Psychiatry Research: Neuroimaging 234 (2015) 90–95

Contents lists available at ScienceDirect

Psychiatry Research: Neuroimaging

journal homepage: www.elsevier.com/locate/psychresns

Comparison of obese adults with poor versus good sleep quality during a functional neuroimaging delay discounting task: A pilot study

Laura E. Martin [a], Lauren Pollack [b], Ashley McCune [b], Erica Schulte [a,c], Cary R. Savage [d], Jennifer D. Lundgren [b,*]

[a] Hoglund Brain Imaging Center, University of Kansas Medical Center, Kansas City, KS, United States
[b] Department of Psychology, University of Missouri Kansas City, Kansas City, MO, United States
[c] Department of Psychology, University of Michigan, Ann Arbor, MI, United States
[d] Center for Health Behavior Neuroscience, University of Kansas Medical Center, Kansas City, KS, United States

ARTICLE INFO

Article history:
Received 14 August 2014
Received in revised form
9 February 2015
Accepted 27 August 2015
Available online 29 August 2015

Keywords:
Sleep quality
Self-regulation
Delay discounting
Brain activation
Obesity

ABSTRACT

This study aimed to determine if obese adults with poor versus good sleep quality demonstrate reduced self-regulatory capacity and different patterns of neural activation when making impulsive monetary choices. Six obese, good quality sleepers (M age=44.7 years, M BMI=38.1 kg/m^2) were compared to 13 obese, poor quality sleepers (M age=42.6, M BMI=39.2 kg/m^2) on sleep and eating behavior and brain activation in prefrontal and insular regions while engaging in a delay discounting task during functional magnetic resonance imaging (fMRI). Poor quality sleepers demonstrated significantly lower brain activation in the right inferior frontal gyrus, right middle frontal gyrus, and bilateral insula when making immediate and smaller (impulsive) monetary choices compared to the baseline condition. Behaviorally, poor compared to good quality sleepers reported higher scores in the night eating questionnaire. Obese adults with poor sleep quality demonstrate decreased brain activation in multiple regions that regulate cognitive control and interceptive awareness, possibly reducing self-regulatory capacity when making immediately gratifying decisions.

1. Introduction

Sleep loss is considered an independent risk factor for obesity and related metabolic disease (Spiegel et al., 1999; Van Cauter and Knutson, 2008). Poor sleep quality and short sleep duration increase the risk of obesity and comorbid health conditions through multiple pathways (Chaput and Tremblay, 2012), including the up-regulation of appetite regulating hormones (e.g., leptin, ghrelin) resulting in increased hunger and decreased satiety (Spiegel et al., 2003, 2004), and alterations in glucose metabolism (Spiegel et al., 1999). In addition to these homeostatic energy regulation mechanisms, non-homeostatic hedonic feeding mechanisms (e.g., eating in response to the reward properties of food, impulsive eating styles) (Chaput and Tremblay, 2012) might, in part, account for the pathway from insufficient and/or poor quality sleep to obesity.

In support of the hypothesis that disturbed sleep could lead to obesity through non-homeostatic mechanisms, such as impulsive eating behavior, several studies have demonstrated that short sleep duration and insomnia are associated with aspects of impulsivity (Drummond et al., 2006; Schmidt et al., 2008; Schmidt et al., 2010; Anderson and Platten, 2011; Libedinsky et al., 2013). For example, Schmidt et al. (2008) found that self-reported insomnia severity was positively correlated with two facets of impulsivity: urgency (tendency to act rashly) and lack of perseverance (inability to remain focused on a task) among healthy adults. Interestingly, both urgency and lack of perseverance have been associated with maladaptive eating behavior and binge frequency in a longitudinal study among young adult women (Peterson and Fischer, 2012).

Restricted sleep duration affects other aspects of self-regulatory capacity, such as risky decision making, as demonstrated in recent brain imaging studies (Venkatraman et al., 2007, 2011). Venkatraman et al. (2007) examined the effect of experimentally induced sleep deprivation on neural response during a monetary gambling task in a sample of healthy young adults. During sleep deprivation, greater activation in the right nucleus accumbens was associated with choosing higher risk gambles, suggesting that sleep deprivation might increase reward-seeking behavior.

A commonly used measure of impulsivity that involves both reward valuation and behavioral self-regulation is delay discounting. Delay discounting is the process whereby the perceived value of a reward is decreased as its receipt is delayed (Kirby and

* Correspondence to: Department of Psychology, University of Missouri-Kansas City, 5030 Cherry Street, Ste. 321, Kansas City, MO 64110, United States. Fax: +816 235 1062.
E-mail address: lundgrenj@umkc.edu (J.D. Lundgren).

http://dx.doi.org/10.1016/j.pscychresns.2015.08.011
0925-4927/

Marakovic, 1996). It involves two primary brain systems, one of which is active in evaluating the value of the reward (typically monetary) and the other is active in prospective thought and future planning (McKell Carter et al., 2010). Regions associated with these two brain systems and commonly activated during delay discounting fMRI paradigms include the prefrontal cortex, insular cortex, ventral striatum, cingulate cortex, parietal cortex, and midbrain (McKell Carter et al., 2010). Although delay discounting paradigms typically use monetary reward, they are successful in predicting BMI (Epstein et al., 2014) and weight gain across time among obese adults (Kishinevsky et al., 2012). With regard to the effects of sleep on delay discounting, Libedinsky et al. (2013) found that total sleep deprivation among young adults was associated with effort discounting (the preference for a smaller reward that requires less effort vs. a larger reward that requires more effort), but not delay discounting. We are aware of no studies that have examined delay discounting, particularly its neural correlates, among obese adults with poor sleep quality.

Critical barriers to understanding more fully the relationship between sleep and obesity exist. In particular, the majority of studies assessing the relationship between sleep and obesity focus on experimentally induced short sleep duration, rather than sleep quality. With an estimated 42% of American adults reporting clinically significant symptoms of insomnia, including difficulty initiating sleep, difficulty maintaining sleep, early morning awakening, and non-restorative sleep (Walsh et al., 2011), the relationship among sleep quality, obesity, and metabolic health is potentially a more significant public health concern than the relationship between short sleep duration and health. Similarly, much of the literature on sleep and impulsivity is experimental, focusing on acute total sleep deprivation among healthy young, non-obese adults. In addition, impulsivity and its neural correlates have not been adequately studied as a potential pathway from poor sleep to obesity. The aim of the current pilot study, therefore, was to determine if obese adults with poor sleep quality, compared to those with good sleep quality, demonstrate greater behavioral impulsivity and altered patterns of neural activation when making impulsive monetary choices during a delay discounting task. As a secondary aim, this study sought to compare the eating behaviors of poor and good quality sleepers.

2. Methods

2.1. Participants and recruitment

Upon approval from University ethic committees, obese adults with a range of sleep quality were recruited from an urban, Midwestern community with flyers posted on University campuses, internet and print classified advertisements, and blast emails sent to University employees. Participants were eligible for the study if they were 18–65 years of age, had a body mass index (BMI) ≥ 30.0 kg/m^2, and were right handed. Individuals with a history of neurological impairment or head trauma, diagnosed sleep disorder including obstructive sleep apnea (based on self-report), current substance use disorder, metal in body, or current pregnancy were not enrolled in the study. In order to ensure a range of sleep quality, separate recruitment efforts with identical inclusion and exclusion criteria targeted good and poor quality sleepers. Eating behavior was not mentioned in recruitment advertisements, nor was it an exclusionary criterion.

Ninety-seven people responded to the recruitment advertisements. Of those, 21 participants met inclusion and exclusion criteria, provided written informed consent, enrolled in the study, completed a baseline assessment appointment, and participated in a functional magnetic resonance imaging (fMRI) paradigm. Two of the 21 participants who completed the study presented with a brain anatomic abnormality upon scanning and were excluded from further analysis. The current report, includes the 19 individuals (13 poor quality and 6 good quality sleepers) who completed the study and had useable fMRI data. Participant demographics are presented in Table 1.

Table 1
Comparison of good versus poor quality sleepers on demographic, sleep, eating behavior, and impulsivity variables.

Variable	Good quality sleepers ($n=6$) mean ± SD (range) or n (%)	Poor quality sleepers ($N=13$) Mean ± SD (Range) or n (%)	Significance t (df), p or χ^2 (df), p
Age (years)	44.7	42.6	NS
BMI (kg/m^2)	38.1	39.2	NS
Sex			7.7 (1), <0.01
Female	3 (50%)	13 (100%)	
Male	3 (50%)	0 (100%)	
Education			3.8 (3), NS
High School	0 (0)	1 (7.7)	
Some college	1 (16.7)	7 (53.8)	
Bachelors	2 (33.3)	3 (23.1)	
Graduate/ professional	3 (50%)	2 (15.4)	
Pittsburgh sleep quality Inventory			
Global score	3.1 (1.5)	11.2 (2.9)	−6.4 (17), <0.001
Sleep quality	0.5 (0.5)	1.9 (0.5)	−5.6 (17), <0.001
Sleep latency	0.3 (0.5)	2.2 (0.8)	−5.1 (17), <0.001
Sleep duration	0.6 (1.0)	1.6 (1.0)	NS
Habitual sleep efficiency	0 (0)	1.0 (1.2)	NS
Sleep disturbance	1.2 (0.4)	1.9 (0.8)	−2.3 (17), <0.05
Use of sleep medications	0 (0)	1.2 (1.3)	−2.2 (17), <0.05
Daytime dysfunction	0.5 (0.5)	1.4 (0.7)	−2.9 (17), <0.05
Eating behavior Assessments			
Eating Inventory			
Cognitive restraint	10.2 (5.2)	6.8 (3.3)	NS
Disinhibition	8.5 (4.4)	9.2 (4.5)	NS
Hunger	4.7 (1.5)	6.8 (4.1)	NS
Gormally Binge Eating Scale	27.5 (4.8)	33.7 (7.6)	NS
Night Eating Questionnaire	6.7 (2.5)	19.3 (5.6)	−5.2 (17), <0.001
Delay discounting (natural log transformation of slope)	−4.5	−3.8	NS

Note. NS = p > 0.05.

2.2. Behavioral Assessments and procedures

2.2.1. Sleep

Subjective sleep quality during the previous month was assessed with the Pittsburgh Sleep Quality Index (PSQI) (Buysse et al., 1989). The PSQI is self-rated questionnaire yielding scores for seven sleep components (scores range 0–3) and global sleep quality (scores range 0–21). Sleep components include subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medications, and daytime dysfunction. Higher scores on the seven components and the global score are indicative of poorer sleep quality. Previous research suggests that a global score greater than five is able to discriminate patients (depressed individuals and sleep disorder