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Sleep quality is differentially related to adiposity in adults

S. Katherine Sweatt[a], Barbara A. Gower[b], Angela Y. Chieh[a], Yang Liu[a], Li Li[a,*]

[a] Department of Psychiatry and Behavioral Neurobiology, University of Alabama at Birmingham, Birmingham, AL, 35294, United States
[b] Department of Nutrition Sciences, University of Alabama at Birmingham, Birmingham, AL, 35294, United States

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ABSTRACT

Objectives: Sleep duration is associated with adiposity in adults. Abdominal adiposity specifically is strongly correlated with metabolic alterations, however, the relationships between abdominal adiposity and sleep quality are incompletely understood. The purpose of this study is to test the hypothesis that abdominal adiposity is related to poor sleep quality while total adiposity is not; and to explore whether pathways, including immune system and hypothalamic-pituitary-adrenal axis, link abdominal adiposity to poor sleep quality.

Methods: Subjects were 101 men and women aged 38.88 ± 11.96 years with body mass index between 29.35 ± 6.93 kg/m^2. Subjective sleep quality was determined by the Pittsburgh Sleep Questionnaire Index (PSQI). Body composition was determined by dual energy X-ray absorptiometry. Saliva and blood samples were collected for assessment of cortisol and markers of inflammation. In a cross-sectional study design, correlation analysis was conducted to determine the relationships between poor sleep quality and adiposity. Participants were stratified based on PSQI score to evaluate differences in main outcomes between subjects with normal (NSQ; PSQI ≤ 5) vs poor sleep quality (PSQ; PSQI > 5).

Results: Poor sleep quality was related to greater visceral fat (r = 0.26; $p < 0.05$), but not total fat. The PSQ group had greater visceral fat compared to the NSQ group (1.11 ± 0.83 kg vs 0.79 ± 0.62 kg; $p < 0.05$), however, there was no difference in total fat mass (33.18 ± 14.21 kg vs 29.39 ± 13.03 kg; $p = 0.24$). The PSQ group had significantly greater leptin (1.37 ± 0.07 ng/ml vs 1.08 ± 0.08 ng/ml; $p < 0.05$), but hypothalamic-pituitary-adrenal axis activity did not differ between the PSQ and NSQ groups.

Conclusions: Poor sleep quality is associated with greater visceral adiposity and leptin secretion. Further research is needed to probe potential cause and effect relationships among visceral adipose tissue, leptin, and sleep quality.

1. Introduction

The prevalence of obesity has steadily risen worldwide in the last few decades and has become a major public health issue. More than one-third of adults (34.9%) in the United States are obese (Ogden et al., 2013). Obesity, particularly in the visceral region, is related to increased risk of diseases such as diabetes, cardiovascular disease (CVD), and some cancers (Tchernof and Despres, 2013). Measures such as body mass index (BMI) and weight give limited information about disease risk because they lack the ability to determine body fat distribution. Body fat itself is not necessarily the causal factor in the occurrence of CVD, diabetes, and other obesity-related metabolic disorders, but the metabolic consequences of body fat location and dysfunction are important. Visceral adipose tissue (VAT), specifically, is body fat in the intra-abdominal cavity surrounding the organs. The relationship between VAT and metabolic disorders, such as insulin resistance, CVD, and diabetes, is well established (Wensveen et al., 2015). VAT has been shown to be a predominant source of chronic systemic inflammation which is a major contributor to the development of obesity-related diseases (Molofsky et al., 2013; Mathis, 2013). Thus, it is critical to identify factors that are related to obesity, and specifically, to VAT.

One of the factors that may increase risk for obesity is insufficient sleep. Short sleep duration is commonly due to waking up too early, inability to fall asleep, and waking for long periods of time during the night. This can result in daytime fatigue, irritability, and reduced concentration (Van Dongen et al., 2003). Recent studies have reported short sleep duration is a predictor of obesity in adults (Cappuccio et al., 2008). Previous studies have shown that short sleep duration is related to increased abdominal adiposity in both adults and children (Chaput et al., 2011; Chaput and Tremblay, 2007; Hairston et al., 2010). In

* Corresponding author at: Department of Psychiatry and Behavioral Neurobiology, University of Alabama at Birmingham, 1720 University Blvd., Birmingham, AL, 35294, United States.
E-mail address: liyli@uabmc.edu (L. Li).

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addition, a meta-analysis revealed that each reduction of 1 h of sleep per day was associated with an increase in BMI by 0.35 per year (Cappuccio et al., 2008). Sleep duration is just one component of sleep quality, however. In addition to total sleep duration, sleep quality includes sleep latency, number of arousals, and subjective "depth" or "restfulness" of sleep (Buysse et al., 1989). Sleep modulates neuroendocrine function and glucose metabolism, and inadequate sleep is related to metabolic abnormalities such as decreased glucose tolerance and altered appetite regulating hormone (Lucassen et al., 2012), which in turn increases the risk of weight gain and obesity. However, limited research has been conducted to examine the relationship between sleep quality, and total and regional adiposity, such as VAT.

Adipose tissue is a secretory organ, and excess accumulation leads to increased release of pro-inflammatory cytokines, such as tumor necrosis factor- alpha (TNF-α), leptin, and interleukin-6 (IL-6), leading to low grade systemic inflammation (Schrover et al., 2016). Inflammation is associated with numerous diseases, including diabetes, CVD, and some cancers. In addition, several studies report plasma levels of cytokines are related to the sleep-wake cycle in humans (Gudewill et al., 1992). IL-6, specifically, is inversely associated with short sleep duration and has been shown to be elevated after sleep restriction (Papanicolaou et al., 1998; Voderholzer et al., 2012). Furthermore, leptin, an adipocyte-derived hormone, is known to play a key role in the regulation of appetite and body weight (Licinio et al., 1997). Findings from the Spiegel group have proposed leptin as a possible link between the risk of obesity and abnormal sleep duration in healthy individuals (Spiegel et al., 2004a,b). Thus, chronic inflammation may be a link between adiposity and poor sleep quality.

It is well established that sleep is intricately linked with the hypothalamic-pituitary-adrenal (HPA) axis. Activation of the HPA axis is characterized by cortisol secretion in humans which leads to arousal (Weitzman et al., 1983). Studies have shown that glucocorticoid administration causes arousal and reduced sleep time (Weitzman et al., 1983). Poor sleep quality was shown to be associated with increased plasma cortisol levels (Spath-Schwalbe et al., 1991). Additionally, it has been shown that cortisol awakening response is higher the morning following short sleep duration (Steiger and Holsboer, 1997). Another study evaluated the diurnal cortisol secretion across the day and found that cortisol was higher in subjects with low sleep efficiency (Raikkonen et al., 2010). In addition, there is a strong relationship between HPA axis and energy homeostasis. Greater salivary cortisol has been shown to be related to increased BMI over time, suggesting elevations in cortisol are related to weight gain (Roberts et al., 2007). Furthermore, glucocorticoid receptor has been shown to be more abundant in VAT, indicating that glucocorticoid secretion may be linked to VAT mass (Pickering et al., 2016). Thus, it is plausible that there is a relationship between adiposity, specifically VAT, and poor sleep quality via HPA axis dysfunction.

While research supports the notion that poor sleep quality is related to obesity, specific fat depots have not been explored in relation to poor sleep quality. Therefore, the objective of this study was to test the hypothesis that abdominal adiposity is related to poor sleep quality while total adiposity is not. A secondary aim was to explore the hypothesis that chronic inflammation and/or HPA axis activity were involved in the relationship between obesity and sleep quality using correlation and regression analyses.

2. Material and methods

2.1. Subjects

This was a cross-sectional study design. All participants were recruited through the community, Birmingham, AL, or the inpatient/outpatient Psychiatric settings at the University of Alabama at Birmingham (UAB). The project was approved by the UAB Institutional Review Board and was conducted in accordance with the Helsinki Declaration of 1975. All participants provided written informed consent prior to participating in any research procedures. Of 120 men and women enrolled in the study, 101 participants completed all study procedures for data analysis. Of the 19 subjects that were excluded, 4 subjects did not complete the dual energy X-ray absorptiometry scan, 14 were excluded for sleep disorders, substance use, mania or diabetes, and 1 refused the blood draw. Participants included males and females between 19 and 55 years of age (Table 1). Participants were excluded if they: (1) had a known history of diabetes; (2) were taking medications known to affect body weight; (3) were pregnant or lactating; or (4) had a history of psychosis, bipolar disorder, or drug or alcohol use disorder within 1 year prior to enrollment. People with a diagnosis of sleep disorders, including sleep apnea, were also excluded from the study. Demographic data, medical history, and medications were recorded by self-report. Depressive symptoms or severity in all participants was measured using the Quick Inventory of Depressive Symptoms (QIDS) questionnaire (Rush et al., 2003). The QIDS was designed to assess the severity of depressive symptoms by self-report. The scoring system of the QIDS comprises 9 domains of depressive symptoms, including 1) sad mood; 2) concentration; 3) self-criticism; 4) suicidal ideation; 5) interest; 6) energy/fatigue; 7) sleep disturbance (initial, middle, and late insomnia or hypersomnia); 8) decrease or increase in appetite or weight; and 9) psychomotor agitation or retardation. Each domain weights 0–3 and the total score ranges from 0 to 27.

Table 1
Descriptive Statistics, n = 101.

Variable	Mean ± S.D. or n
Age (years)	38.88 ± 11.96
Race (Caucasian/African American)	56/45
Sex (male/female)	43/58
Body mass index (kg/m^2)	29.35 ± 6.93
Waist/hip	0.87 ± 0.08
Pittsburgh Sleep Quality Index total score	7.61 ± 5.75
Education	
< 10 years	2
10–12 years	14
> 12 years	85
Employed	39
Smoking	17
Use of sleeping medication	13
QIDS score	8.10 ± 6.58

Abbreviations: kg/m^2, kilograms per meter squared; QIDS, Quic, Quick Inventory of Depressive Symptoms.

2.2. Self-reported sleep quality

The Pittsburgh Sleep Quality Index (PSQI) is a 19-item self-rated questionnaire that measures sleep quality along seven dimensions: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbance, use of sleeping medications, and daytime dysfunction over the last month (Buysse et al., 2008). Each dimension is rated on a four-point Likert scale. Scores from these dimensions are added together to generate a global score ranging from 0 to 21. A score greater than 5 indicates poor sleep quality (Buysse et al., 1989, 2008).

2.3. Determination of body composition

BMI was calculated using the Quetelet index (kg/m^2). Waist-to-hip ratio was calculated from waist and hip circumferences. Body composition was determined by dual energy X-ray absorptiometry (Lunar iDXA, GE-Healthcare Madison, WI). Participants wore light clothing and removed metal objects from their body. CoreScan software was used to estimate the visceral fat mass based on the measurement of abdominal and subcutaneous adipose tissues (Xia et al., 2014).