical activity, education, ethnicity, alcohol consumption, cigarette use, gender, waking during the night, daytime sleepiness, age</td>
<td>Independent association between short sleep durations and obesity. Short sleep predicted a non-significant increase in BMI over a 10 year period</td>
</tr>
<tr>
<td>(2005)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Taheri et al.</td>
<td>BMI (measured)</td>
<td>Polysomnography</td>
<td>Age and gender</td>
<td>U shape relationship between sleep durations (sleep diary) and BMI. Short sleep linked to decreased leptin and increased ghrelin</td>
</tr>
<tr>
<td>(2004)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vorona et al.</td>
<td>BMI (measured)</td>
<td>Self-reported bedtimes, wake times, total sleep time, sleep disorders</td>
<td>None</td>
<td>U shape relationship between sleep duration and BMI</td>
</tr>
<tr>
<td>(2005)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>BMI (measured)</td>
<td>Self-reported:</td>
<td>Controlled Variables</td>
<td>Short sleep durations linked with higher BMI</td>
</tr>
<tr>
<td>---------------------------</td>
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<td>--------------------------------------------------------------------------------------</td>
<td>---------------------------------------------</td>
</tr>
<tr>
<td>Kohatsu et al. (2006)</td>
<td>BMI (measured)</td>
<td>Sleep duration - ‘How many hours of sleep do you get in a typical work day?’ Snoring</td>
<td>Gender, age, education, physical job demand, income, alcohol consumption, depressive symptoms, marital status, snoring</td>
<td>Short sleep durations (less than 6 hours) linked with higher BMI</td>
</tr>
<tr>
<td>Bjorvatn et al. (2007)</td>
<td>BMI (measured)</td>
<td>Self-reported sleep duration</td>
<td>Gender and smoking</td>
<td>Short sleep durations were associated with increased BMI after controlling for gender and smoking</td>
</tr>
<tr>
<td>Ko et al. (2007)</td>
<td>BMI (measured)</td>
<td>Self-reported sleep duration.</td>
<td>Age, smoking, alcohol consumption, blood pressure, and work hours</td>
<td>Independent association between short sleep and increased WC and BMI in males</td>
</tr>
<tr>
<td>Fogelholm et al. (2007)</td>
<td>BMI (measured)</td>
<td>Self-reported sleep duration.</td>
<td>Age, education, physical activity sleep apnoea, and sleep disturbances</td>
<td>Independent associations between short sleep durations (≤ 6 hours) and increased WC and BMI</td>
</tr>
</tbody>
</table>

BMI, body mass index; WC, waist circumference.

* Note: only the variables that were controlled when examining the relationship between sleep and obesity are included. Although some studies measured other variables, these were not necessarily included in such analyses.
leptin and ghrelin) in a manner consistent with increased appetite and may predispose individuals to weight gain (Spiegel et al., 2004b). For example, sleep restricted to four hours per night over two days led to an 18% decrease in leptin and a 28% increase in ghrelin, which corresponded with a 24% increase in hunger and a 23% increase in appetite (Spiegel et al., 2004b). These findings are consistent with a large cross-sectional study of 1024 adults, where relationships were evident between short sleep durations, increased BMI’s, elevated ghrelin and reduced leptin (Taheri et al., 2004).

Together, these studies provide compelling, albeit tentative, evidence that chronic sleep restriction contributes to obesity. It might therefore be possible to improve the efficacy of current treatment and prevention interventions for obesity by targeting short sleep durations. However, this area of research is only in its infancy and a comprehensive understanding of how sleep and obesity are related in the general population is currently lacking. Part of the problem is that previous epidemiological studies have been predominantly cross-sectional and have also been hampered by varied methodological limitations relating to the measurement of obesity and sleep, which need to be addressed. Furthermore, few studies have controlled for the effects of multiple confounding variables on the link between sleep and obesity. The aim of the present chapter is to address these limitations and provide recommendations for future research to examine how chronic sleep restriction contributes to obesity. This will have implications for public health programs that target chronic sleep restriction in the treatment and prevention obesity.

3.2 Measurement of Obesity

Obesity refers to an excess of body fat that can be estimated using a range of measures, which vary by feasibility of field use, cost and accuracy (see Table 3.2)
(Deurenberg & Yap, 1999; Jebb & Wells, 2005). Previous studies examining the relationship between sleep and obesity in adults have utilised BMI to measure body fat levels, although Ko et al. (2007) and Fogelholm et al. (2007) also measured waist circumference (WC) (see Table 3.1). Both BMI and WC are convenient for use in large samples and for the most part correspond well with laboratory based methods outlined in Table 3.2 (r = .60 to .80) (Deurenberg & Yap, 1999). However, BMI and WC do not provide optimal estimates of body fat levels or obesity-related health risk. For example, BMI does not distinguish between lean mass and fat mass, and the relationship between BMI and percentage body fat varies by age, fitness levels and ethnicity (Jebb & Wells, 2005; He, Tan, Li & Kung, 2001; Prentice & Jebb, 2001; Zhu et al., 2002). BMI also does not provide information about body fat distribution, which is important since visceral fat poses a greater health risk compared to subcutaneous fat (Jebb & Wells, 2005; Zhu et al., 2001). Five studies have also relied on self-reported height and weight to calculate BMI (see Table 3.1), which has the potential to result in substantial underestimations of obesity-related health risk (Flood, Webb, Lazarus, & Pang, 2000).

Measuring WC in addition to BMI overcomes some of these issues, since WC provides an estimate of abdominal body fat which is an independent risk factor for a range of diseases such as diabetes and heart disease (Zhu et al., 2002). Measurements of WC are particularly useful in estimating health risk in individuals with a BMI between 18.5 and 34.9 (Health Canada, 2003; National Heart, Lung, and Blood Institute, 1998). This suggests that WC should be used more extensively in future studies to complement the information obtained from BMI. However, it should be noted that WC measurements cannot distinguish between subcutaneous and visceral fat, and as such lacks some precision in assessing health risk (Zhu et al., 2002). As a consequence, more accurate estimates of body fat levels and obesity-related health risk are needed to
Table 3.2. Current techniques used to estimate body fat and body composition.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Description</th>
<th>Accuracy</th>
<th>Field Use Feasibility</th>
<th>Cost</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI</td>
<td>A measure of weight corrected for height</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>Convenient for field studies but does not distinguish between lean and fat mass</td>
</tr>
<tr>
<td>WC</td>
<td>An estimate of central body fat</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>A good marker of health risk but cannot distinguish between subcutaneous and visceral fat</td>
</tr>
<tr>
<td>Skinfold thickness</td>
<td>Estimates total body fat from measurements of subcutaneous fat</td>
<td>3 2 1</td>
<td>2 1</td>
<td>1</td>
<td>Assumes a constant relationship between subcutaneous and visceral fat</td>
</tr>
<tr>
<td>Bioelectrical</td>
<td>Estimates FM and FFM by</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>Data corresponds</td>
</tr>
<tr>
<td>Method</td>
<td>Description</td>
<td>Ease of Use</td>
<td>Cost</td>
<td>Accuracy</td>
<td>Suitable for Field Use</td>
</tr>
<tr>
<td>---------------------------------------</td>
<td>-----------------------------------------------------------------------------</td>
<td>------------</td>
<td>------</td>
<td>----------</td>
<td>------------------------</td>
</tr>
<tr>
<td>Impedance</td>
<td>measuring body impedance to a small current</td>
<td>1</td>
<td>3</td>
<td>3</td>
<td>well with laboratory based methods, suitable for field use, assumes body hydration is constant</td>
</tr>
<tr>
<td>Dual energy X-ray absorptiometry</td>
<td>2D scan of the whole body using X-rays of two energy levels</td>
<td>1</td>
<td>3</td>
<td>3</td>
<td>not suitable for field use</td>
</tr>
<tr>
<td>Imaging Techniques (CT, MRI)</td>
<td>Provides 3D information on body composition</td>
<td>1</td>
<td>3</td>
<td>3</td>
<td>not suitable for field use</td>
</tr>
<tr>
<td>Hydrometry</td>
<td>FFM, FM and % body fat determined from estimates of total body water</td>
<td>1</td>
<td>3</td>
<td>3</td>
<td>not suitable for field use</td>
</tr>
<tr>
<td>Densitometry</td>
<td>FM and FFM calculated from body density. Includes water and air displacement, underwater weighing</td>
<td>1</td>
<td>3</td>
<td>3</td>
<td>not suitable for field use</td>
</tr>
</tbody>
</table>

BMI, body mass index; CT, computerized tomography; FFM, fat free mass, FM, fat mass; MRI, magnetic resonance imaging; WC, waist circumference
ensure a clearer understanding of how sleep and obesity are linked. A simple alternative could be to combine BMI and WC into a single index of obesity-related health risk, which is more accurate than either measure alone (Zhu et al., 2004). Other field techniques such as skinfold thickness measures are less appropriate since they are potentially invasive, require trained observers and can be imprecise (Deurenberg & Yapp, 1999). Laboratory based methods, such as dual energy X-ray absorptiometry (DXA) or magnetic resonance imaging (MRI), provide the most precise and detailed information regarding body composition, but are expensive and cumbersome for field use (see Table 3.2). A feasible alternative could be to use bio-electrical impedance devices, which provide estimates of body composition (e.g. fat mass and fat free mass) that correspond well with DXA and MRI ($r = .73 - .92$) (Deurenberg, Tagtiabue, & Schouten, 1995; Steijaert, Deurenberg, Van Gaal, & De Leeuw, 1997). The accuracy of these devices is influenced by factors relating to the amount of total body water and extracellular fluid, as well as body shape and ethnicity (Deurenberg et al., 1995; Steijaert et al., 1997). Nevertheless, these do provide relatively inexpensive, portable and non-invasive estimates of body composition that are suitable for field research and would complement the information obtained from BMI and WC.

3.3 Measurement of Sleep

The studies listed in Table 3.1 have used self-report measures to estimate sleep durations. The only exception has been Taheri et al. (2004) who examined polysomnography (PSG) in addition to several self-report items. Self-report measures are convenient for use in field studies, but vary by accuracy and are subject to age and gender biases (Reynor & Horne, 1995). This has been compounded by inconsistencies relating to the types of self-report items used to measure sleep duration across different
studies (see Table 3.1). Furthermore, most studies have simply measured sleep duration and not other components of sleep quality such as sleep satisfaction and sleep disturbances that are important to physical and mental health (Pilcher, Ginter & Sadowsky, 1997) and might also be relevant to obesity.

Valid and reliable estimates of sleep quality therefore need to be obtained in future studies, and there are a variety of methods that could potentially be used (see Table 3.3). Polysomnography (PSG) is currently considered the ‘gold standard’ for measuring sleep and provides accurate and precise estimates of important sleep parameters. However, this technique is expensive, time consuming and is not ideal for large sample sizes. Furthermore, it requires participants to be attached to multiple electrodes and sleep overnight in a laboratory; these factors may disrupt normal sleep patterns (Ancoli-Israel, 2005).

An alternative could be to utilise actigraphs, which are small devices that can be worn on the wrist or ankle and provide a continuous record of sleep and wake patterns over a period of several days (Ancoli-Israel, 2005). Importantly, since actigraphs are portable and non-invasive, the participant can sleep at home and engage in normal daily activities; this is likely to provide a more realistic indication of sleep patterns compared to PSG (Ancoli-Israel, 2005). Compared to PSG, however, it should be noted that actigraphs can be less accurate in individuals who experience disturbed sleep, and can overestimate sleep duration (Kushida et al., 2001; Lockley, Skene & Arendt, 1999).

Various components of sleep can also be examined via subject measures. For example, sleep diaries provide an easy and efficient estimate of habitual sleep duration, but there is evidence that individuals overestimate their sleep amount (Silva et al., 2007). However, the advantage of self-report measures is that they provide insight in subjective components of sleep such as sleep satisfaction and daytime sleepiness, which may be
Table 3.3. Current techniques used to estimate components of sleep quality.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Description</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Polysomnography</strong></td>
<td>Sleep data obtained from a combination of electrooculogram, electromyogram, and electroencephalogram</td>
<td>Provides objective estimates of sleep parameters including sleep stages</td>
<td>Not suitable for field use:</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- expensive</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- laboratory based</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- invasive</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- not reflective of normal sleep</td>
</tr>
<tr>
<td><strong>Actigraphy</strong></td>
<td>Activity monitors are attached to the wrist, ankle or trunk</td>
<td>Suitable for field use</td>
<td>Overestimates some sleep parameters such as total sleep time and awakenings during the night. Consequently, less accurate than polysomnography and some sleep diaries</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- inexpensive</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- non invasive</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Provides a continuous record of sleep/wake cycles over several days</td>
<td></td>
</tr>
<tr>
<td><strong>Sleep Diaries</strong></td>
<td>Participant keeps a daily log of self-reported sleep/wake times and other relevant information</td>
<td>Sleep patterns measured over one to two weeks. Data corresponds well with polysomnography.</td>
<td>Some biases in self-reported sleep. The reliability and validity of data depends on how well the diary is designed</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Suitable for field use</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- inexpensive</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- easy to administer</td>
<td></td>
</tr>
<tr>
<td><strong>Pittsburgh Sleep Quality Index</strong></td>
<td>Questionnaire that categorizes responses into seven sleep quality components and provides</td>
<td>Suitable for field use</td>
<td>Possibility of response biases</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- low cost</td>
<td></td>
</tr>
</tbody>
</table>
| a global sleep quality score | - easy to administer and time efficient  
| | Provides subscale and total scores of sleep quality |
| **Epworth Sleepiness Scale** | Eight item scale that examines sleep propensity across 8 different situations |
| Suitable for field use | - low cost  
| | - quick and easy to administer and complete |
| Only provides information relating to one component of sleep quality (daytime sleepiness) |
relevant to obesity. In particular, the Pittsburgh Sleep Quality Index (PSQI) (Buysse, Reynolds, Monk, Berman & Kupfer, 1989) and the Epworth Sleepiness Scale (Johns, 1991) are widely used to assess important components of subjective sleep quality and have sound psychometric properties (see Table 3.3).

As a consequence, it is suggested that studies examining the relationship between sleep and obesity measure sleep using a combination of actigraphy and appropriate subjective scales. Polysomnography remains the ‘gold standard’ for assessing sleep in laboratory settings, but actigraphy and sleep scales could be more appropriate for large scale population studies that attempt to link sleeping patterns to the development of obesity.

3.4 Consideration of Confounding Variables

Studies examining the relationship between sleep and obesity also need to adequately control for potential confounding variables. Confounding occurs when the variance of one or more independent variables that are extraneous to the research question overlaps with the variance of the independent variable of interest (Kerlinger, 1969). As a consequence, it becomes unclear whether the observed relationship is due to extraneous independent variables, the independent variable of interest, or a combination of both (Kerlinger, 1969). Table 3.4 lists some of the main behavioural, lifestyle, health and environmental factors that could potentially confound the relationship between sleep and obesity. Inter-individual variation in levels of hormones such as leptin, ghrelin and growth hormones might also confound the association between sleep and obesity, and need to be considered. These factors must be controlled for to ensure that the association between sleep and obesity is independent and not simply the by-product of confounding variables. Five of the studies listed in Table 3.1
<table>
<thead>
<tr>
<th><strong>Factor</strong></th>
<th><strong>Impact on Sleep</strong></th>
<th><strong>Evidence Rating</strong></th>
<th><strong>Impact on Obesity</strong></th>
<th><strong>Evidence Rating</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol Consumption</td>
<td>Moderate, frequent alcohol consumption close to normal bedtimes reduces sleep quality and duration (Obermeyer &amp; Benca, 1996)</td>
<td>1</td>
<td>Moderate to heavy, frequent alcohol consumption is associated with obesity (Suter, 2005)</td>
<td>1</td>
</tr>
<tr>
<td>Age</td>
<td>Sleep duration and quality decline with age (Foley et al., 2005)</td>
<td>1</td>
<td>Obesity increases with age (Ogden et al., 2006)</td>
<td>1</td>
</tr>
<tr>
<td>Chronic Diseases</td>
<td>Cancer, asthma, arthritis, back pain, heart disease, diabetes etc can contribute to chronic sleep restriction through a number of mechanisms (e.g. behaviour changes, hormonal alterations) (National Sleep Foundation, 2005)</td>
<td>1</td>
<td>Certain conditions contribute to weight gain. A number of mechanisms can be involved: hormonal alterations, behaviour changes (National Sleep Foundation, 2005)</td>
<td>1</td>
</tr>
<tr>
<td>Psychological Disorders</td>
<td>Decreased sleep quality (Benca, 2005)</td>
<td>1</td>
<td>Positively associated with obesity (Wurtman, 1993)</td>
<td>1</td>
</tr>
<tr>
<td>Physical Activity</td>
<td>Promotes good sleep quality. However, vigorous exercise within three hours of bedtime can impair sleep (Stepaniski &amp; Wyatt, 2003)</td>
<td>1</td>
<td>Inversely associated with obesity (Lahti-Koski et al., 2002)</td>
<td>1</td>
</tr>
<tr>
<td>Smoking</td>
<td>Heavy cigarette smoking, close to normal bed times impairs sleep duration and quality linked to poor sleep. Ex-smokers also have poor sleep quality and duration (Obermeyer &amp; Benca, 1996)</td>
<td>1</td>
<td>Current smokers less obese. Ex-smokers more obese (Lahti-Koski et al., 2002)</td>
<td>1</td>
</tr>
</tbody>
</table>
Television and Video Game Use

Impairs sleep particularly if close to bedtime (Stepaniski & Wyatt, 2003)

2 Positively associated with obesity (Hill & Peters, 1998)

Caffeine Consumption

Reduces sleep duration and quality, particularly with heavy consumption within four to six hours of bedtime (Obermeyer & Benca, 1996)

1 Energy drinks containing caffeine and sucrose could contribute to obesity (Rush et al., 2006)

1 = strong, consistent evidence of a link; 2 = factors appear linked, but the evidence is weak or insufficient.

have not controlled for any potential confounding variables, while the remaining studies have been inconsistent in regards to the types of variables that have been controlled. Some potentially important factors such as caffeine consumption and stress have been overlooked completely and should be addressed (Björntorp, 2001; Landolt, Werth, Borbély & Dijk, 1995).

Furthermore, studies that have previously controlled for some confounding variables such as alcohol consumption, physical activity and smoking have typically only accounted for information relating to amount (e.g. number of alcoholic drinks per day) and/or status (e.g. smoker versus non-smoker). However, the impact of these factors on sleep quality and obesity is complex and also depends on the type, frequency, timing and history of the particular behaviour (see Table 3.4). For example, the effect of alcohol on sleep and obesity is most pronounced when consumption is moderate, frequent and within four to six hours of normal bedtime (Roehrs & Roth, 2001). Similar patterns are observed for other factors such as smoking, physical activity, napping behaviour and caffeine consumption (Table 3.4). Future research must
therefore aim to control for a wider range of potential confounding variables, taking factors such as type, frequency, timing and history into account where relevant.

### 3.5 Recommendations for Future Research

If the link between sleep and obesity is to be comprehensively understood, valid and reliable measures of sleep and obesity need to be obtained. Laboratory methods provide precise estimates of body composition, but are not feasible for use in field studies (see Table 3.2). BMI and WC are convenient for use in the field, but provide inexact estimates of percentage body fat and limited body composition information. It is recommend that future studies also utilise bioelectrical impedance devices, as these instruments could be used in this context to complement the information obtained from BMI and WC (see Table 3.2).

Similarly, PSG provides accurate estimates of sleep parameters but is not appropriate for field research. Instead actigraphs and subjective scales such as the PSQI remain the most viable options for examining sleep in large populations. These instruments are relatively inexpensive and can monitor sleeping patterns and subjective sleep quality over periods of several days or weeks.

Future studies also need to examine the influence of variables that affect sleep and obesity. This research could potentially include a wide range of lifestyle, behavioural, health and environmental factors, some of which are known to directly influence both sleep quality and obesity. As a minimum, it is recommended that future studies control for age, mental health (e.g. stress and depression), physical activity, alcohol and caffeine consumption, and sedentary activities such as television viewing. The type, frequency, history, amount and timing of each variable should also be taken into account where relevant (see Table 3.4).
These recommendations will enable a more comprehensive understanding of the complex link between sleep quality and obesity. Cross-sectional studies will be important in describing this link, but are limited since cause and effect relationships cannot be determined. This is significant since the relationship between sleep and obesity is bi-directional and complex. Studies will therefore need to move from merely describing this link to demonstrating that poor sleep quality and chronic sleep restriction contributes to obesity. One possible strategy is to investigate the effects of chronic sleep restriction on metabolic and endocrine functioning. These studies would need to be laboratory based and would be difficult to implement in large samples. Alternatively, prospective studies could examine the long-term impact of poor sleep quality on body composition taking into account the considerations discussed above. Biomarkers such as leptin and ghrelin could be monitored to provide a further indication of how chronic sleep restriction leads to obesity. Studies should also examine the effects of improved sleep amount and quality on body composition in overweight and obese individuals who experience chronic sleep restriction. If improvements in sleep lead to even minor decreases in percentage body fat, it would have significant health implications.

It is critical that the complex relationships between sleep, obesity, lifestyle, behaviour, health and the environment are determined as this will have important implications for combating the current obesity epidemic. The recommendations provided in this chapter will aid future research in clarifying the nature and the extent of these relationships. It is possible that targeting poor sleep in addition to diet and exercise will improve the efficacy of current public health programs and could be a key step in combating the obesity epidemic.
CHAPTER 4

EMPIRICAL STUDY 1

A Path Model Examining the Relationships between
Sleep Duration, Obesity and Psychosocial Variables in
Adults

MANUSCRIPT SUBMITTED TO THE JOURNAL OF PSYCHOSOMATIC RESEARCH, MARCH 2008

4.1 Introduction

The prevalence of obesity is increasing at an alarming rate (Flegal et al., 1998; Hedley et al., 2004) and is associated with conditions such as cardiovascular disease, stroke, metabolic related cancers and psychological disorders (WHO, 2000). Interventions for obesity generally target poor diet, low physical activity and sedentary behaviours, but these have been ineffective in the long-term treatment and prevention of obesity at a population level (Anderson et al., 2001). This emphasizes the need to consider other possible risk factors for obesity.

Chronic sleep restriction (sleep durations less than seven hours a night) is an increasingly common condition in the US, Europe and Australia (Bartlett et al., 2008; Kronholm et al., 2006; National Sleep Foundation, 2005) and has recently been associated with obesity in adults (Bjorvatn et al., 2007; Ko et al., 2007; Kohatsu et al., 2006; Singh et al., 2005; Vioque et al., 2000; Vorona et al., 2005). Four long-term prospective studies have also demonstrated that chronic sleep restriction predicts the development of obesity over several years (Gangwisch et al., 2005; Hasler et al., 2004; López-Garcia et al., 2008; Patel et al., 2006). These data raise the possibility that chronic sleep restriction contributes to the development of obesity and could potentially be targeted in the treatment and prevention of obesity.

However, this is an under-researched area and many aspects of the relationship between chronic sleep restriction and obesity remain unclear. For example, the majority of previous studies have simply examined for an association between sleep duration and obesity without considering the possible impact of relevant psychosocial factors. This is important because both sleep and obesity are influenced by a range of psychological (e.g. stress and depression) and behavioural factors (e.g. physical activity, caffeine and alcohol consumption) (Benca, 2005; Dijk, Duffy, & Czeisler, 2000; Reynor & Horne,
1995; Roberts, Deleger, Strawbridge, & Kaplan, 2003; Roehrs & Roth, 1997; Stepanski & Wyatt, 2003; Van Reeth et al., 2000; Vgontzas et al., 2000). It is feasible that some of these factors could mediate the relationship between chronic sleep restriction and obesity, and hence require identification. Furthermore, there is also a need to investigate the possible determinants of chronic sleep restriction, as these factors could be targeted as a way to promote healthy sleep durations, which may facilitate the treatment and prevention of obesity.

Therefore, in order to address these limitations, the present study examined a path model linking sleep, obesity and relevant psychosocial factors. To the best of our knowledge, the relationship between sleep and obesity has not previously been examined using path analysis, which is important because this approach allows for multiple relationships between several variables to be examined simultaneously (Chin, 1998). Therefore, the present approach will provide a more definitive insight into the nature of the relationship between chronic sleep restriction and obesity.

4.2 Method

4.2.1 Participants

Three hundred and twenty-five adults (199 females and 126 males) aged 18 to 87 years participated in the study (see Table 4.1 for demographic information). Participants were recruited from a sample of university students as well as businesses and local community groups. The protocol for this study was approved by the Human Research Ethics Committee at the University of Wollongong; informed consent was obtained from all participants.
4.2.2 Apparatus

Weight was measured to the nearest 0.1kg using electronic weight scales whilst the participant was lightly clothed (e.g. t-shirt and shorts) and height was measured without shoes to the nearest 0.1cm using a stadiometer. The same scales were used for all participants to ensure consistency in measurement. Body mass index (BMI) was then calculated by dividing weight (kg) by height (m²). Waist circumference (WC) was measured to the nearest 0.5cm by placing a tape measure around each participant at the level of the umbilicus following normal expiration whilst the participant was standing (ACSM, 2005).

BMI and WC cut-offs suggested by the World Health Organization (WHO, 2000) were used to categorise individuals as lean, overweight or obesity. Leanness was defined by a BMI between 18.5 and 24.9 and a WC less than 80.0cm in females and less than 94.0cm in males. Overweight was defined by a BMI between 25.0 and 29.9, and a WC less than 88.0cm in females and less than 102.0cm in males. However, individuals were also considered overweight if they had a BMI less than 25.0, but a WC of 80.0cm or more in females and 94.0cm or more in males. Obesity was defined by a BMI of 30.0 or greater, or a WC of 88.0cm or more in females and 102.0cm or more in males.
4.2.3 Materials

Participants completed a questionnaire that assessed sleep quality and duration, and lifestyle, behavioural, environmental and psychological factors that could influence sleep and/or body composition (Benca, 2005; Dijk et al., 2000; Reynor & Horne, 1995; Roberts et al., 2003; Roehrs & Roth, 1997; Stepanski & Wyatt, 2003; Van Reeth et al., 2000; Vgontzas et al., 2000); participants were instructed to base their answers to each question on a normal or typical week in the previous month.

Sleep quality was measured using the Pittsburgh Sleep Quality Index (PSQI) (Buysse et al., 1989), which assesses sleep quality in relation to seven components that are scored to provide a single sleep quality score. The PSQI has appropriate levels of internal consistency (Cronbach $\alpha=.72-.83$), test-retest reliability ($r=.85-.87$) and construct validity (Backhaus, Junghanns, Brooks, Riemann, & Hohagen, 2002; Beck, Schwartz, Towsley, Dudley, & Barsevick, 2004; Buysse et al., 1989; Carpenter & Andrykowski, 1998). Self-reported sleep durations were determined from the sleep duration component of the PSQI.

The Depression Anxiety Stress Scale (DASS) (Lovibond & Lovibond, 1995) was used to assess depression, anxiety and stress levels amongst respondents. The DASS provides contains 42 items addressing depression, anxiety and stress that are measured on four point scale (Lovibond & Lovibond, 1995). Previous studies have demonstrated appropriate levels of internal consistency for the depression (Cronbach $\alpha=.91–.97$), anxiety (Cronbach $\alpha=.81–.92$) and stress (Cronbach $\alpha=.89–.95$) subscales (Antony, Bieling, Cox, Enns, & Swinson, 1998; Brown, Chorpita, Korotitsch, & Barlow, 1997; Crawford & Henry, 2003; Lovibond & Lovibond, 1995).

Participants were also asked to indicate the type, frequency, amount and timing of their caffeine and alcohol consumption. Caffeine consumption was converted to total...
milligrams consumed each week based on the caffeine contents of different drinks specified in the questionnaire (FSANZ, 2007). Alcohol consumption was scored in relation to the number of standard drinks consumed each week, where one standard drink contained 10 grams of alcohol (NHMRC, 2001).

The number of long naps (greater than 20 minutes) per week was also assessed as it is associated with poor sleep (Fukuda & Ishihara, 2002). The number of nights in which sleep was disturbed due to noise, temperature (i.e. the sleep environment was either too hot or too cold) and/or discomfort was measured using separate items. Participants were also asked to indicate the number of nights per week they went to bed and woke up at approximately the same time; this information was used to indicate the regularity of sleep/wake cycles.

Questions from the Active Australia Survey (Armstrong, Bauman, & Davies, 2000) were used to estimate the number of minutes of walking, moderate activity and vigorous activity in the previous week. The time spent in each activity was then converted to kilocalories of energy expended per week. This conversion was achieved by multiplying each individual’s body weight by the MET (metabolic) value of each activity and the total duration per week in hours (Ainsworth et al., 2000). MET refers to the ratio of the metabolic rate for a particular activity compared to resting metabolic rate (Ainsworth et al., 2000). The kilocalorie value for each activity was then summed to provide an overall estimate of total energy expenditure per week.

The amount of time (in minutes) that each individual spent watching television/DVDs, working on a computer and playing video games each week was also measured. These activities were summed to provide an estimate of total screen time each week.
4.2.4 Statistical Analyses

The data were analysed using the partial least squares (PLS) approach to testing path models (see Figure 4.1). This path analytic approach allows for the interactions between multiple factors to be examined simultaneously and is suitable for exploratory data analyses. PLS is a parameter estimation method that is appropriate for small sample sizes, and it does not require the assumptions of multivariate normality and homogeneity of variance to be met (Chin, 1998).

PLS-Graph (Chin, 1998) was used to conduct the PLS analyses, which involved two phases. The first phase identified factors associated with sleep duration, sleep quality and obesity. In the second phase, these factors were entered into a path model (see Figure 4.1), and a bootstrapping procedure determined the precision and reliabilities of each path coefficient between two variables (Chin, 1998). This procedure involved a total of 100 sample sets being drawn from the original data set without replacement. The subsequent standard errors provided t statistics for each path coefficient specified in the model, which were then tested at 99 degrees of freedom (2-tailed) (Chin, 1998).

In the first phase of the analysis age, alcohol consumption, anxiety, caffeine consumption, depression, discomfort, napping, noise, physical activity, regular bedtimes, regular wake times, screen time, stress and temperature were entered into PLS-Graph to predict sleep duration and overall sleep quality. Age, alcohol consumption, anxiety, caffeine consumption, depression, physical activity, screen time, sleep duration, sleep quality and stress were then entered as predictors of obesity. For each step, non-significant factors were removed in a stepwise procedure until only the significant factor(s) remained.
On the basis of the results from the first phase, the second phase of the analysis tested a path model that incorporated: (1) factors significantly associated with short sleep durations; (2) factors significantly associated with overall sleep quality; (3) factors significantly associated with obesity; (4) factors associated with both sleep (quality and/or duration) and obesity; and (5) the presence of a significant relationship between sleep and obesity. In these analyses, the PSQI global sleep quality score was adjusted by removing the sleep duration component so that sleep duration and other sleep quality components did not explain shared variance in obesity.

4.3 Results

4.3.1 Phase 1: Factors associated with short sleep durations, poor sleep quality and/or obesity

In the presence of all variables, age \((t = 4.58, p < .001)\), discomfort \((t = 2.62, p = .010)\), irregular bedtimes \((t = 2.84, p = .006)\) and irregular wake times \((t = 2.34, p = .021)\) were significantly associated with sleep duration. There was a weak association between anxiety and sleep duration that did not reach significance \((t = 1.83, p = .073)\), but was still included in the subsequent analyses since mental health has been shown to influence sleep duration and obesity (Akerstedt et al., 2002; Wurtman, 1993). When the non-significant factors were removed in a stepwise manner, age \((t = 4.88, p < .001)\), anxiety \((t = 4.14, p < .001)\), discomfort \((t = 2.60, p = .011)\), irregular bedtimes \((t = 3.59, p = .001)\) and irregular wake times \((t = 2.12, p = .037)\) were associated with short sleep durations.

When all variables were included, age \((t = 3.54, p = .001)\), alcohol consumption \((t = 2.22, p = .029)\), anxiety \((t = 2.63, p = .010)\), depression \((t = 4.37, p < .001)\), discomfort \((t = 2.63, p = .010)\), noise \((t = 1.97, p = .052)\) and irregular bedtimes \((t =
2.21, p = .029) were significantly, or near significantly, associated with sleep quality. When the non-significant factors were removed in a stepwise manner, age (t = 4.83, p < .001), alcohol consumption (t = 2.52, p = .013), anxiety (t = 3.15, p = .002), depression (t = 5.24, p < .001), discomfort (t = 2.95, p = .004), noise (t = 2.35, p = .021) and irregular bedtimes (t = 2.20, p = .030) were negatively associated with sleep quality.

Age (t = 5.27, p < .001) and sleep duration (t = 2.70, p = .008) were the only variables significantly associated with obesity. When the non-significant factors were removed using a stepwise procedure, age (t = 7.56, p < .001) and reduced sleep durations (t = 3.24, p = .002) were positively associated with obesity.

4.3.2 Phase 2: Path Model Linking Sleep to Obesity

The factors that were significantly associated with short sleep durations, poor sleep quality and obesity were entered into a path model. The path model is shown in Figure 4.1 and the t statistics, β coefficients and associated p values for each path are provided in Table 4.2. Although non-significant in the preliminary analyses, physical activity and screen time were included as predictors of obesity to be consistent with previous research (Lahti-Koski et al., 2002). This model testing indicated that increased age, alcohol consumption, anxiety, depression, discomfort, noise and irregular bedtimes were significantly associated with poor sleep quality. Increased age, anxiety, caffeine consumption and discomfort, as well as irregular bedtimes and wake times, were significantly (or near significantly) associated with reduced sleep durations. These factors explained 39% of the variance in sleep quality and 21% of the variance in sleep duration. Increased age and reduced sleep durations were significantly associated with, and explained 23% of the variance in, obesity. Similar results were obtained when BMI and WC were examined separately.
4.4 Discussion

To the best of my knowledge, this study was the first to examine the relationships between chronic sleep restriction, obesity and relevant psychosocial variables using path analysis. This path model explained a considerable amount of the variation in both sleep duration (21%) and obesity (23%). The results indicate that short sleep durations were associated with obesity, which is consistent with previous findings (Bjorvatn et al., 2007; Fogelholm et al., 2007; Gangwisch, et al., 2005; Hasler et al., 2004; Ko et al., 2007; Kohatsu et al., 2006; Singh et al., 2005; Vioque et al., 2000; Vorona et al., 2005). This relationship remained significant after systematically identifying and controlling for a range of lifestyle, behavioural, psychological and environmental factors. The present study also found that overall sleep quality, as
Table 4.2. Path coefficients and t statistics for the models predicting BMI, WC and obesity.

<table>
<thead>
<tr>
<th>Regression Path</th>
<th>Obesity</th>
<th>t statistic</th>
</tr>
</thead>
<tbody>
<tr>
<td>IV</td>
<td>DV</td>
<td>β†</td>
</tr>
<tr>
<td>Age</td>
<td>Duration</td>
<td>-.35</td>
</tr>
<tr>
<td>Anxiety</td>
<td>Duration</td>
<td>-.20</td>
</tr>
<tr>
<td>Discomfort</td>
<td>Duration</td>
<td>-.13</td>
</tr>
<tr>
<td>Bedtime</td>
<td>Duration</td>
<td>.20</td>
</tr>
<tr>
<td>Wake time</td>
<td>Duration</td>
<td>-.13</td>
</tr>
<tr>
<td>Age</td>
<td>Quality</td>
<td>.26</td>
</tr>
<tr>
<td>Alcohol</td>
<td>Quality</td>
<td>.18</td>
</tr>
<tr>
<td>Anxiety</td>
<td>Quality</td>
<td>.19</td>
</tr>
<tr>
<td>Depression</td>
<td>Quality</td>
<td>.28</td>
</tr>
<tr>
<td>Discomfort</td>
<td>Quality</td>
<td>.18</td>
</tr>
<tr>
<td>Noise</td>
<td>Quality</td>
<td>.13</td>
</tr>
<tr>
<td>Bedtime</td>
<td>Quality</td>
<td>-.12</td>
</tr>
<tr>
<td>Activity</td>
<td>Obesity</td>
<td>.06</td>
</tr>
<tr>
<td>Age</td>
<td>Obesity</td>
<td>.40</td>
</tr>
<tr>
<td>Duration</td>
<td>Obesity</td>
<td>-.16</td>
</tr>
<tr>
<td>Quality</td>
<td>Obesity</td>
<td>.01</td>
</tr>
<tr>
<td>Screen time</td>
<td>Obesity</td>
<td>.06</td>
</tr>
</tbody>
</table>

† Positive β coefficients indicate a positive relationship and negative β coefficients indicate a negative relationship. * p < .05, ** p < .001

assessed by the PSQI, was not associated with obesity. This finding suggests that it might be the duration, not the quality, of sleep that is associated with obesity. As a consequence, the remainder of this paper focuses on the results associated with sleep duration.

There are several possible explanations for the observed relationship between chronic sleep restriction and obesity. Emerging evidence indicates that chronic sleep restriction contributes to obesity over several years (Gangwisch et al., 2005; Hasler et al., 2004; López-Garcia et al., 2008; Patel et al., 2006), possibly by altering the hormonal regulation of body weight (Spiegel et al., 2004b). It has also been suggested that chronic sleep restriction could lead to fatigue and a reduction in physical activity levels, which could also contribute to obesity (Fogelholm et al., 2007). However, there is also evidence that obesity contributes to chronic sleep restriction by increasing the risk of obstructive sleep apnoea (i.e. partial collapses of the upper airway during sleep...
caused by excess fat) and mental health problems, which lead to disturbed and reduced sleep (Roberts et al., 2003; Vgontzas et al., 2000). Therefore, it is argued that the relationship between chronic sleep restriction and obesity is bi-directional, and likely to involve complex psychosocial and physiological factors that require further investigation.

The path model also indicated that increased age was associated with obesity. This finding is not surprising given that body composition changes considerably with age (Flegal et al., 1998), possibly because of physiological (e.g. hormonal regulation of body weight) and/or lifestyle changes (e.g. increased work hours and family commitments). Importantly, the present results indicate that the relationship between chronic sleep restriction and obesity remained significant when controlling for the effects of age.

There was no evidence that that any of the psychosocial variables examined in this paper mediated the relationship between sleep and obesity. However, the path model indicated that increased age and anxiety levels, uncomfortable sleep environments and irregular sleep/wake cycles were significantly associated with short sleep durations (see Figure 4.1). Although the direction of causation cannot be inferred from the present data, it is argued that increased age, uncomfortable sleep environments and irregular sleep/wake cycles contribute to chronic sleep restriction, not vice-versa. For example, sleep durations decline with age (Reynor & Horne, 1995) possibly because of changes in sleep physiology and/or social and lifestyle changes associated with increased age. Irregular sleep/wake cycles could be caused by work or study demands and may contribute to chronic sleep restriction by affecting the circadian regulation of sleep (Dijk et al., 2000). An uncomfortable sleep environment caused by physical (e.g. an uncomfortable bed, pain or discomfort) and/or psychological factors
(e.g. stress, anxiety), could also contribute to chronic sleep restriction (Stepanski & Wyatt, 2003). Poor mental health (e.g. depression, anxiety) can potentially contribute to chronic sleep restriction by altering monoaminergic pathways in the brain that are involved in sleep and mood regulation (Benca, 2005). However, the relationship between chronic sleep restriction and mental health may be bi-directional, given that chronic sleep restriction can contribute to, or exacerbate, mental health problems (Van Reeth et al., 2000).

The factors identified in this path model could have potential practical applications for the treatment and prevention of obesity. For example, healthy sleep durations could be promoted through behavioural interventions targeting irregular sleep/wake cycles and uncomfortable sleep environments as well as psychological interventions aiming to minimise the impact of mental health problems on sleep patterns. These interventions could aid the treatment and prevention of obesity and warrant further investigation.

However, since the present study was cross-sectional, it was not possible to determine the direction of causation from the path analysis. This is a problem since many of the relationships identified in this paper could be bi-directional. Therefore, it is recommended that long-term prospective studies in representative samples are conducted to examine the relationships identified in this paper to address this issue. Furthermore, the variables in the present study, with the exception of BMI and WC, were based on self-report measures which may be inaccurate and/or subject to systematic biases in self-reporting. For example, individuals differ in how accurately they can recall their physical activity levels, and often overestimate the amount and intensity of physical activity (Montoye et al., 1996). Similarly, self-reported sleep data can also be inaccurate and are potentially influenced by age and gender (Reynor &
Horne, 1995). As a consequence, future studies should utilise a combination of subjective and objective measures for sleep, and the psychosocial factors examined in the present study. For example, sleep could be examined using a combination of self-report scales such as the PSQI and actigraphs (activity monitors), which provide more objective estimates of sleep patterns (Ancoli-Israel, 2005). Actigraphs could also be used in addition to self-report measures to assess physical activity levels (Leenders, Sherman, & Nagaraja, 2006). Additional measures of body composition such as bioelectrical impedance devices should also be utilised to complement the information derived from BMI and WC. Future research should also consider the role of other factors not included in the present study such as dietary intake (Spiegel et al., 2004b), specific measures of energy expenditure (e.g. metabolic rate), metabolic hormones, and work hours.

To the best of our knowledge, the present study represents the first attempt to test a path model linking sleep and obesity in the context of relevant psychosocial factors. The path model indicated that short sleep durations and age were significantly associated with obesity, and also identified possible determinants of short sleep durations which could have implications for the treatment and prevention of obesity. These findings provide an improved insight into the nature of the relationship between chronic sleep restriction and obesity that have potential practical implications for addressing the present obesity epidemic. Given that obesity and chronic sleep restriction are increasing health problems in society, there is a need to further investigate the relationships identified in this paper.
EMPIRICAL STUDY 2

The Relationship between Multiple Dimensions of Sleep Quality and Obesity

MANUSCRIPT SUBMITTED TO THE JOURNAL OF BEHAVIORAL MEDICINE IN OCTOBER 2007

5.1 Introduction

Average sleep durations have declined in recent decades and in the US the prevalence of chronic sleep restriction (sleep durations that are regularly less than seven hours but more than four hours per 24-hour period) increased from 14% to 40% between 1962 and 2005 (Dinges et al., 2005; Kripke et al., 2002; National Sleep Foundation, 2005). The precise factors contributing to the rise in chronic sleep restriction are unknown but are likely to reflect intentional and unintentional sleep restriction (Dinges et al., 2005). Several cross-sectional studies have recently reported associations between chronic sleep restriction and obesity (Bjorvatn et al., 2007; Fogelholm et al., 2007; Gangwisch et al., 2005; Hasler et al., 2004; Ko et al., 2007; Kohatsu et al., 2006; Singh et al., 2005; Taheri et al., 2004; Vioque et al., 2000; Vorona et al., 2005), and two prospective studies have found that chronic sleep restriction significantly predicts obesity over a period of several years (López-García et al., 2008; Patel et al., 2006). These data raise the intriguing possibility that chronic sleep restriction contributes to obesity, and could be targeted in the treatment and prevention of obesity (Flier & Elmquist, 2004; Knuston et al., 2007).

However, few studies have examined whether other components of sleep quality, such as sleep disturbances, sleep satisfaction and daytime sleepiness are also associated with obesity. Six studies have attempted to incorporate additional measures of sleep, but have been inconsistent in regards to the dimensions of sleep quality examined (Fogelholm et al., 2007; Gangwisch et al., 2005; Hasler et al., 2004; Kohatsu et al., 2006; Taheri et al., 2004; Vorona et al., 2005). Furthermore, these studies have typically assessed different components of sleep quality using self-report measures that are based on single items with unknown psychometric properties. As a consequence,
these studies do not provide a valid or clear insight into the nature of the relationship between other components of sleep quality and obesity.

Therefore, the aim of this chapter was to examine the relationship between multiple dimensions of sleep quality and obesity by utilizing the Pittsburgh Sleep Quality Index (PSQI) (Buysse et al., 1989), which is the most widely used scale to assess sleep quality. The PSQI consists of 19 items that are grouped into seven components (subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication and daytime dysfunction) and are summed to provide a single score of sleep quality (Buysse et al., 1989). The PSQI is able to distinguish between good and poor sleepers with high sensitivity and specificity, and also demonstrates appropriate levels of internal consistency (Cronbach $\alpha = .72 - .83$), test-retest reliability ($r = .85 - .87$) and construct validity (Backhaus et al., 2002; Beck et al., 2004; Buysse, et al., 1989; Carpenter & Andrykowski, 1998; Doi et al., 2000; Tsai, et al., 2005).

However, Cole et al. (2006) recently suggested that it might be more appropriate to score the PSQI in relation to three separate dimensions (i.e. sleep efficiency, perceived sleep quality and daily disturbances) as opposed to the single score. The validity of the three factor scoring method was supported by Aloba et al. (2007) and it has been suggested that this approach could improve the ability of the PSQI to assess the severity of specific sleep problems (Cole et al., 2006). This could have direct implications for determining the nature of the relationship between multiple components of sleep quality and obesity.

Therefore, the present chapter investigated the possible relationships between obesity and the three sleep quality dimensions of the PSQI identified by Cole et al. (2006). This involved performing a confirmatory factor analysis (CFA) to ensure that
the three-factor model proposed by Cole et al. (2006) was suitable for the present sample. Based on these results, the sleep quality dimensions were then entered into a multivariate model predicting obesity alongside factors such as depressed mood, physical activity levels, sex, snacking behaviour, television viewing, alcohol and caffeine consumption. These factors are associated with poor sleep quality and/or obesity and could potentially confound the association between sleep and obesity (Benca, 2005; Hill et al., 2003; Lahit-Koski et al., 2002; Roberts et al., 2003; Stepanski and Wyatt, 2003). A secondary aim of the present chapter was to examine possible for behavioural/lifestyle, mental health and environmental factors (as listed above) associated with each of the sleep quality dimensions. The identification of these factors is important as they could be modified as a way to improve sleep and may have implications for future obesity interventions that target sleep.

5.2 Method

5.2.1 Participants

Two hundred and sixty two adults (58% female) aged 18 to 35 years (M = 20.95, SD = 3.75) volunteered to participate in this study by responding to advertisements placed through a university course, and at local businesses and community groups. Participants self-reported an average of 7.63 hours sleep each night (SD = 1.18), and had an average PSQI total score of 5.44 (SD = 3.08). On the basis of body mass index (BMI) and waist circumference (WC), 65.6% of participants were considered lean (low health risk), 25.2% overweight (medium health risk) and 9.2% obese (high health risk) (see Table 5.1 for criteria). The protocol for this study was approved by the Human Research Ethics Committee at the University of Wollongong; informed consent was obtained from all participants.
5.2.2 Measures

*Anthropometric measures.*

Weight was measured to the nearest 0.1kg using electronic weight scales (Naleon, Australia), with the participant lightly clothed (e.g. t-shirt and shorts). Height was measured without shoes to the nearest 0.1cm using a stadiometer. BMI was calculated by dividing weight (kg) by height (m²). WC was measured to the nearest 0.5cm by placing a tape measure around each participant at the level of the umbilicus; the participant was standing erect but relaxed, and the measurement was taken at the end of normal expiration (American College of Sports Medicine, 2005). A combined index of BMI and WC was used to determine the level of obesity-related health risk (see Table 5.1). The advantage of this approach is that it provides a continuous score of obesity-related health risk (low, medium and high) that is more precise than BMI or WC alone (Zhu et al., 2004).

Table 5.1 Criteria used to classify individuals by obesity-related health risk (WHO, 2000; Zhu et al., 2004).

<table>
<thead>
<tr>
<th>Questionnaire</th>
</tr>
</thead>
<tbody>
<tr>
<td>Participants completed a questionnaire that consisted of the PSQI and items assessing environmental, psychological and behavioural factors that have been associated with sleep quality and/or obesity; for each of these items, participants were instructed to base their answers on a normal or typical week during the preceding month.</td>
</tr>
</tbody>
</table>
Depressed mood was examined using the Depression Anxiety Stress Scale-42 (DASS) (Lovibond & Lovibond, 1995). The DASS is widely used in samples of Australian adults and contains 42 items that address depression, anxiety and stress (Lovibond & Lovibond, 1995). The depression scale contains 14 items that assesses dysphoria, hopelessness, devaluation of life, self-deprecation, lack of interest/involvement, anhedonia, and inertia (Lovibond and Lovibond, 1995), and has appropriate levels of internal consistency (Cronbach \( \alpha = .91 – .97 \)) (Antony et al., 1998; Brown et al., 1997; Crawford & Henry, 2003; Lovibond & Lovibond, 1995). Consistent with Lovibond and Lovibond (1995), the depression subscale was utilised as a continuous score of depressed mood (higher scores indicate increased severity of depressed mood) in the subsequent analyses.

Participants were asked to indicate the type, frequency, amount and timing of their caffeine and alcohol consumption. Caffeine consumption was converted to milligrams of consumption per week based on the caffeine contents of the drinks specified in the questionnaire (Food Standards Australia and New Zealand, 2007). Alcohol consumption was scored in relation to the number of standard drinks consumed each week, where one standard drink contained 10 grams of alcohol (National Health and Medical Research Council, 2001).

Questions from the Active Australia Survey (Armstrong et al., 2000) were used to estimate the number of minutes of walking, moderate activity and vigorous activity in the previous week. The Active Australia Survey is used in Australian Government surveys to assess the physical activity levels of Australian adults and has adequate levels of reliability (Cronbach \( \alpha = .71 – .86 \)) (Australian Institute for Health and Welfare, 2003). The time spent in each activity was converted to kilocalories of energy expended per week by multiplying each individual’s body weight by the MET value.
(this represents the ratio of active metabolic rate compared to rest) of each activity and the total duration per week in hours (Ainsworth et al., 1993; Montoye et al., 1996). The kilocalorie value for each activity was then summed to provide an overall estimate of total energy expenditure per week.

Participants were also asked to indicate the number of days per week and the hours per day that they watched TV/DVDs, played video or computer games, and used a computer for work or study (these were assessed in separate items). The amount of time engaged in each of these activities per week was summed to provide an estimate of total ‘screen time’ each week. Participants were also asked to indicate the number of nights per week they snacked on food late in the evening after dinner.

The number of nights in which sleep was disturbed due to environmental noise and discomfort was measured by separate items. Participants were also asked to indicate the number of nights per week they went to bed at approximately the same time (i.e. within 15 to 30 minutes). Participants were then categorised as having regular bedtimes (i.e. the same bedtimes 4 or more nights a week) or irregular bedtimes (i.e. same bedtimes 3 or less nights a week).

5.2.3 Statistical analyses.

Confirmatory factor analyses (CFA) were performed to assess the factor structure of the three-factor scoring method for the PSQI suggested by Cole et al. (2006). This step is required because the factor structure identified by Cole et al. (2006) was obtained on a sample of aged 60 years and over, and may not be relevant to the present sample of adults aged 18 – 35 years. Once the factor structure of the PSQI was identified from the CFA, a general linear modelling approach was used to analyse these data. This involved testing a model where age, alcohol consumption, depression, sex,
physical activity levels and the PSQI dimensions identified in the CFA, were entered as predictors of obesity-related health risk (i.e. the combination of BMI and WC). Additional models were then tested that involved age, alcohol consumption, caffeine consumption, depression, discomfort at night, environmental disturbances at night, sex, obesity, regularity of bedtimes and screen time entered as predictors of each sleep quality dimension identified in the CFA.

5.3 Results

5.3.1 CFA

The three-factor model suggested by Cole et al. (2006) had a reasonable model fit as indicated by the root mean square error of approximation (RMSEA) index of .066 and the comparative fit index (CFI) of .953, but the chi-square value was significant ($\chi^2(11) = 23.591, p = .015$). Figure 5.1a indicates that the sleeping medication use component loaded poorly on the perceived sleep quality dimension; this is not surprising given that only 9.5% of participants in the present sample reported sleep medication use. There was also evidence of overestimation between the perceived sleep quality and daily disturbances dimensions, suggesting that it might be more appropriate to merge these two dimensions into a single dimension.

Therefore, the sleep disturbances component was grouped with perceived sleep quality, which is justified since all of these factors address poor sleep quality during the sleep episode. However, because the level of daytime dysfunction (i.e. sleepiness) is the result of sleep quality at night (Roehrs et al., 2005), it was decided to leave this as a separate dimension. On the basis of these changes, a two factor model of the PSQI was then tested (note: daytime dysfunction could not be included in this model given that it was comprised of only one component) and led to an improved model fit as indicated by
the RMSEA (.052), CFI (.987) and chi-square ($\chi^2(4) = 6.802, p = .147$). Therefore, the PSQI was scored in relation to three dimensions as presented in Figure 5.1b, and with the daytime dysfunction dimension as an additional dimension.

Figure 5.1 a. Factor loadings for three-factor model proposed by Cole et al. (2006); b Factor loadings for a two-factor model
5.3.2 General Linear Modelling

As shown in Table 5.2, ‘Sleep duration/efficiency’ was the only variable significantly associated with obesity (p = .004), but there was also a weak association between ‘daytime dysfunction’ and obesity which did not reach statistical significance (p = .063). This model explained 5.2% of the variance in obesity.

Table 5.2 Factors entered into the univariate model predicting obesity. β coefficients and significance levels are provided for each variable.

<table>
<thead>
<tr>
<th></th>
<th>$\beta$</th>
<th>$p$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>.926</td>
<td>.003</td>
</tr>
<tr>
<td>Sex = 0</td>
<td>.004</td>
<td>.969</td>
</tr>
<tr>
<td>Sex = 1</td>
<td>0</td>
<td>-</td>
</tr>
<tr>
<td>Duration/efficiency</td>
<td>.129</td>
<td>.004</td>
</tr>
<tr>
<td>Perceived quality</td>
<td>-.049</td>
<td>.158</td>
</tr>
<tr>
<td>Daytime dysfunction</td>
<td>.142</td>
<td>.063</td>
</tr>
<tr>
<td>Snack</td>
<td>.011</td>
<td>.565</td>
</tr>
<tr>
<td>Depression</td>
<td>-.002</td>
<td>.849</td>
</tr>
<tr>
<td>Screen time</td>
<td>.000</td>
<td>.900</td>
</tr>
<tr>
<td>Age</td>
<td>.011</td>
<td>.353</td>
</tr>
<tr>
<td>Physical activity</td>
<td>.001</td>
<td>.081</td>
</tr>
<tr>
<td>Alcohol</td>
<td>.004</td>
<td>.297</td>
</tr>
<tr>
<td>$R^2$</td>
<td></td>
<td>.052</td>
</tr>
</tbody>
</table>

Depressed mood (p < .001), discomfort (p = .005), irregular bedtimes (p = .012) and obesity (p = .019) were significantly associated with sleep duration/efficiency (Table 5.3). Alcohol consumption (p = .012), depressed mood (p < .001), discomfort (p < .001), environmental disturbances (p < .001) and irregular bedtimes (p = .026) were significantly associated with perceived sleep quality (Table 5.3). These models explained 20.9% of the variation in sleep duration/efficiency and 43.2% of the variation in perceived sleep quality.

Since daytime dysfunction is a consequence of poor night-time sleep (Roehrs et al., 2005), ‘perceived sleep quality’ and ‘sleep duration/efficiency’ were entered into the model predicting daytime dysfunction alongside the other predictors. The results indicated that ‘perceived sleep quality’ (p = .026) and depressed mood (p < .001) were
the only factors significantly associated daytime dysfunction (see Table 5.4). This model explained 18.4% of the variance in daytime dysfunction.

Table 5.3 Factors entered into the univariate models predicting duration/efficiency and perceived sleep quality. β coefficients and significance levels are provided for each variable.

<table>
<thead>
<tr>
<th></th>
<th>Duration/Efficiency</th>
<th>Perceived Quality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>-.584</td>
<td>2.679</td>
</tr>
<tr>
<td>Sex = 0</td>
<td>.148</td>
<td>.319</td>
</tr>
<tr>
<td>Sex = 1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Bedtime = 0</td>
<td>.448</td>
<td>.464</td>
</tr>
<tr>
<td>Bedtime = 1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Alcohol</td>
<td>.006</td>
<td>.017</td>
</tr>
<tr>
<td>Obesity</td>
<td>.297</td>
<td>-.123</td>
</tr>
<tr>
<td>Caffeine</td>
<td>.001</td>
<td>.648</td>
</tr>
<tr>
<td>Disturbances</td>
<td>-.019</td>
<td>.236</td>
</tr>
<tr>
<td>Discomfort</td>
<td>.168</td>
<td>.369</td>
</tr>
<tr>
<td>Depressed mood</td>
<td>.051</td>
<td>.095</td>
</tr>
<tr>
<td>Age</td>
<td>.017</td>
<td>-.032</td>
</tr>
<tr>
<td>Snacks</td>
<td>.004</td>
<td>.909</td>
</tr>
<tr>
<td>R²</td>
<td>.209</td>
<td>.432</td>
</tr>
</tbody>
</table>

Table 5.4 Factors entered into the univariate model predicting daytime dysfunction. β coefficients and significance levels are provided for each variable.

<table>
<thead>
<tr>
<th></th>
<th>β</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>.868</td>
<td>.001</td>
</tr>
<tr>
<td>Sex = 0</td>
<td>-.003</td>
<td>.970</td>
</tr>
<tr>
<td>Alcohol</td>
<td>.003</td>
<td>.368</td>
</tr>
<tr>
<td>Duration/efficiency</td>
<td>.054</td>
<td>.186</td>
</tr>
<tr>
<td>Perceived quality</td>
<td>.066</td>
<td>.026</td>
</tr>
<tr>
<td>Age</td>
<td>-.017</td>
<td>.118</td>
</tr>
<tr>
<td>Obesity</td>
<td>.058</td>
<td>.357</td>
</tr>
<tr>
<td>Depressed mood</td>
<td>.033</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>R²</td>
<td></td>
<td>.184</td>
</tr>
</tbody>
</table>

5.4 Discussion

The present results indicate that scores on the sleep duration/efficiency dimension of the PSQI were positively associated with obesity after controlling for multiple potentially confounding variables. This suggests that chronic sleep restriction and poor sleep efficiency were associated with obesity, which is consistent with previous studies (Bjorvatn et al., 2007; Ko et al., 2007; Kohatsu et al., 2006; Kripke et al., 2002; Singh et al., 2005; Taheri, 2006; Vioque et al., 2000; Vorona et al., 2005).
Chronic sleep restriction could contribute to obesity by altering the hormonal regulation of body weight (Knuston et al., 2007). Therefore, it has been suggested that chronic sleep restriction could potentially be targeted as a modifiable risk factor alongside diet and exercise to treat and prevent obesity (Flier & Elmquist, 2004). However, it should also be noted that obesity is associated with conditions such as obstructive sleep apnea (OSA) that can lead to chronic sleep restriction (Young et al., 2005). This suggests that the relationship between chronic sleep restriction and obesity is likely to be bi-directional.

The present results indicate that depressed mood, discomfort, irregular bedtimes and obesity were significantly associated with ‘poor sleep duration/efficiency’. To the best of our knowledge, only Fogelholm et al. (2007) have attempted to identify possible determinants of sleep duration. Their results indicated that age, depressed mood, physical activity and obesity were associated with chronic sleep restriction and disturbed sleep. However, the present chapter also demonstrated that the sleep environment (i.e. discomfort) and sleep-related behaviours (i.e. regularity of bedtimes) were associated with sleep duration/efficiency. It seems reasonable to assume that irregular bedtimes and discomfort contribute to poor sleep duration/efficiency, not vice-versa, whereas obesity is likely to be both a cause and a consequence of ‘poor sleep duration/efficiency’ as noted above. The relationship between depressed mood and ‘poor sleep duration/efficiency’ may also be bi-directional, reflecting shared monoaminergic pathways in the brain (Benca, 2005; Saper et al., 2001). It may be possible to improve sleep durations by modifying these factors through behavioural and psychological interventions; this could aid the treatment and prevention of obesity and warrants further investigation.
There was no evidence of a direct relationship between perceived sleep quality and obesity, but there was an association between daytime dysfunction and obesity that approached significance and warrants discussion. The daytime dysfunction component of the PSQI refers to daytime sleepiness and enthusiasm for daily activities (Buysse et al., 1989). This finding is consistent with some previous studies that have reported associations between excessive daytime sleepiness and obesity (Dixon et al., 2007; Vgontzas et al., 1998). The mechanisms underlying this link are unclear, but it has been suggested that obesity could cause circadian and/or metabolic disturbances that lead to under-arousal during the day (e.g. sleepiness) and increased arousal at night (Vgontzas et al., 1998). However, we also contend that daytime dysfunction could contribute to obesity by leading to reductions in physical activity levels due to fatigue or a lack of motivation, or promote the over-consumption of energy dense foods as a way to combat sleepiness.

Poor perceived sleep quality and depressed mood were associated with higher levels of daytime dysfunction, which is consistent with previous findings (Bixler et al., 2005; Dixon et al., 2007; Roehrs et al., 2005). For example, poor sleep quality at night is a predictor of sleepiness and fatigue on the following day (Roehrs et al., 2005), whilst sleepiness and a lack of enthusiasm for daily activities are symptomatic of depressed mood (APA, 2000). It is also feasible that increased levels of daytime dysfunction could contribute to depressed mood, but this requires further investigation. We recommend that future research is conducted to further examine these relationships given that poor daytime dysfunction could have implications for obesity. Since the PSQI consists of only two items assessing daytime dysfunction, future research could benefit from additional scales such as the Epworth Sleepiness Scale, which focus specifically on daytime sleepiness (Johns, 1991).
A limitation of the present chapter is that the cross-sectional design does not allow for the cause-and-effect to be determined. This is particularly a problem given that many of the relationships identified in this paper could be bi-directional. Therefore, it is recommended that future studies examine the longitudinal relationships between the variables identified in this paper to address this issue. The present chapter also recruited participants using advertisements, which may have influenced the characteristics of this sample. For example, it is possible that individuals sensitive about their weight (e.g. obese individuals and females) were less likely to participate in this study. This could explain the relatively low proportion of overweight and obese individuals who participated in this study. As a consequence, future studies are needed to examine the relationships identified in this chapter on samples that are more representative of the general population. A further limitation is that all of the variables apart from BMI and WC were based on self-report measures which could be inaccurate or subject to systematic biases in reporting. This may explain why physical activity and screen time were not associated with obesity as would be expected (Fogelholm et al., 2007; Hill et al., 2003; Jeffery and French, 1998) and that the model accounted for a relatively small amount of the variance in obesity (5%). A better understanding of how sleep quality dimensions interact with other risk factors for obesity could be achieved using more detailed and/or objective measures of physical activity, screen time and daytime sleepiness.

The present chapter adopted a novel approach and tested a three-factor model of the PSQI. On the basis of these results, the relationship between three dimensions of sleep quality and obesity were examined. The findings indicate that reduced sleep duration/efficiency and increased levels of daytime dysfunction could be potential risk factors for obesity, and warrant further investigation. Furthermore, the results also
identified possible determinants of these sleep dimensions, which could potentially be targeted as a way to improve sleep quality. Such interventions may have implications for combating the present obesity epidemic. These results are preliminary but warrant further investigation, especially since obesity, chronic sleep restriction and sleep disturbances are increasingly common health problems in society.
CHAPTER 6

EMPIRICAL PAPER 3

A Preliminary Investigation of the Effects of Short-Term Sleep Restriction on Energy Regulatory Hormones and Energy Expenditure

MANUSCRIPT SUBMITTED TO THE JOURNAL OF CLINICAL ENDOCRINOLOGY AND METABOLISM, MARCH 2008
6.1 Introduction

Approximately 40% of US adults currently experience chronic sleep restriction (sleep durations less than seven hours a night), which is an increasingly common condition in modern society (National Sleep Foundation, 2005). Mounting evidence indicates that chronic sleep restriction contributes to obesity (López-García et al., 2008; Patel, et al., 2006), and this may have important implications for combating the present obesity epidemic. However, the mechanisms linking chronic sleep restriction to obesity are unclear. One possibility is that sleep restriction contributes to obesity by altering neuroendocrine hormones involved in energy balance regulation (Spiegel et al., 2004b).

Energy balance refers to the long-term balance between energy intake (diet) and expenditure (basal metabolic rate [BMR], physical activity, diet induced thermogenesis) and is physiologically regulated to ensure a constant body weight (Spiegelman & Flier, 2001). Neuroendocrine hormones arising from the gastrointestinal tract (ghrelin, PYY, insulin, GLP-1) and adipose tissue (leptin, adiponectin) are critical in the short and long-term regulation of energy balance (Spiegelman & Flier, 2001; Wisse et al., 2007). Although energy balance is tightly regulated, genetic, environmental and psychosocial factors can disrupt the hormonal regulation of energy balance and promote obesity (Spiegelman & Flier, 2001).

The aim of the present chapter was to investigate the effects of sleep restriction on the adipose tissue hormones leptin and adiponectin, and the gastrointestinal hormones insulin, ghrelin, PYY and GLP-1, which are involved in regulating energy balance. This chapter also investigated the effects of sleep restriction on energy expenditure (EE) using indirect calorimetry. This novel approach provides a more comprehensive insight into plausible mechanisms linking sleep restriction to obesity.
6.2 Method

6.2.1 Participants

Ten males aged 19 to 23 years (M = 20.40, SD = 1.55), with self-reported habitual sleep durations between seven and nine hours per night participated in this study. All participants were non-obese based on BMI (M = 22.90, SD = 3.05) and WC measurements (M = 82.65cm, SD = 6.07). The protocol for this study was approved by the Human Research Ethics Committee at the University of Wollongong; informed consent was obtained from all participants.

6.2.2 Measures

Adiponectin, ghrelin, PYY, leptin, insulin and GLP-1 were assayed in duplicate using Millipore Lincoplex kits and a Luminex IS100 instrument (Lincoplex, USA). The intra-assay variation for the kit analysing ghrelin, PYY, insulin, leptin and GLP-1 was <11%, with sensitivity’s of 157.2pg/ml (leptin), 5.2pg/ml (GLP-1), 44.5pg/ml (insulin), 1.8pg/ml (ghrelin) and 8.4pg/ml (PYY). The assay kit for adiponectin had an intra-assay variation of 1.4-7.9% and a sensitivity of 80.3pg/ml.

EE was measured continuously for 10 hours between 2130 h and 0730 h by indirect calorimetry using a WRC (a closed room that monitors oxygen consumption and carbon dioxide production using gas analyzers) (Levine, 2005). The collected data were converted to EE in kilocalories using Weir’s formula (Weir, 1949) and were averaged to provide estimates of EE at 1 hour intervals (see Figure 6.1). BMR was determined from EE data collected under standardised conditions for a 30-40 minute period between 0630 h and 0730 h after 10 hours fasting (Levine, 2005).

Visual analogue scales (VAS) were used to monitor changes in ratings of hunger, the amount participants perceived they could eat (referred to as ‘eat’) and satiety (Stubbs
et al., 2000). Each item consisted of a 10cm line with two opposing statements at either end (e.g. ‘I am not hungry at all’/‘I have never been hungrier’). Participants responded by placing a mark on the line relative to how they felt at that particular time.

6.2.3 Procedure

To simulate the effects of chronic sleep restriction, this study was conducted over a period of four consecutive nights, with a baseline night (eight hours sleep), two nights of sleep restriction (five hours sleep) and a recovery night (approximately nine hours sleep). At baseline and sleep restriction, participants reported to the laboratory at approximately 1930h, completed the VAS, were served an evening meal (energy content: 1546-1992KJ) and entered the WRC. Participants were permitted to engage in light activities (e.g. watching television, reading, studying) but were instructed to sleep between 2230h and 0630h at baseline and between 0130h and 0630h on the sleep restriction nights; this was monitored by a supervisor. Upon waking, participants were instructed to lie in the supine position for 30-40 minutes to allow for BMR data collection. Participants then exited the WRC to complete the VAS and had blood drawn on each morning (except after the first night of sleep restriction). Participants were provided with breakfast consisting of 30g of cereal with full-cream milk (energy content: 475KJ) and left the laboratory to engage in their normal daily activities, but were asked to keep physical activity levels constant across the four study days. At recovery, participants reported to the laboratory at approximately 1930h to complete the VAS, were served dinner (as above), and slept at home for a self-reported period of approximately nine hours. They returned to the laboratory the following morning to complete the VAS and had blood drawn.
6.2.4 Statistical Analyses

The data were analysed using permutation tests, which are recommended for small sample sizes (n ≤ 10), particularly when normality is violated (Todman & Dugard, 2001). Matched pairs t-tests using permutation methods examined differences in hormone levels and appetite ratings between baseline and sleep restriction, and between sleep restriction and recovery sleep. Since ghrelin and PYY exert opposing effects on appetite (Batterham et al., 2002; Wren et al., 2001), a ratio of ghrelin-to-PYY was calculated by dividing ghrelin by PYY; a ratio of ‘eat’-to-satiety was also calculated by dividing ratings of ‘eat’ by ‘satiety’. These ratios were subject to the above analyses. Correlations calculated using permutation procedures examined relationships between changes in hormone levels and appetite ratings from baseline to sleep restriction, and from sleep restriction to recovery sleep. Matched pairs t-tests using permutation methods also compared EE between the baseline and sleep restriction nights at each time point (see Figure 6.1), and also examined changes in EE on the sleep restriction nights (see Figure 6.2a, b).

Since the present study had low statistical power caused by a small sample, effect sizes (Cohen’s d) were calculated for each matched-pair comparison to provide an indication of the magnitude of each effect. Results indicating a small effect or greater (i.e. d ≥ .20) are reported regardless of significance as these indicate potentially ‘real’ effects that may warrant investigation in future studies. The t-statistics, p values and effect sizes for each matched pair comparison are listed in Table 6.1. There is considerable debate as to whether it is appropriate for Bonferroni adjustments to be performed when conducting multiple comparisons. For example, Pernerger (1998, 1999) argues that multiple comparisons are inappropriate if the data do not consist of repeated observations, as it increases the risk of a type-II error and confounds statistical
inference. As a consequence, Bonferroni adjustments were not performed for this study and p values < 0.05 are considered statistically significant.

### 6.3 Results

BMR did not differ between the baseline and sleep restriction nights, but EE was lower on the second night of sleep restriction at 0130 h \( (t(5) = -1.981, \ p = .093, \ d = .44) \) and 0630 h \( (t(5) = -2.189, \ p = .095, \ d = .49) \) compared to the first night of sleep restriction (see Figure 6.1). Furthermore, EE remained constant between 2130 h and 0130 h on the first night of sleep restriction, but declined significantly between 2230 h and 0130 h on the second night of sleep restriction (see Figure 6.2a, b).

![Metabolic Rate](image.png)

**Figure 6.1.** Changes in EE across the baseline and sleep restriction nights between 2130h and 0730h. Solid lines indicate EE whilst participants are awake, whereas dotted lines indicate EE whilst asleep.
Compared to baseline, sleep restriction led to significant increases in leptin and the eat-satiety ratio, and a near significant increase in insulin (see Table 6.1). One participant did not return for the recovery sleep condition; therefore data were available for nine participants for the restriction-recovery comparisons. Compared to recovery sleep, sleep restriction was associated with significant increases in the ghrelin-to-PYY ratio and the eat-satiety ratio, a significant reduction in satiety levels and a near significant reduction in PYY (see Table 6.1). There was also a trend towards an
Table 6.1 Comparison of hormone levels and appetite ratings between the baseline and sleep restriction conditions (N = 10) and between the sleep restriction and recovery conditions (N = 9).

<table>
<thead>
<tr>
<th></th>
<th>Baseline Sleep (7.3 hours) versus Sleep Restriction (4.9 hours)</th>
<th>Sleep Restriction (4.8 hours) versus Recovery Sleep (9.2 hours)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>Restriction</td>
</tr>
<tr>
<td>Adiponectin*</td>
<td>10.93</td>
<td>10.60</td>
</tr>
<tr>
<td>Ghrelin*</td>
<td>12.02</td>
<td>13.73</td>
</tr>
<tr>
<td>GLP-1*</td>
<td>43.91</td>
<td>46.52</td>
</tr>
<tr>
<td>Insulin*</td>
<td>516.18</td>
<td>590.85</td>
</tr>
<tr>
<td>Leptin*</td>
<td>3.27</td>
<td>3.84</td>
</tr>
<tr>
<td>PYY*</td>
<td>77.75</td>
<td>78.40</td>
</tr>
<tr>
<td>Ghrelin-PYYc</td>
<td>1.70</td>
<td>1.96</td>
</tr>
<tr>
<td>Eat*</td>
<td>62.50</td>
<td>71.80</td>
</tr>
<tr>
<td>Hunger*</td>
<td>58.80</td>
<td>71.20</td>
</tr>
<tr>
<td>Satiety*</td>
<td>24.40</td>
<td>17.20</td>
</tr>
<tr>
<td>Eat-satietyc</td>
<td>5.07</td>
<td>8.73</td>
</tr>
</tbody>
</table>
increase in ghrelin, but this did not reach significance. There was a positive correlation between the ghrelin-PYY and eat-satiety ratios that approached significance ($r (5) = .772, p = .056$), suggesting that the ghrelin-PYY ratio accounted for 60% of the variance in the eat-satiety ratio.

6.4 Discussion

The present results indicate striking differences in EE on two consecutive nights of sleep restriction. For example, EE was lower on the second night of sleep restriction compared to the first night of sleep restriction prior to sleep and upon waking (see Figure 6.2a, b); although non-significant, these differences represented medium effects ($ds = .44 - .49$) that may be clinically important. Furthermore, EE was constant on the first night of sleep restriction prior to sleep, but declined significantly on the second night of sleep restriction ($ds = .48 - .66$). This could be the result of increased fatigue with continued sleep restriction, which possibly leads to lower EE. These novel results provide, to the best of our knowledge, the first indication that sleep restriction has the potential to affect EE. However, EE was measured for 10 hours and hence the present results do not provide an indication of the effects of sleep restriction on 24 hour EE. As a consequence, there is a need for future studies to extend on these preliminary findings and examine the effects of sleep restriction on 24 hour EE.

Sleep restriction was also associated with increased ghrelin, which did not reach statistical significance but is consistent with previous findings (Spiegel et al., 2004b). Sleep restriction also led to a near significant reduction in PYY, which has not been previously examined. Since ghrelin stimulates and PYY inhibits food intake by acting on receptors in the arcuate nucleus of the hypothalamus (Batterham et al., 2002; Wren et al., 2001), the obtained results suggest that sleep restriction could promote increased
food intake by altering these hormones. This assertion is supported by the strong positive correlation between changes in the ghrelin-PYY and ‘eat-satiety’ ratios. The changes in ghrelin and PYY could reflect increased sympathetic nervous system activity, which has been associated with sleep restriction (Irwin, Thompson, Miller, Gillin, & Ziegler, 1999) and stimulates ghrelin (Heath, Jones, Frayn, & Roberston, 2004) and inhibits PYY release (Koda et al., 2005). However, the precise mechanisms leading sleep restriction to alterations in these hormones need to be clarified.

Sleep restriction did not alter GLP-1 or adiponectin, but leptin and insulin were elevated with sleep restriction compared to baseline. These results are suggestive of reduced energy intake and increased EE, which is inconsistent with previous findings (Guilleminault et al., 2003; Spiegel et al., 2004a, b). However, these could be aberrant findings reflecting methodological issues relating to the short period of sleep restriction (i.e. 2 nights) and/or the timing of blood sampling. This explanation is possible since the changes in leptin and insulin were unrelated to changes in hunger, appetite and/or EE.

The present results were obtained under conditions that resembled free-living conditions (whilst energy intake was kept constant), which contrasts previous studies where participants were confined to continuous bed rest, 24-hour blood sampling and overnight polysomnography recordings (Spiegel et al., 2004a, b). Therefore, the present results provide a preliminary, but useful, insight into the potential mechanisms linking sleep restriction to altered energy balance and obesity. However, the small sample size limited the external validity of these findings and also reduced the statistical power. Furthermore, since blood was sampled at one time point, it was not possible to establish the effects of sleep restriction on the 24-hour profile of energy regulatory hormones measured in this study.
Nevertheless, the present findings demonstrate that sleep restriction over a period of two nights leads to striking changes in EE and the hormonal regulation of energy balance in a manner that could promote a positive energy balance and contribute to obesity over time. These results are preliminary, but provide a promising indication of potential physiological pathways linking sleep restriction to obesity; these warrant further investigation in long-term prospective studies of free-living individuals. There is an urgency to this research since chronic sleep restriction could potentially be targeted to combat the growing obesity epidemic.
CHAPTER 7

SUMMARY AND CONCLUSIONS
7.1 Summary

This chapter summarises the major findings from the two literature reviews and the three empirical chapters, and discusses the implications these findings have for understanding the relationship between chronic sleep restriction and obesity. Literature review 1 (Chapter 2) comprehensively examined available data indicating an association between chronic sleep restriction and obesity. This review focused specifically on the possible causes of chronic sleep restriction at a population level, as well as the potential physiological pathways linking chronic sleep restriction to obesity. These issues are critical in understanding how chronic sleep restriction and obesity are related, and have been overlooked in existing literature. Based on epidemiological data from the past 40 years, it was hypothesised that the recent rise in chronic sleep restriction could be the result of an increase in mental health problems, increased work demands and increased use of specific prescription medications. It was then hypothesised that chronic sleep restriction could lead to obesity by altering metabolic, neuroendocrine and neural pathways involved in the regulation of energy balance. These pathways were then summarised in an integrated schematic representation, providing what I believe is the first theoretical model linking chronic sleep restriction to obesity. It is recommended that future research examine the pathways identified in this model to provide a clearer insight into how chronic sleep restriction and obesity are related.

Literature review 2 (Chapter 3) identified the major methodological limitations of previous epidemiological studies examining the relationship between chronic sleep restriction and obesity. It was argued that previous studies have typically relied on inadequate techniques to assess sleep and body composition, and this could limit our understanding of how chronic sleep restriction and obesity
are related. As a consequence, this review outlined the methods that should be used in future studies to assess sleep patterns and obesity in this context. It was recommended that sleep should be measured using a combination of actigraphy and subjective scales such as the PSQI and the ESS, whereas body composition should be assessed using a combination of BMI, WC and bioelectrical impedance. It was also argued that future studies should control for potential confounding variables such as age, mental health (e.g. stress and depression), physical activity, alcohol and caffeine consumption, and sedentary activities (e.g. television viewing). Finally, this review recommended that the relationship between chronic sleep restriction and obesity should be examined by carefully designed long-term prospective studies that incorporate the methodological limitations outlined in this chapter and examine the pathways identified in chapter 2.

The purpose of empirical study 1 (chapter 4) was to test a path model linking chronic sleep restriction to obesity, based on the model identified in chapter 2 and the methodological considerations provided in chapter 3. Three hundred and twenty five adults aged 18 to 87 years participated in this study and completed the PSQI as well as items assessing lifestyle, behavioural, environmental and psychological factors associated with sleep and/or obesity; body composition was assessed using a combination of BMI and WC. The relationships between these variables were assessed simultaneously using path analysis. The results indicated that short sleep durations were associated with obesity, which is consistent with previous research. However, age, anxiety, uncomfortable sleep environments and irregular sleep/wake cycles were significantly associated with short sleep durations. It is possible that these factors could be targeted in behavioural interventions as a
way to increase sleep durations, which may have beneficial implications for obesity.

Empirical study 2 (chapter 5) examined the relationship between multiple dimensions of sleep quality and obesity in 262 adults (58% female) aged 18 to 35 years. This involved utilising a three-factor scoring method for the PSQI, which assesses ‘sleep duration/efficiency’, ‘perceived sleep quality’ and ‘daytime dysfunction’. Scores on the sleep duration/efficiency component were associated with obesity, providing further evidence for an association between sleep duration and obesity. However, the results also indicated that increased levels of daytime dysfunction (i.e. sleepiness) were associated with obesity. It is possible that daytime dysfunction could contribute to obesity by leading to a reduction in physical activity levels and the over consumption of energy dense foods. However, this relationship may be bi-directional since daytime dysfunction could be a consequence of obesity. This chapter also identified the psychosocial factors associated with each of the three sleep quality dimensions, which could potentially be targeted to improve sleep quality and have implications for the treatment and prevention of obesity. Importantly, these results demonstrate the importance of assessing multiple components of sleep quality.

Finally, empirical study 3 (chapter 6) examined the effects of two nights of sleep restriction on neuroendocrine hormones involved in the regulation of energy balance (as identified in chapter 2) and energy expenditure (EE) in 10 healthy males aged 18 to 23 years. Sleep restriction led to a reduction in EE and an increase in ghrelin levels, a reduction in PYY and an increase in the ratio of ghrelin to PYY. The nature of these changes suggests that sleep restriction could promote a positive energy balance by increasing food intake relative to EE. These novel
findings therefore provide evidence that sleep restriction could contribute to obesity by altering the pathways involved in the regulation of energy balance, which is consistent with the pathways identified in Chapter 2.

### 7.2 Limitations and Suggestions for Future Research

The empirical and theoretical work in this thesis extends on previous research and provides novel insights into the relationship between chronic sleep restriction and obesity. However, there are some general limitations of the three empirical studies that warrant consideration and would need to be addressed in future research. In particular, study 1 and study 2 were both cross-sectional and hence do not allow for causal inferences to be drawn. Therefore, the results do not indicate that chronic sleep restriction causes obesity, but rather that the two variables are related. This needs to be acknowledged because as noted in Chapter 2, it is likely that the relationship between chronic sleep restriction and obesity is bi-directional. Furthermore, the majority of variables in these studies were assessed using self-report measures, which may have affected the accuracy of the data. Finally, there are other factors such as diet, shift-work, work hours and EE that were not directly assessed in these studies but could also influence the relationship between chronic sleep restriction and obesity and would need to be addressed.

The findings from study 3 are novel and have important implications for identifying and understanding the potential mechanisms linking chronic sleep restriction to obesity. However, this study was limited by a small and unrepresentative sample, and the period of sleep restriction was brief (i.e. two days). As a consequence, it is unclear how closely the effects of sleep restriction
observed in this study correspond with chronic sleep restriction in the general population.

As a consequence, there is a need for further research examining the relationship between chronic sleep restriction and obesity. These studies should aim to utilise more objective measures for many of the variables identified and examined throughout this thesis. Cross-sectional studies will be important in further understanding and describing the relationship between chronic sleep restriction and obesity. In particular, there is a need for research that clarifies the relationships between chronic sleep restriction, obesity, physical activity, diet, work hours and EE are related. However, research will ultimately need to move from providing descriptive data to demonstrating cause and effect relationships. Laboratory studies will be useful in identifying potential physiological mechanisms linking chronic sleep restriction to obesity under controlled settings. However, the results may not be generalisable given that sleep restriction is not necessarily equivalent to or reflective of chronic sleep restriction in the general population.

As a consequence, it is recommended that long-term prospective studies are developed to assess the effects of chronic sleep restriction in free-living adults. These studies should examine the impact of chronic sleep restriction on specific components of energy balance given the current lack of data examining the impact of chronic sleep restriction on comprehensive measures of energy intake (i.e. diet), physical activity, 24 hour EE and the efficiency of energy storage. These studies will also need to acknowledge that the causes of chronic sleep restriction are diverse and vary between individuals. This is potentially important because the precise effects of chronic sleep restriction could differ based on the specific cause of chronic sleep restriction. A first step could be to group individuals according to
the cause of their chronic sleep restriction (e.g. due to work hours or medical conditions etc).

7.3 Conclusions

This thesis has addressed major conceptual and methodological limitations of previous research examining the relationship between chronic sleep restriction and obesity. The results provide further evidence that chronic sleep restriction is not only associated with obesity, but could contribute to obesity by altering EE and hormones involved in energy balance regulation. The identification of factors associated with chronic sleep restriction is important because these could potentially be targeted in the treatment and prevention of obesity. For example, factors such as irregular bedtimes, alcohol consumption, work hours and poor mental health could be targeted to promote healthy sleep durations and possibly aid in the treatment and prevention of obesity. These results also demonstrate that additional sleep quality factors such as sleepiness and fatigue may also be implicated in obesity, and warrant further investigation. The obtained results have potential practical implications for combating the obesity epidemic, which is increasing at a global level and contributes to pervasive health, social and economic problems. The results are important because it is possible that targeting chronic sleep restriction could be a critical step in combating the rising obesity epidemic in Australia and at a global level.
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APPENDIX A. Participant Information Sheet for Empirical Studies 1 and 2.

Participant Information Sheet

The relationship between sleep behaviours and weight in adults

Researcher

Christopher Magee (4221 4513)

Christopher Magee, Dr. Peter Caputi (4221 3717), Professor Don Iverson, Professor Xu-Feng Huang, and Dr. Nancy Humpel (4221 5441) are conducting research investigating the relationship between sleep and obesity in adults. This research is being conducted in the School of Psychology.

Changes in sleep quality and body weight represent two growing health concerns in Australia, and are associated with an increased risk for depression, hypertension, stroke, cancer, and heart disease. It is possible that poor sleep quality and weight are related, and could be associated with lifestyle and mood disturbances. The aim of the current research is to examine how sleep and obesity are related in adults.

In this study we will measure your height and weight to calculate your Body Mass Index (BMI), as well as your waist circumference. We will also use a bioelectrical impedance device to obtain a safe, accurate, and non-invasive measure of body composition. Two small electrodes will be placed on your foot and another two on your
hand/wrist. These areas will be cleaned first with an alcohol swab to ensure a good reading. We will ask you to lie on your back on a bed in the laboratory for three minutes. A reading will be taken in less than one second. These measures are commonly used, non invasive, and currently recommended by the World Health Organization.

We will also require you to complete a questionnaire that will take approximately 20 minutes. This will cover a range of issues regarding your current lifestyle, as well as your sleep patterns, your mood and general health. In total this procedure will take approximately 30 to 45 minutes of your time. You will also be provided with a sleep diary to complete over the next seven days. This will require you to indicate the times you go to bed, wake up and nap during the day, and will take no longer than 2 or 3 minutes to complete each day. All data will be coded and treated confidentially, and will be stored and locked in a secure filing cabinet in the School of Psychology.

We are also seeking volunteers for a future study that will incorporate physiology measures (such as blood samples) to further understand the relationship between sleep and obesity. This study will also be conducted at the University of Wollongong within the next two to three months. If you are interested in being sent an information and consent form regarding this additional study please indicate this on the current consent form. Please note that participation in this future study is separate from the current study. Even if you consent to be contacted for future research, you are free to refuse to participate and can withdraw at any time.
If you have any concerns or questions regarding the way in which the research is or has been conducted, please feel free to contact the University of Wollongong Ethics Officer on (02) 4221 4457.
APPENDIX B. Consent form for Empirical studies 1 and 2.

Consent Form

The relationship between sleep behaviours and weight in adults

Christopher Magee (02 4221 4513)

Increasing bodyweight and sleep disturbances are growing problems that represent a significant health risk. The aim of the current study is to further understand the relationship between overweight/obesity and sleep. In this project I understand that I will be asked to complete a questionnaire, and also have my height, weight and waist circumference measured. I also understand that a bioelectrical device will be used to measure body composition. I will also be provided with a sleep diary to complete and return in a week. In total this will take approximately 30 to 45 minutes.

I have read the participant information sheet and have had the opportunity to ask the researcher any questions regarding this project. If I have any enquiries about this research I can contact Christopher Magee (ph: 4221 4513). I understand that my participation in this research is voluntary, and that I can refuse to participate or can withdraw from the research at any time. My withdrawal will not affect my relationship with the School of Psychology or the University of Wollongong. I understand that the collected data will be used for research (with potential journal publication) and I consent for it to be used in that way.
By signing below I am consenting to participate in this research study.

Signed ..................................................  Date .....................................

.......................................................................  ....../....../......

Name (please print) ...........................................  Please place your code here

............................................................................

I have also been given information regarding a further study. By checking the box below, I am consenting to be sent information regarding this additional project. I understand that my participation in any future research is completely voluntary and that by signing I am not committing myself to taking part.

Yes, I consent to be contacted via email for an additional study ☐

Email: .................................................................

Phone number: ..............................

Thank you for your time and assistance with this project.
In order for us to link your responses while maintaining your confidentiality we need you to supply us with a code. Please fill out the boxes on the front page and Consent Form according to the following instructions (see example below).

We will ask you to generate a six digit code based on your mother’s maiden name and your date of birth.

For example: John’s mother’s maiden name was Black. He was born in May in 1945.

His code is

BL 0 5 4 5

BLACK

May = 05
Please place your code in here:

We would like to ask you a few questions. Some of these ask you to report certain behaviours/activities that you engage in during a normal week. Please base your answers on an average or typical week during the past month.

1. Are you (please circle): MALE FEMALE

2. How old are you? __________ years

3. What is the highest level of education you have completed? (please tick one)
   - [ ] Primary school only
   - [ ] Some secondary school - no certificate completed
   - [ ] Completed Year 10 or equivalent (leaving certificate or matriculation)
   - [ ] Completed Year 12
   - [ ] Technical (TAFE) certificate/ diploma or trade
   - [ ] University or tertiary degree
   - [ ] Other

4. Please indicate whether you have been diagnosed with, or treated for, any of the following conditions by your doctor (please tick):
   - [ ] Diabetes
   - [ ] Cancer
   - [ ] Heart Disease
   - [ ] Asthma
   - [ ] Ulcers
   - [ ] High Blood Pressure
   - [ ] Depression
   - [ ] Please specify any other conditions ___________________________

5. Have you suffered from any headaches/migraines in the last month?
   - [ ] YES
   - [ ] NO

6. Please indicate whether you are currently taking, or have taken in the past month, any of the following medications (please tick):
   - [ ] Antibiotics
   - [ ] Anti-depressants
   - [ ] Sleeping pills
   - [ ] The Pill
   - [ ] Panadeine Forte
   - [ ] Please specify any other medications ___________________________

7. Are you currently pregnant?  YES NO N/A

8. When you were at your fittest and healthiest as an adult:
   How much did you weigh? _________ kilograms OR _________ stones/pounds
   How old you were at the time? _________ years
9. We would like to ask you about any physical activity you may have done IN THE LAST WEEK (7 days):

a. IN THE LAST WEEK how many times have you walked continuously, for at least 10 minutes, for recreation/exercise or to get to and from places? ________ times

b. What do you estimate was the total time that you spent walking for recreation/exercise or to get to and from places IN THE LAST WEEK? ________ hours ________ minutes

c. IN THE LAST WEEK, how many times did you do any vigorous physical activity which made you breathe harder or puff and pant? (e.g. jogging, cycling, aerobics, team sports, competitive tennis, etc) ________ times

d. What do you estimate was the total time that you spent doing this vigorous physical activity IN THE LAST WEEK? ________ hours ________ minutes

e. IN THE LAST WEEK how many times did you do any other more moderate physical activity that you haven't already mentioned? (e.g. gentle swimming, social tennis, golf, bowls etc) ________ times

f. What do you estimate was the total time that you spent doing moderate activity IN THE LAST WEEK? ________ hours ________ minutes

10. At what time of the day do you normally engage in vigorous exercise?

☐ Never
☐ Morning
☐ Afternoon
☐ Within 2 to 3 hours of usual bedtime.

Questions 11a and 11b are for males only. Females please skip to question 12.

11a. Please select the figure that you think best represents your current body shape.

![Figure choices for current body shape]

11b. Please select the figure that you think best represents what your body shape was five years ago.

![Figure choices for past body shape]
Questions 12a and 12b are for females only. Males please skip to question 13.

12a. Please select the figure that you think best represents your current body shape.

![Figure Selection](image)

12b. Please select the figure that you think best represents what your body shape was five years ago.

![Figure Selection](image)

13. How many days in a normal week do you consume caffeinated drinks (e.g. coffee, tea, cola, Mountain Dew, Red Bull, etc)? (please circle)

NEVER \[1\ 2\ 3\ 4\ 5\ 6\ 7\]

If you answered NEVER please skip to question 16

14. On the days that you consume caffeinated drinks, please indicate the amount you would normally drink (please state the average number of cups/cans per day)

Coffee

- Instant \[\_\_\_\_\_\_\_\_\_\_\_\] cups per day
- Espresso \[\_\_\_\_\_\_\_\_\_\_\_\] cups per day
- Drip/Percolated \[\_\_\_\_\_\_\_\_\_\_\_\] cups per day
- Decaffeinated \[\_\_\_\_\_\_\_\_\_\_\_\] cups per day

Energy or Sports Drinks

- ‘V’/Red Bull \[\_\_\_\_\_\_\_\_\_\_\_\] cans per day
- Other \[\_\_\_\_\_\_\_\_\_\_\_\_\] cans/bottles per day

Tea \[\_\_\_\_\_\_\_\_\_\_\_\] cups per day

Caffeinated soft drinks (e.g. Pepsi, Coke, Mountain Dew) \[\_\_\_\_\_\_\_\_\_\_\_\_] cups/cans per day
15. On the days that you consume caffeinated drinks, please indicate the number of hours on average between your last drink and your bedtime: ____________ hours before sleep.

16. Do you currently smoke cigarettes/cigars/pipes etc?  YES  NO

If you answered NO please skip to question 20

17. Please indicate which of the following you currently smoke:

☐ manufactured cigarettes
☐ roll-your-own cigarettes
☐ cigars
☐ a pipe

18. Please indicate how often you smoke cigarettes/cigars/pipes? (please tick one)

☐ Daily  How many cigarettes/cigars/pipes per day? __________
☐ At least weekly  How many cigarettes/cigars/pipes per week? __________
☐ (but not daily)
☐ Less often than weekly  How many cigarettes/cigars/pipes per month? _______

19. On the days that you smoke, please indicate the number of hours on average between your last cigarette/cigar/pipe and your bedtime: ____________ hours

20. If you don’t currently smoke, did you ever smoke regularly?

☐ Yes
☐ No
☐ N/A – I smoke now

If you answered YES, please indicate

How long you smoked for _________ years ________ months
How long ago you quit _________ years ________ months
The average number of cigarettes/cigars/pipes you had each week ____________

21. How many days in a normal week do you take a nap? (please tick)

NEVER 1  2  3  4  5  6  7

If you answered NEVER please skip to question 24.

22. How long do you normally nap for on any one day?

☐ Less than 10 minutes  ☐ 10 to 20 minutes  ☐ Longer than 20 minutes
23. When do you usually take a nap during the day?

☐ Morning ☐ Midday ☐ Afternoon ☐ Evening

24. How many days in a normal week do you consume alcohol? (please circle)

NEVER  1  2  3  4  5  6  7

If you answered NEVER please skip to Question 27

25. On the days that you have consumed alcohol, please indicate the average number of hours between your last drink and your bedtime: ___________ hours

26. On the days that you consume alcohol, how many drinks would you normally have? (Please use the guide below to estimate the number of standard drinks you normally consume)

Beer __________ standard drinks /day
Wine __________ standard drinks /day
Spirits __________ standard drinks /day
Other __________ standard drinks /day

Please see print copy for Table of Standard Drinks

27. Please indicate the number of nights in a normal week that your sleep is disturbed because of a bed partner, other people in your household, pets or neighbours: (please circle)

NEVER  1  2  3  4  5  6  7
28. How many nights in a normal week do you go to bed at approximately the same time?

NEVER 1 2 3 4 5 6 7

29. How many days in a normal week do you wake/get up at approximately the same time?

NEVER 1 2 3 4 5 6 7

30. How many days in a normal week do you use your bedroom to study, watch television, play video games etc?

NEVER 1 2 3 4 5 6 7

31. In the past month have you normally slept with people in the same room?

YES  NO

If you answered YES, has this been

☐ with another person in the same bed?

AND/OR

☐ with one or more persons in the same room?

32. Please indicate the number of nights in a normal week that your sleep has been disturbed because your sleep environment is: (please tick)

<table>
<thead>
<tr>
<th>Nights Per Week</th>
<th>NEVER</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>Too noisy</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Too light</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Too cold</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Too hot</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Uncomfortable</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

33. How many days during a normal week do you watch television, DVD’s?

NEVER 1 2 3 4 5 6 7

If NEVER, please skip to question 36.

a. What is the average amount of time that you would watch television, DVD’s etc on any given day? ________ hours/day

b. How long prior to your bedtime do you normally watch television, DVD’s? ________ hours before bedtime

34. How many days during a normal week do you play video/computer games etc?

NEVER 1 2 3 4 5 6 7
If NEVER, please skip to question 35.

a. What is the average amount of time that you would play video/computer games etc on any given day? __________ hours/day
b. How long prior to your bedtime do you normally play video/computer games etc? __________ hours before bedtime

35. How many days during a normal week do you use (e.g. for work or study) a computer?

NEVER 1 2 3 4 5 6 7

If NEVER, please skip to question 36.

a. What is the average amount of time that you would use a computer for on any given day? __________ hours/day
b. How long prior to your bedtime do you normally use a computer? __________ hours before bedtime

36. How many days in a normal week do you eat:

a. Breakfast: NEVER 1 2 3 4 5 6 7
b. Lunch: NEVER 1 2 3 4 5 6 7
c. Dinner: NEVER 1 2 3 4 5 6 7

37. How many days in a normal week do you eat snacks? (a snack refers to any additional food that is not consumed during breakfast, lunch or dinner).

NEVER 1 2 3 4 5 6 7

If you answered NEVER, please skip to question 41.

38. How many snacks do you normally have each day? __________ number of snacks/day

39. Please indicate the number of days in a normal week that you consume snacks during the following times, and the type of food that you snack on (e.g. potato chips, fruit, yoghurt, chocolate bars etc).

Morning: _______ days/week ______________________ most common type of food
Afternoon: _______ days/week ______________________ most common type of food
Evening: _______ days/week ______________________ most common type of food
(after dinner)
Late at night: _______ days/week ______________________ most common type of food
(within 2 hours of bedtime)

40. Please indicate the number of days in a normal week that you consume snacks while watching TV/DVD’s etc and the type of food that you usually snack on (e.g. potato chips, fruit, yoghurt, chocolate bars etc).

_______ days/week ___________________________ type of food
Questions 41 to 49 relate to your usual sleeping 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.

41. When have you usually gone to bed?  Usual bed time ___________ 

42. How many minutes has it taken for you to fall asleep each night? ___________

43. When have you usually got up in the morning?  Usual getting up time ___________

44. How many hours of sleep did you usually get each night? (This may be different than the number of hours you spend in bed) Hours of sleep each night __________

45. This question asks you to indicate how often your sleep is disturbed. Please answer by placing a tick in the relevant column.

<table>
<thead>
<tr>
<th>During the past month, how often have you had trouble sleeping because you…</th>
<th>Not during the past month</th>
<th>Less than once a week</th>
<th>Once or twice a week</th>
<th>Three or more times a week</th>
</tr>
</thead>
<tbody>
<tr>
<td>a) Could not get to sleep within 30 minutes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>b) Woke up in the middle of the night or early morning</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>c) Had to get up to use the bathroom</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>d) Could not breathe comfortably</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>e) Cough or snore loudly</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>f) Felt too cold</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>g) Felt too hot</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>h) Had bad dreams</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>i) Had pain</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>j) Other reason(s). Please describe, including how often you have trouble sleeping because of this reason(s).</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

46. During the past month, how often have you taken medicine (prescribed or ‘over the counter’) to help you sleep?  

☐ Not during the past month  
☐ Less than once a week  
☐ Once or twice a week  
☐ Three or more times a week
47. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?

- Not during the past month
- Less than once a week
- Once or twice a week
- Three or more times a week

48. During the past month, how would you rate your sleep quality overall?

- Very good
- Fairly good
- Fairly bad
- Very bad

49. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?

- No problem at all
- A slight problem
- Somewhat of a problem
- A very big problem

Questions 50 to 56 ask for your views about your health. Please answer these questions by ticking one box. If you are unsure about how to answer a question please give the best answer you can.

50. In general, would you say your health is: (Please tick one box)

- Excellent
- Very Good
- Good
- Fair
- Poor

51. Does your health now limit you in the following activities? If so, how much?

<table>
<thead>
<tr>
<th>Activity</th>
<th>Yes Limited A Lot</th>
<th>Yes Limited A Little</th>
<th>No, Not Limited At All</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Moderate activities, such as moving a table, pushing a vacuum cleaner, bowling, or playing golf?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>b. Climbing several flights of stairs</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

52. During the past 4 weeks, have you had any of the following problems with your work or other regular daily activities as a result of your physical health?

<table>
<thead>
<tr>
<th>Problem</th>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Accomplished less than you would like</td>
<td></td>
<td></td>
</tr>
<tr>
<td>b. Were limited in the kind of work or other activities</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
53. During the past 4 weeks, have you had any of the following problems with your work or other regular daily activities as a result of any emotional problems (such as feeling depressed or anxious)?

<table>
<thead>
<tr>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Accomplished less than you would like</td>
<td></td>
</tr>
<tr>
<td>b. Were limited in the kind of work or other activities</td>
<td></td>
</tr>
</tbody>
</table>

54. During the past 4 weeks, how much did pain interfere with your normal work (including both work outside the home and housework)?

<table>
<thead>
<tr>
<th>Not at All</th>
<th>A Little Bit</th>
<th>Moderately</th>
<th>Quite a Bit</th>
<th>Extremely</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

55. This question asks you about how you feel and how things have been with you during the past 4 weeks. Please give the one answer that comes closest to the way you have been feeling.

How much of the time during the past 4 weeks:

<table>
<thead>
<tr>
<th>All of the time</th>
<th>Most of the time</th>
<th>A Good Bit of the Time</th>
<th>Some of the Time</th>
<th>A little of the Time</th>
<th>None of the Time</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

56. During the past 4 weeks, how much of the time has your physical health or emotional problems interfered with your social activities (like visiting with friends, relatives etc)?

<table>
<thead>
<tr>
<th>All of the Time</th>
<th>Most of the Time</th>
<th>Some of the Time</th>
<th>A Little of the Time</th>
<th>None of the Time</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

For each of the remaining questions, please read each statement and circle a number 0, 1, 2 or 3 which indicates how much the statement applied to you over the past week. There are no right or wrong answers. Do not spend too much time on any statement.
The rating scale is as follows:
0  Did not apply to me at all
1  Applied to me to some degree, or some of the time
2  Applied to me to a considerable degree, or a good part of time
3  Applied to me very much, or most of the time

<p>| | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>57</td>
<td>I found myself getting upset by quite trivial things.</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>58</td>
<td>I was aware of dryness of my mouth.</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>59</td>
<td>I couldn't seem to experience any positive feeling at all.</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>60</td>
<td>I experienced breathing difficulty (eg, excessively rapid breathing, breathlessness in the absence of physical exertion).</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>61</td>
<td>I just couldn't seem to get going.</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>62</td>
<td>I tended to over-react to situations.</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>63</td>
<td>I had a feeling of shakiness (eg, legs going to give way).</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>64</td>
<td>I found it difficult to relax.</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>65</td>
<td>I found myself in situations that made me so anxious I was most relieved when they ended.</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>66</td>
<td>I felt that I had nothing to look forward to.</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>67</td>
<td>I found myself getting upset rather easily.</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>68</td>
<td>I felt that I was using a lot of nervous energy.</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>69</td>
<td>I felt sad and depressed.</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>70</td>
<td>I found myself getting impatient when I was delayed in any way (eg, lifts, traffic lights, being kept waiting).</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>71</td>
<td>I had a feeling of faintness.</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>72</td>
<td>I felt that I had lost interest in just about everything.</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>73</td>
<td>I felt I wasn't worth much as a person.</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>74</td>
<td>I felt that I was rather touchy.</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>75</td>
<td>I perspired noticeably (eg, hands sweaty) in the absence of high temperatures or physical exertion.</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>76</td>
<td>I felt scared without any good reason.</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>77</td>
<td>I felt that life wasn't worthwhile.</td>
<td>0</td>
<td>1</td>
</tr>
</tbody>
</table>
Reminder of rating scale:
0  Did not apply to me at all
1  Applied to me to some degree, or some of the time
2  Applied to me to a considerable degree, or a good part of the time
3  Applied to me very much, or most of the time

Over the past week:

<p>| | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>78</td>
<td>I found it hard to wind down.</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>79</td>
<td>I had difficulty in swallowing.</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>80</td>
<td>I couldn't seem to get any enjoyment out of the things I did.</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>81</td>
<td>I was aware of the action of my heart in the absence of physical exertion (eg, sense of heart rate increase, heart missing a beat).</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>82</td>
<td>I felt down-hearted and blue.</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>83</td>
<td>I found that I was very irritable.</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>84</td>
<td>I felt I was close to panic.</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>85</td>
<td>I found it hard to calm down after something upset me.</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>86</td>
<td>I feared that I would be &quot;thrown&quot; by some trivial but unfamiliar task.</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>87</td>
<td>I was unable to become enthusiastic about anything.</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>88</td>
<td>I found it difficult to tolerate interruptions to what I was doing.</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>89</td>
<td>I was in a state of nervous tension.</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>90</td>
<td>I felt I was pretty worthless.</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>91</td>
<td>I was intolerant of anything that kept me from getting on with what I was doing.</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>92</td>
<td>I felt terrified.</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>93</td>
<td>I could see nothing in the future to be hopeful about.</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>94</td>
<td>I felt that life was meaningless.</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>95</td>
<td>I found myself getting agitated.</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>96</td>
<td>I was worried about situations in which I might panic and make a fool of myself.</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>97</td>
<td>I experienced trembling (eg, in the hands).</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>98</td>
<td>I found it difficult to work up the initiative to do things.</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>
Christopher Magee, Professor Don Iverson, Professor Xu-Feng Huang, Dr. Peter Caputi (4221 3717), and Dr. Nancy Humpel (4221 5441) are conducting research investigating the effects of sleep loss on different aspects of body physiology in adults. This research is being conducted in the School of Psychology.

Study Aims

The aim of this research is to investigate the effects of two nights of sleep restriction (four hours sleep a night) on your appetite and important hormones involved in regulating food intake. To examine this, we will monitor your appetite and food intake over four consecutive days after one night of normal sleep at your home, two nights of sleep limited to four hours in the Whole Room Calorimeter at the University of Wollongong, followed by another night of normal sleep at your home.

What is a Whole Room Calorimeter?

There are two Whole Room Calorimeters at the University of Wollongong which are located in the Health Sciences building. The rooms look like small bed-sitters and are equipped with basic facilities such as desk, chair, bed, telephone, television, computer,
and a private sink and toilet. We will ask you to spend 12 hours in the whole room calorimeter on each of these nights, during which time you may watch television, read or use the computer at your own leisure.

**What will be measured?**

In this study you will have your blood taken and your appetite, hunger and dietary intake monitored over a period of four days. Your appetite and hunger will be measured by a brief questionnaire on each morning of this study. Your dietary intake will also be measured by a short questionnaire each evening before bed.

Blood will be drawn on three of the four mornings by a qualified person. The amount of blood that will be taken on each morning will be 40mls. The taking of blood samples may produce some temporary discomfort and could result in some mild bruising at the site of sampling. Any bruising would resolve within a few days.

**Details of this study**

**Day 1:** This study will begin at 7:00pm, where you will report to the laboratory at the University of Wollongong and be served an evening meal. After you have finished this meal, you will be able to leave the laboratory and engage in your normal activities.

**Day 2:** This study will begin in the morning after a normal night of sleep at home and 12 hours of fasting. We will ask you to report to the laboratory in the Faculty of Health and Behavioural Sciences before breakfast and complete a brief questionnaire on your
appetite and hunger. You will then have 40mls of blood drawn by an accredited person and you may then leave the laboratory and engage in normal daily activities, but avoid alcohol and not drink more than two caffeinated drinks. We will ask you to return to the laboratory at 7:00pm where an evening meal will be served. After this we will ask you to complete a dietary questionnaire and spend from 8:00pm to 8:00am in the Whole Room Calorimeter. Although you will spend approximately 12 hours in the Whole Room Calorimeter, we will ask that you to stay awake beyond your normal bedtime and sleep only in the last four hours of your normal sleep period. This will be monitored by a supervisor.

**Day 3:** Upon waking in the Whole Room Calorimeter, we will ask you to complete the hunger and appetite questionnaire before breakfast. We will not draw any blood and you may leave the laboratory during the day. However, we ask that you avoid taking a nap during the day and do not consume any alcohol or drink more than two caffeinated drinks. We will ask that you return to the laboratory at 7:00pm where an evening meal will be served. After this we will ask you to complete a dietary questionnaire and spend from 8:00pm to 8:00am in the Whole Room Calorimeter. Again we will ask that you only sleep for four hours on this night, and this will be monitored by a supervisor.

**Day 4:** Upon waking in the Whole Room Calorimeter, we will ask you to complete the appetite questionnaire and have 40mls of blood drawn before breakfast by an accredited person. Again you may leave the laboratory during the day, but we ask that you avoid taking a nap during the day and do not consume any alcohol or drink more than two caffeinated drinks. We will ask that you return to the laboratory at 7:00pm where an
evening meal will be served and you will need to complete the dietary questionnaire. Once this has been completed you may leave the laboratory and sleep at home for as long as you wish on this night. We will ask that you wear a small wrist-watch like device on this night called an actigraph. This will provide us with an accurate and non-invasive measure of your sleep duration on this night.

**Day 5:** We will ask that you return to the laboratory in the morning at approximately 8:00am before breakfast and after 12 hours of fasting. You will be asked to complete the visual analogue scale and 40mls of blood will be drawn by an accredited person before breakfast. You may then leave the laboratory during the day but will need to complete the 24 hour dietary recall questionnaire after your evening meal and prior to bedtime.

**Possible Risks of this Study**

As mentioned some bruising might occur with blood sampling. You may also feel fatigued after the two nights of restricted sleep. If you do begin to feel fatigued we ask that you inform one of the researchers so that appropriate transportation home can be arranged.

**Maintaining Confidentiality**

All data obtained from this study will be coded and treated confidentially. Questionnaires will be stored and locked in a secure filing cabinet in the School of Psychology. Blood samples will be stored securely in a freezer in the Faculty of Health and Behavioural Sciences.
If you have any concerns or questions regarding the way in which the research is or has been conducted, please feel free to contact the University of Wollongong Ethics Officer on (02) 4221 4457.

We will provide you with two movie tickets as a token of appreciation for participating in this study.
APPENDIX E. Consent form for Empirical Study 3.

Consent Form

Physiological Effects of Sleep Restriction

Christopher Magee (02 4221 4513)

The aim of this study is to examine the effects of two days of partial sleep loss on various aspects of hunger and appetite as well as important body hormones. I understand that my involvement in this study will last four consecutive days and that blood will be drawn on three of these days. I will also be asked to complete a short questionnaire on my hunger levels each morning and complete a dietary questionnaire each night. I also understand that I will be asked to spend two of these nights in the Whole Room Calorimeter at the University of Wollongong where I will be required to sleep for four hours on each of these nights.

I have read the participant information sheet and have had the opportunity to ask the researcher any questions regarding this project. If I have any enquiries about this research I can contact Christopher Magee (ph: 4221 4513). I understand that my participation in this research is voluntary, and that I can refuse to participate or can withdraw from the research at any time. My withdrawal will not affect my relationship with the School of Psychology or the University of Wollongong. I understand that the
collected data will be used for research (with potential journal publication) and I consent for it to be used in that way.

By signing below I am consenting to participate in this research study.

Signed

..........................................................

Date

..........................................................

Name (please print)

..........................................................

Thank you for your time and assistance with this project.
APPENDIX F. Visual Analogue Scale for Empirical Study 3.

Code: ___________
Time: morning
Date: ______________

Instructions: The following items ask you about your hunger and your desire to eat different foods. For each of these, please place a mark on the line at a point that best corresponds with how you feel at the moment.

1. How hungry do you feel?

I am not hungry at all | ______________________________ | I have never been more hungry

2. How full do you feel?

Not at all | ______________________________ | I feel completely full
completely full

3. How much do you think you can eat?

Nothing at all | ______________________________ | A lot
all

4. How strong is your desire to eat something sweet (e.g. cake, candy cookies, ice cream, and pastry)?

Not strong at all | ______________________________ | Extremely strong

5. How strong is your desire to eat something salty (e.g. chips, salted nuts, pickles, and olives)?

Not strong at all | ______________________________ | Extremely strong

6. How strong is your desire to eat something starchy (e.g. bread, pasta, cereal, and potatoes)?

Not strong at all | ______________________________ | Extremely strong
7. How strong is your desire to eat some fruit or drink some fruit juice?

<table>
<thead>
<tr>
<th>Not strong at all</th>
<th>Extremely strong</th>
</tr>
</thead>
</table>

8. How strong is your desire to eat some vegetables?

<table>
<thead>
<tr>
<th>Not strong at all</th>
<th>Extremely strong</th>
</tr>
</thead>
</table>

9. How strong is your desire to eat some meat, poultry, fish, and/or eggs?

<table>
<thead>
<tr>
<th>Not strong at all</th>
<th>Extremely strong</th>
</tr>
</thead>
</table>

10. How strong is your desire to eat some dairy products?

<table>
<thead>
<tr>
<th>Not strong at all</th>
<th>Extremely strong</th>
</tr>
</thead>
</table>