Published in final edited form as:

Psychiatry Res. 2015 October 30; 234(1): 90–95. doi:10.1016/j.psychres.2015.08.011.

Comparison of Obese Adults with Poor versus Good Sleep Quality during a Functional Neuroimaging Delay Discounting Task: A Pilot Study

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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 = 83.1 kg/m²) were compared to 13 obese, poor quality sleepers (M age = 42.6, M BMI = 39.2 kg/m²) 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 interoceptive awareness, possibly reducing self-regulatory capacity when making immediately gratifying decisions.

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Conflict of Interest

The authors declare no conflict of interest.

Author Contributions

Drs. Lundgren and Martin designed the study, conducted the data analyses, co-drafted the manuscript, edited the final version of the manuscript, and served as PI (Lundgren) and Co-I (Martin) on the grant which funded it. Dr. Savage served as Co-I on the grant which funded it and contributed to manuscript editing. Ms. McCune, Pollack, and Schulte collected the data, preprocessed the data, and provided feedback on the manuscript drafts.

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Keywords

sleep quality; self-regulation; delay discounting; brain activation; obesity

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; Spiegel et al., 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 and colleagues (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; Venkatraman et al., 2011). Venkatraman and colleagues (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 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 and colleagues (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) $\geq 30.0 \text{ kg/m}^2$, 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.

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 patients) from healthy controls with a sensitivity of 89.6 and specificity of 86.5 (Buysse et al., 1989). In this study good quality sleep was operationalized as a score of five or less on the PSQI.

2.2.2 Eating Behavior—Eating behavior was assessed using three self-report inventories. The Eating Inventory (Stunkard and Messick, 1985) is a 51-item self-report scale with a three factor structure and good internal consistency. The first scale assesses cognitive restraint, or the ability to successfully restrict food intake and consequently control body weight. The second scale assesses disinhibition in relation to uncontrolled over eating and the final scale assesses perceived hunger. Because both binge eating and night eating behavior have been associated with poor sleep quality (Rogers et al., 2006; Trace et al., 2012), the Gormally Binge Eating Scale (GBES) (Gormally et al., 1982), and the Night Eating Questionnaire (NEQ) (Allison et al., 2008) were also administered. The GBES is a 16-item questionnaire used to assess the presence of binge eating behavior indicative of an eating disorder and which frequently co-occur with impulsivity. The questions are based on behavioral characteristics (e.g., amount of food consumed) and the emotional, cognitive response, guilt or shame. The NEQ is a 14 item (range of scores 0–52) self-report instrument that assesses symptoms of NES, including night eating behavior, cravings to eat at night, morning hunger and timing of food intake, as well as sleep and mood disturbance.

2.2.3 Impulsivity—Delay discounting is the behavioral impulsivity paradigm on which we have chosen to focus our work because it is increasingly being used in the obesity literature (Appelhans et al., 2012; Kishinevsky et al., 2012; Stoeckel et al., 2013; Epstein et al., 2014) and our broad aim with this line of research is to explore mechanisms by which poor sleep quality leads to obesity. Prior to scanning, participants were seated in a quiet room by themselves and completed a computerized delay discounting task based on Wittmann and colleagues (2007). Participants were presented with 8 choices for each delay (2 weeks, 4 weeks, 6 weeks and 12 weeks) that varied the amount of reward (from \$0 – 60). In all cases, a smaller more immediate reward was juxtaposed with a larger, more delayed reward. Two sources of data were obtained from the behavioral discounting task. The first type of data obtained from the behavioral discounting task was the indifference points for 2, 4, 6, and 12-week delayed time periods. Indifference points determine when an individual prefers the smaller, immediate reward as opposed to the larger, delayed one (Whittman and Paulus,

2008). The indifference points were used to calculate the rate of discounting, expressed as a slope (k value) was obtained (Reed et al., 2012). Steeper slopes are indicative of a preference for smaller, immediate rewards. Because the data are highly skewed, k values were natural log transformed using SPSS (version 21) before they were analyzed.

2.3 fMRI procedures

2.3.1 Delay Discounting Paradigm—Participants completed the delay-discounting task in the fMRI scanner, modeled after the procedure of McClure et al (2004). Five blocks of 24 monetary decision making trials were presented, with each functional run being seven minutes long. A fixation cross was presented as a baseline for 3–12 seconds to jitter trial presentation. Participants indicated with a key-press whether they preferred the option on the left or right. Optimal timing of trials was determined using Analysis of Functional Neuroimage (AFNI) stimulus timing program RSFgen. Figure 1 illustrates the presentation of a trial.

Indifference points, calculated during the behavioral delay discounting task described above, were used to tailor the delay-discounting task during fMRI to each participant in an attempt to balance the number of trials on which the participant made immediate and delayed choices. This procedure resulted in approximately 60 trials on which the decision should be choosing the immediately available reward and 60 trials on which the decision should be choosing the delayed reward.

2.3.2. fMRI Data Acquisition—Scanning was performed at the University of Kansas Medical Center's Hoglund Brain Imaging Center on a 3-Tesla Siemens Skyra scanner. T1-weighted 3D MPRAGE anatomic images were obtained (TR/TE 23/2ms, flip angle 9°, FOV = 256mm, matrix = 256 × 176, slice thickness = 1 mm). These images provided slice localization for functional scans, Talairach transformation, and co-registration with fMRI data. Gradient echo blood oxygen level dependent (BOLD) scans were acquired in 35 contiguous slices at a 40° angle to the AC/PC line (TR/TE = 2000/25ms, flip angle = 90°, matrix = 80 × 80, slice thickness = 3 mms, in-plane resolution = 2.9 mm). All participants were positioned so the angle of the AC-PC plane was between 17–22°, which was verified by a localization scan.

2.3.3 fMRI Data Analysis—Data pre-processing and statistical analyses were performed in AFNI (Medical College of Wisconsin). The fMRI images were realigned to the first slice collected to correct for motion across the runs for every subject. The images were spatially smoothed with a 4 mm FWHM Gaussian blur. Spatial normalization was performed using AFNI's automated algorithm (@auto_t1rc) and the TT_N27+tlrc template. Statistical contrasts were conducted using multiple regression analysis with motion parameters included as nuisance regressors. Regressors representing the experimental conditions for the decision-making phase were modeled with a hemodynamic response filter and entered into the multiple-regression analysis using a random-effects model. Duration modulation regression in AFNI was used so that the decision-making phase included the time from the presentation of the choice until the participant responded. The decision-making phase included two experimental conditions: Immediate and Delay. Immediate trials were trials on

which the participant chose the immediately available reward and the Delay trials were trials on which the participant chose the delayed reward.

Data analyses focused on whole-brain voxel-wise t-tests (3dttest++) to determine the brain activations when participants chose the immediately available reward. Because we were particularly interested in brain activation while making impulsive decisions only the 1) Immediate Delay and 2) Immediate Baseline (e.g. fixation) contrasts are presented¹. The whole brain voxelwise analysis was corrected for multiple comparisons at FDR < .05.

Based on the whole brain analysis, mean percent signal change was extracted from clusters of significant activation in the prefrontal and limbic regions for the immediate versus baseline contrast to compare activation between sleep groups (Table 2). There were no areas in the whole brain analysis that showed a significant difference in immediate vs. delayed choice contrast; consequently this contrast was not compared between groups. Poor and good quality sleep groups were compared with t-tests using SPSS (version 21.0).

3. Results

3.1 Behavioral Results

Group comparisons for the eating, sleep, and impulsivity assessments are included in Table 1. Groups differed significantly on several of the PSQI sleep variables and on night eating behavior. There were no statistically significant differences between groups on delay discounting slopes.

To confirm that we tailored the delay-discounting task during fMRI to each participant based on their choices during the pre-scan behavioral task, we examined the percentage of fMRI trials where participants chose the immediate and delayed options to ensure the equal balance of trial type across participants. Overall, participants chose the immediately available option about 53% of the time and the delayed option about 47% of the time.

3.2 fMRI Results

The contrast of immediate, smaller monetary choice versus baseline condition indicated significant percent signal change in the middle frontal gyrus, medial frontal gyrus, inferior frontal gyrus, insula, lentiform nucleus/medial globus pallidus, and cingulate (Table 2). The difference in brain activation (percent signal change) when choosing immediate, smaller monetary rewards (impulsive decisions) for each brain region was then compared by sleep group.

Poor quality sleepers, compared to good quality sleepers, demonstrated statistically significantly decreased brain activation in several regions, including the right inferior frontal gyrus, right middle frontal gyrus, and bilateral insula. These are illustrated in Table 3.

¹We chose not to focus on Delay Baseline contrasts because choices in which participants chose the delayed option did not represent impulsive decisions. In preliminary analyses, however, eight prefrontal and limbic brain regions demonstrated significant percent signal change in the Delay Baseline contrasts. Of those brain areas, only activation in the right inferior frontal gyrus (46, 9, 31) was statistically significantly different between poor and good quality sleep groups ($p = 0.018$), with the good quality sleepers demonstrating greater brain activation than the poor quality sleepers.

4. Discussion

The purpose of this pilot study was to determine if the brain activation of obese good and poor quality sleepers differs in prefrontal and insular regions while engaged in impulsive decision making. We were also interested in comparing the eating behavior and delay discounting behavior of good and poor quality sleepers.

Behaviorally, obese adults classified as having poor sleep quality on the PSQI, compared to better quality sleepers, differed only in their degree of night eating syndrome symptomatology. Not surprisingly, poor quality sleepers reported clinically and statistically significantly higher scores on the NEQ (19.3) compared to better quality sleepers (6.7). An average score of 19 is not necessarily indicative of night eating syndrome, a pattern of non-normative eating behavior for which the core criterion are the consumption of 25% of one's total daily intake after the evening meal and/or awakening at night to consume food at least twice/week (Allison et al., 2010). Previous research has suggested that scores of 25 or above on the NEQ have a positive predictive value of 62% and a cut score of 30 has a positive predictive value of 77% (Allison et al., 2008). Nonetheless, these results suggest that obese adults with poor sleep quality do report greater NES symptomatology compared to those with better sleep quality.

Previous research by Trace and colleagues (2012) found that self-reported lack of sleep, poor quality sleeping, difficulty falling asleep, and daytime sleepiness predicted the endorsement of a lifetime history of binge eating among adults enrolled in the Swedish Twin Study of Adults: Genes and Environment study. In the current study we did not compare participants who endorsed binge eating to those who did not on sleep quality variables, but rather found no differences between binge eating symptomatology for good and poor quality sleepers.

Although there were no sleep group differences in slope of the delay discounting behavioral data, the fMRI data revealed that 1) when comparing the selection of smaller, immediate rewards (impulsive decisions) to the baseline condition of viewing a fixation cross, the task activated brain regions that have been found in other fMRI delay discounting studies and 2) when choosing smaller, immediate rewards in contrast to the baseline condition, interesting differences in brain activation emerged between good and poor quality sleepers. As noted, brain regions associated with delay discounting include the prefrontal cortex, insular cortex, ventral striatum, cingulate cortex, parietal cortex, and midbrain (McKell Carter et al., 2010). In the current study, we focused on activation in prefrontal, insular, and limbic regions and found significant activation in the middle frontal gyrus, medial frontal gyrus, inferior frontal gyrus, insula, lentiform nucleus/medial globus pallidus, and cingulate when participants made immediate, smaller rewards (vs. baseline).

Consistent with our hypothesis, good and poor quality sleepers demonstrated different patterns of neural activation when making impulsive monetary choices during the delay discounting task. Specifically, good quality sleepers showed greater activation than poor quality sleepers in the right inferior frontal gyrus, right middle frontal gyrus, and bilateral insula. Our interpretation the current findings is that poor sleep quality might down-regulate

brain activation in multiple regions that regulate cognitive control and sensory processing, thereby reducing self-regulatory capacity when making immediately gratifying decisions. For example, the right inferior frontal gyrus has been associated with response inhibition difficulties, and brain stimulation in this region improved cognitive control in healthy adults on a stop signal task (Jacobson et al., 2011). Less activation in the inferior and middle frontal gyri during a delay discounting task has also been associated with weight gain over time in obese females (Kishinevsky et al., 2012).

The insular findings in the current study are particularly interesting because of the insula's role in integrating perception, emotion, interoception, hedonics, cognition, and food-related processes (Frank et al., 2012). Although the current study was not designed to assess a causal relationship between sleep quality and eating behavior, decreased activation in the insular cortex of poor compared to good quality sleepers supports our conceptual model that poor sleep quality could increase risk of obesity through neural and behavioral processes that affect energy intake. The current findings, however, are difficult to compare with much of the literature on obesity and insular activity because many of the previous studies have used food cue stimuli.

This study is the first of which we are aware to use fMRI to examine self-regulatory capacity among obese adults with poor sleep quality. It is particularly important because, in contrast to much of the sleep and obesity literature that has examined acute sleep restriction in healthy young adults, we assessed sleep quality in an ecologically valid sample of obese adults. Despite the benefits of using a community sample and the significant findings, the study was limited by small sample size, lack of male participants in the poor sleep quality group, and reliance on self-reported eating and sleep behaviors. Post hoc analyses compared the male ($n = 3$) and female ($n = 3$) good quality sleepers on the percent signal change when making immediate monetary decisions vs. baseline and no statistically significant differences were found (all $ps > 0.05$). In addition, it is possible that participants who reported poor sleep quality had undiagnosed sleep disorders or obstructive sleep apnea that could have been identified with polysomnography. Sleep quality, particularly for those who suffer from insomnia, however, is a subjective experience; polysomnography is of limited value in its assessment (Schutte-Rodin et al., 2008). We did collect actigraphy data on all participants, but did not include it in the current report because a few of the participants had valid actigraphy data on only a few nights (out of seven); consequently, we chose to limit our classification of sleep to the PSQI, a validated sleep quality assessment, so that we could maximize the number of participants included in the analyses. We did not conduct a full psychiatric assessment on our participants, and it is possible that sleep quality was confounded by mood or anxiety disorders. Our sleep groups, however, did not differ on level of depressed mood assessed with the NEQ ($p > 0.05$). We did not rule out participants for occasional sleep medication use, although post hoc analyses showed that medication use was not significantly correlated with brain activation. Future studies of poor quality sleepers who are medication free would be beneficial to determine if sleep medications affect brain activation during the delay discounting task. Finally, we did not see a difference in brain activation when comparing immediate to delayed choices. This is similar to previous studies looking at delay discounting in addiction that show no difference in brain activation between immediate compared to delayed choices (Monterosso et al., 2007).

Despite these limitations, we observed several interesting differences in eating behavior and brain activation between the sleep groups. These data suggest that better sleep quality could lead to increased self-regulation when making decisions, and consequently improve eating behavior. Future studies that manipulate sleep quality (e.g., examine brain activation before and after treatment for insomnia) and assess changes in eating behavior, particularly night eating behavior, are necessary to test this hypothesis.

Acknowledgments

This work was supported by a CTSA grant from NCATS awarded to the University of Kansas Medical Center for Frontiers: The Heartland Institute for Clinical and Translational Research #UL1TR000001 (formerly #UL1RR033179). The contents are solely the responsibility of the authors and do not necessarily represent the official views of the NIH or NCATS.

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Highlights

- Poor quality sleepers have decreased brain activation in self-regulatory regions
- Poor quality sleepers report greater night eating syndrome symptomatology
- Poor sleep quality is associated with reduced self-regulatory capacity

Figure 1.

Example of delay discounting trial presentation. A fixation cross was presented as a baseline for 3–12 seconds to jitter trial presentation. Participants indicated with a key-press whether they preferred the option on the left or right.

Table 1

Comparison of Good versus Poor Quality Sleepers on Demographic, Sleep, Eating Behavior, and Impulsivity Variables.

Variable	Good Quality Sleepers (N = 6)	Poor Quality Sleepers (N = 13)	Significance t (df), p or χ^2 (df), p
	Mean $\pm$ SD (Range) or n (%)	Mean $\pm$ SD (Range) or n (%)	
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 = $p > 0.05$.

Table 2
Prefrontal and Insular Regions Reaching Significance during Contrasts of Immediate Monetary Reward vs. Baseline

Region and contrast	Peak Talairach Coordinates						
	L/R	BA	X	Y	Z	Voxels	t
Inferior frontal gyrus	R	9	46	9	31	205	6.98
Middle frontal gyrus	R	46	41	34	21	211	6.18
	R	6	36	-1	49	149	6.42
	L	9	-51	9	36	252	6.58
	L	46	-46	41	24	62	5.65
Medial frontal gyrus	L	32	-1	9	46	752	8.49
Insula	R	13	44	14	1	198	8.25
	L	13	-34	16	9	153	11.68
	L	13	-36	-24	19	32	5.37
	L	13	-34	-4	14	27	6.34
Lentiform nucleus/medial globus pallidus	L	NA	-11	1	1	111	7.68
Cingulate	L	24	-1	4	31	23	5.56

Note. L = left; R = right; BA = Brodmann Area; all FDR < 0.05

Comparison of poor and good quality sleepers on brain activation when making immediate, smaller monetary choices versus baseline.

Table 3

Brain Region (X, Y, Z)	df	Mean Difference	Std. Error Difference	t	Sig. (2-tailed)
Right inferior frontal gyrus (46, 9, 31)	17	0.22	0.09	2.41	0.03
Right middle frontal gyrus (41, 34, 21)	17	0.11	0.06	2.00	0.06
Right middle frontal gyrus (36, -1, 49)	17	0.13	0.05	2.76	0.01
Left middle frontal gyrus (-51, 9, 36)	17	0.02	0.10	0.22	0.83
Left middle frontal gyrus (-46, 41, 24)	17	0.15	0.13	1.20	0.25
Left medial frontal gyrus (-1, 9, 46)	17	0.13	0.09	1.49	0.16
Right insula (44, 14, 1)	17	0.19	0.07	2.67	0.02
Left insula (-34, 16, 9)	17	0.14	0.06	2.51	0.02
Left insula (-36, -24, 19)	17	0.10	0.08	1.29	0.21
Left insula (-34, -4, 14)	17	0.22	0.06	3.72	<0.01
Left lentiform nucleus/medial globus pallidus (-11, 1, 1)	17	0.12	0.06	1.92	0.07
Left cingulate (-1, 4, 31)	17	0.08	0.06	1.40	0.18