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Reward-Related Brain Function and Sleep in Pre/Early Pubertal and Mid/Late Pubertal Adolescents

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Abstract

Purpose—The onset of adolescence is a time of dramatic changes, including changes in sleep, and a time of new health concerns related to increases in risk-taking, sensation-seeking, depression, substance use, and accidents. As part of a larger study examining puberty-specific changes in adolescents' reward-related brain function, the current paper focuses on the relationship between functional neuroimaging measures of reward and measures of sleep.

Methods—58 healthy participants age 11-13 completed a functional magnetic resonance imaging scan using a guessing task with monetary rewards and four-days of at home actigraphy and self-reported sleep ratings. Sleep variables included actigraph measures of mean weekend *minutes asleep*, *sleep onset time*, and *sleep offset time*, as well as self-reported *sleep quality*.

Results—During reward anticipation, less activation in the caudate (part of the ventral striatum) was associated with fewer *minutes asleep*, later *sleep onset time*, and lower *sleep quality*. During reward outcome, less caudate activation was associated with later *sleep onset time*, earlier *sleep offset time*, and lower *sleep quality*.

Conclusions—It has been hypothesized that adolescents' low reactivity in reward-related brain areas could lead to compensatory increases in reward-driven behavior. This study's findings suggest that sleep could contribute to such behavior. Because decreased sleep has been associated with risky behavior and negative mood, these findings raise concerns about a negative spiral whereby the effects of puberty and sleep deprivation may have synergistic effects on reward processing, contributing to adolescent behavioral and emotional health problems.

Keywords

adolescence; puberty; sleep; fMRI; reward

Introduction

The onset of adolescence is a time of dramatic physical, cognitive, emotional, behavioral, and social changes. With puberty also comes a new set of health concerns—rapid increases in rates of risk-taking, sensation-seeking, depression, substance use, and accidents. Accordingly, there is growing interest in understanding the neurobehavioral underpinnings

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of these changes, with a particular focus on pubertal increases in risk-taking and sensation-seeking [1,2].

The onset of adolescence is also a time of both physiological and social changes that impact sleep [3]. Of particular concern is evidence that sleep deprivation appears to be nearly epidemic among adolescents [4] along with a growing recognition of the importance of sleep for physical health and cognitive and affective function. This myriad of changes in early adolescence raises a series of questions about the interactions between domains. One step toward understanding these important questions is to examine the interrelationships among pubertal maturation, neural systems of reward, and sleep patterns in healthy adolescents—the focus of this paper.

Health risks associated with reward-related changes at puberty

Puberty is strongly associated with an increase in health consequences related to risky behaviors including substance use, accidents, and sexual behavior [5,6]. Part of this increase in risky behavior appears to reflect pubertal increases in sensation-seeking [2,6] (which is hypothesized to be related to some maturational changes in aspects of reward seeking). Thus, changes in neural systems of reward processing in adolescence may underpin these behavioral tendencies [7], and there is growing interest in understanding puberty-specific changes in these systems [5,8]. A second area of health problems that may be related to pubertal changes in reward systems is the large increase in the incidence of affective disorders at adolescence [9-11]. There is a growing body of evidence showing that sleep problems predict the onset of depression across the lifespan and evidence of a bidirectional relationship between sleep and mood [12,13]. Moreover, it was recently shown prospectively that chronic insomnia in adolescents increases the risk of affective problems [14], making a study of the relationship between reward and sleep even more relevant.

Adolescent sleep

According to the model developed by Carskadon [3], physiological and psychosocial factors combine in adolescence to make sleep later, shorter, and different between weekends and weekdays. There is also a tendency for a delay in the circadian timing of sleep [3,15,16]. This natural preference for a more delayed sleep timing typically manifests as later sleep onset and offset and decreasing amounts of sleep at night [3,17]. Taken together these contribute to a well-recognized set of problems in adolescents: late night and erratic sleep/wake schedules and insufficient sleep on school nights. Not only do adolescents typically obtain less nighttime sleep than children [4,15], they also tend to be sleepier even if they obtain as much sleep as children [18], suggesting that they need more sleep. This raises further concerns about the short and long-term health consequences from obtaining insufficient sleep in adolescence—with a particular emphasis on mood, irritability, risk-taking, and accidents [12,13,19-21].

Sleep, reward, and risk-taking

Studies examining the relationship between sleep and decision-making [19] have reported that insufficient sleep is associated with changes in reward-related decision making: people take greater risks and are less concerned with negative consequences [20]. However, most of these studies have focused on adults and on measures in laboratory settings. These effects may be amplified in adolescence with the high rates of both risk-taking and sleep deprivation. Therefore, it is important to investigate these questions in adolescents in natural sleeping environments.

Neural correlates of reward processing in adolescents

The striatum, part of the brain's basal ganglia [22], has been shown to be an important region for reward-related brain function, including positive emotions, motivation to pursue reward and response to reward [23-25]. The caudate, putamen and nucleus accumbens are parts of the well-connected striatum [22], which undergoes structural and functional change during adolescence; it has many gonadal steroid receptors and develops over the course of puberty [26]. Adolescents exhibit reward-related brain function in many striatal regions, with several studies reporting differing striatal reactivity to reward in adolescents compared to other age groups [7,27]. A limited number of studies have looked at the relationship between puberty and striatal reactivity [8].

Hypotheses

Because age and puberty are correlated in adolescents and age is measured more precisely and easily, many studies do not disentangle the effects of age from puberty-specific effects [7,27]. Given our interest in puberty-specific changes in neural systems of reward, this study was designed from the outset to allow us to directly examine the effects of pubertal maturation by recruiting subjects in a narrow age range, but varying in pubertal development. In a parallel paper from this study (focusing on puberty-specific changes in reward processing) adolescents who were mid/late pubertal showed less reactivity in reward systems compared to their pre/early pubertal counterparts *and* compared with adults [8]. In that paper, we interpreted our findings as suggesting that less striatal reactivity to reward could lead pubertal adolescents to seek greater levels of excitement. Based on those previous findings, we hypothesized that with sleep characteristics associated with adolescent maturation would be associated with alterations in striatal reactivity in response to reward. Because of the possible influence of pubertal development on both sleep and reactivity to reward, we included sexual maturation in our model for sleep and reward processing.

Method

Participants

All participants provided informed consent according to the guidelines of the University of Pittsburgh Institutional Review Board. Adolescents were recruited from the community through advertisements, flyers, and demographically targeted phone lists. Participants were recruited to be in a narrow age range (11-13 years) but vary in pubertal development. Because on average girls are mid-puberty around age 11 and boys are around age 12 [28-30], girls were recruited to be 11-12 years old, and boys to be 12-13 years old (Final Sample: girls $M=11.84$, $SD=.12$; boys $M=12.95$, $SD=.12$). Adolescents were free of current and lifetime psychiatric disorders, did not have braces, and had no history of head injury, serious medical illness, psychotropic medication use, alcohol use, or illicit drug use.

128 adolescents were enrolled in the larger study, however subjects were excluded for excessive head movement during the scan ($n=21$), claustrophobia ($n=3$), and missing actigraphy data ($n=25$), resulting in a final sample of 58 adolescents for the analyses reported in this paper (Table 1).

Materials

Pubertal development—Adolescents underwent physical examination by a trained research nurse to determine stage of pubertal development [31], because it is an inexpensive, biologic measure of puberty. Consistent with our prior approach to examining affective aspects of pubertal development [32], our *a priori* approach was to classify participants as *pre/early pubertal* if they were Tanner stage one or two and as *mid/late pubertal* if they

were Tanner stage three, four, or five on the Tanner scale that assesses breast/genital development, an index reflecting changes in levels of gonadal steroids, which influence neural development and affect-related brain function [33]. We chose to use the Tanner Breast/genital scale because gonadal changes are part of the main HPA axis in both sexes, and therefore may represent something more central to puberty. 34% of this sample was classified as pre/early pubertal adolescents.

Actigraphy-measured sleep—Actigraphy was conducted over two weekday nights and two weekend nights in participants' home environments, using Octagonal Basic Motionlogger Actigraphs (Ambulatory Monitoring, Ardsley, NY).

Self-reported sleep—Participants completed daily sleep diaries [34] to report time they went to bed, time they fell asleep, who woke them up, how well they slept, and how difficult it was to wake up. One of the visual analogue scale variables from the sleep diaries, *sleep quality* (assessed from very bad to very good) was included in our analyses.

Reward processing—The fMRI paradigm [10] was an adaptation of a card-guessing paradigm [35] to probe striatal response to reward, with a section to measure anticipation of reward as well as outcome.

During the first three seconds of each 27s trial, participants had to guess, via button press, whether the value of a visually presented card with a possible value of 1-9 was higher or lower than five (*decision making phase*). During the next 12 seconds, the trial type (either *reward/neutral* or *loss/neutral*) was presented visually. (During a *reward/neutral* trial it was impossible to lose money and during a *loss/neutral* trial it was impossible to win money; *anticipation phase*.) This was followed by the “actual” numerical value of the card (500ms); outcome feedback (a green upward-facing arrow for win, a red downward-facing arrow for loss, or a yellow circle if they did not win or lose money that trial; 500ms); and a crosshair presented for 11s (*outcome* includes the actual value, outcome feedback and first 8 seconds of the crosshair). The *baseline* condition is the final three seconds of staring at the crosshair before the next trial commences. Trials were presented in four runs, with 12 trials per run and a balanced number of outcome trial types within runs.

As has been previously done with this task [10,35], participants were told that they would receive \$1 for each win, lose 50 cents for each loss, and experience no earnings change for neutral outcomes. Participants were unaware of the fixed outcome probabilities (each participant had \$12 of winnings). During practice and between runs, participants' engagement was maintained by verbal encouragement to stay on task. To maximize sample size, data from only run one were included in analyses. Also, focusing on run one minimizes the influences of fatigue and habituation that can occur with repeated runs [10].

Procedure

Each participant completed a lab session including an fMRI scan and training in using the actigraph and completing sleep diaries. The 4-day sleep study took place on the following weekend (90% after scan; $M=2.86$ days, $SD=3.19$). We verified that the subjects who completed the sleep study before the scan did not systematically bias our results.

Data Scoring, Processing and Reduction

Actigraphy-measured sleep—Participants were instructed to wear the actigraph on their non-dominant wrist from Friday afternoon at 4 pm until they awoke Tuesday morning, removing it only for contact sports, swimming, or bathing. Actigraphs recorded

continuously. Subjects pressed a button to indicate when they were trying to go to sleep and when they woke up, which inserted a marker into the actigraph record.

Actigraphy data were preprocessed and scored in 60-s epochs using Action W 2.5. *Sleep onset* and *sleep offset* times were determined using the actigraph record, supported by the button-press marker and sleep diary. Data were processed using the Cole-Kripke procedure [36]. Coders were trained by scoring records collectively, then by individually scoring the same records, comparing and discussing discrepancies.

Actigraphy variables used were mean *minutes asleep*, mean *sleep onset*, and mean *sleep offset* across the 2 weekend nights. *Minutes asleep* was operationalized as the total epochs of actigraphy-recorded sleep during time in bed. *Sleep onset* was the first minute of several contiguous periods of low activity scored as sleep. *Sleep offset* was the last minute of low-activity scored during the night. As would be expected, some of the sleep variables correlated with each other. *Sleep Onset Time* correlated with *Sleep Offset Time* ($r=0.541$), *Minutes Asleep* correlated with *Sleep Offset Time* ($r=0.259$), and *Minutes Asleep* correlated with *Sleep Onset Time* ($r=-0.565$). Although we collected four nights of data, the variability of the weeknight data (some adolescents were on school schedules while some were on holiday schedules) led us to focus exclusively on the weekend data, allowing all subjects to be studied on more natural self-selected schedules. We verified that the subjects who were on holiday schedules did not bias our results. (See Table 2 for sleep variables by development and gender.)

Self-reported sleep—The variable used in data analyses, intended to capture the adolescents' subjective experience of sleep, was mean weekend *sleep quality* ($M=77.3$, $SD=15.5$).

Reward processing—Each participant was scanned using a Siemens 3T Allegra scanner. Blood-Oxygenation-Level-Dependant-Response functional images were acquired with a gradient echo planar imaging (EPI) sequence and covered 34 axial slices (3mm thick) beginning at the cerebral vertex and encompassing the entire cerebrum and the majority of the cerebellum (Time to Repetition/Time to Echo = 2000/25 ms, Field Of View= 20 cm, matrix = 64×64). For each participant, we first acquired a reference EPI scan and visually inspected it for artifacts (e.g., ghosting) and for good signal across the entire volume of acquisition.

Whole-brain image analysis was completed using SPM2 (Statistical Parametric Mapping) (<http://www.fil.ion.ucl.ac.uk/spm>). For each scan, images for each participant were realigned to the first volume in the time series to correct for head motion. We confirmed that each participant's data reflected <4 mm or degrees of motion. Realigned images were spatially normalized into a standard Montreal Neurological Institute template space using a 12-parameter affine model. Normalized images were smoothed to minimize noise and residual difference in gyral anatomy with a 6mm full-width at half-maximum Gaussian filter. Voxel-wise signal intensities were ratio normalized to the whole-brain global mean.

Preprocessed data sets were analyzed using second-level random effect models that account for both scan-to-scan and participant-to-participant variability to determine task-specific regional responses. For each participant and scan, predetermined condition effects at each voxel were calculated using a *t*-statistic, producing a statistical image for each of the two contrasts of interest: (1) reward anticipation > baseline and (2) reward outcome > baseline. Group-level analyses were thresholded at a voxel level of $p<.05$ and an extent of at least 10 contiguous voxels; masked for the effects of the task across the striatal region of interest (ROI); and corrected for multiple comparisons with False Discovery Rate using a functional

mask within the activation clusters of interest. Our striatal ROI was constructed using the PickAtlas Tool (v1.04) in SPM2 and defined as a sphere with 20mm radius, centered on Talairach coordinates (0,10,-10) [37] that encompassed the entire ventral striatum and adjacent regions of the caudate nucleus.

Data Analysis—A general linear model was used in SPSS (v.16.0.1) to analyze the relationship between each sleep variable, gender, and development (GLM Univariate with sleep variable as the dependent variable and gender and development as fixed factors.) Response to reward was examined in SPM using regressions, with the sleep variable as the independent variable, and gender and development as covariates. Because of multicollinearity between age and puberty, age was not included in the final model. The results of the SPM regressions were exported to SPSS for traditional regression analyses yielding R^2 .

Results

Development and sleep

Pubertal maturation was associated with fewer total *minutes asleep* ($F = 5.72$, $df = 1$, $p = 0.020$, $M_{\text{early}} = 542.2$, $SD_{\text{early}} = 38.2$, $M_{\text{late}} = 503.5$, $SD_{\text{late}} = 61.3$), with adolescents who were mid/late pubertal obtaining about 39 minutes less sleep time on average than their pre/early pubertal counterparts. There was considerable variation in sleep time in both groups. Development was not associated specifically with *sleep onset*, *sleep offset*, or *sleep quality*. There were no gender differences evident.

Actigraphy-measured sleep and reward-related brain function

In the reward anticipation phase, subjects with fewer *minutes asleep* and later *sleep onset time* exhibited less caudate activation (Table 1, Figure 1a). In the reward outcome phase, subjects with later *sleep onset time* showed less caudate activation, but later *sleep offset time* was associated with *greater* caudate activation (Table 1, Figure 1b). There were no significant sleep by development or sleep by gender interactions.

Self-reported sleep and reward-related brain function

In both the reward anticipation and outcome phases, lower caudate activation was associated with lower subjective *sleep quality* (Table 1, Figure 2). There were no significant sleep by development or sleep by gender interactions.

Discussion

This study provides preliminary evidence of potentially important links between reward-related brain function and sleep during a period of maturational changes to both systems. Getting less sleep or subjectively worse sleep was associated with less striatal reactivity to reward. Also, participants who went to bed later had less striatal reactivity to reward. The overall pattern is that the sleep pattern associated with adolescence—that is, lower quantity and quality of sleep—is associated with less reactivity of reward-related brain systems.

It is currently debated in the developmental neuroscience literature whether adolescents have increased or decreased striatal reactivity in response to reward. Models, such as the Triadic [5], have been proposed to account for findings of increased reactivity, while other investigators have found decreased reactivity in adolescence [27] and related to puberty [8]. At the simplest level, the data in this paper are consistent with the hypothesis that less reactivity in reward systems may contribute to real-life patterns of increased risk-taking behavior in adolescence. That is, pubertal adolescents may require more exciting rewards in

order to create the same level of neural activation as pre-pubertal adolescents, and thus be more prone to risk-taking and sensation-seeking. This explanation could also fit with the seemingly paradoxical observations that the onset of adolescence is not only a time of greater sensation-seeking but also a time when youth often complain of feeling bored [38]. Thus, the findings in this paper are consistent with the idea that insufficient sleep may exacerbate low-positive-affect in ways that may have important health consequences.

Taken together, the initial findings suggest that getting less sleep in adolescence could represent a key element in a negative spiral of health-relevant effects. Obtaining less sleep may impact neural systems of reward in ways that exacerbate mood and behavioral problems. It is noteworthy that the pattern of reduced striatal reactivity to reward is consistent with findings on reward-related brain function in adolescents with major depressive disorder [9,10]. More generally, these results raise several provocative questions, particularly about the direction of the relation between sleep and reward processing. For example, rather than sleep impacting reward systems, changes in reward systems may be influencing sleep (e.g., increased pursuit of late-night rewarding social activities could reduce total quantity of sleep). Another possibility is that a third factor changing with development—for example, alterations in dopamine system function with puberty—might impact both sleep and reward processing.

We also found that, contrary to our hypotheses, earlier wake time was associated with less striatal reactivity to reward. Because actigraphy-measured total sleep was correlated with actigraphy-measured wake-up time, it is possible that total sleep time, which is also reflected in earlier wake time, may be driving the pattern of association between sleep characteristics with brain function. In addition, our findings differ from those of the only extant study of sleep and reward-related brain function. In that study [39], which used an experimental sleep deprivation procedure in adults, participants showed no statistically significant difference in response to reward receipt after sleep deprivation.

It also is important to acknowledge several limitations to this study. These data are cross-sectional and correlational, and thus they do not allow any conclusions about directionality. Longitudinal data are needed to investigate the direction of influence between sleep and reward processing. This study is also limited by the use of only two nights of weekend sleep data, the lack of subjective data regarding in-scanner experience, and the absence of behavioral data on reward processing or risk-taking.

Although these results are preliminary, they raise concerns about a negative synergy of health risks emerging in early adolescence that center on sleep and reward. These include evidence that sleep deprived people take more risks [20], that puberty is associated with decreased reward activation and increased risk-taking [2], that puberty is associated with sleep changes leading to very high rates of sleep deprivation [3], and that puberty is associated with sharply increasing rates of depression, suicide, risk-taking, and substance use [1].

This study further highlights the need for conceptual advances and more comprehensive models for understanding the interrelationships between these developing regulatory systems in adolescents. While some conceptual models have been described [3,12], we are at an early point in understanding how these complex, overlapping systems of sleep/arousal and affect regulation mature across adolescence—and how puberty-specific changes in reward systems may play a key role in some aspects of these changes in ways that have clinical relevance.

By examining variables that have not previously been studied together—puberty, sleep in natural environments (using both objective and subjective measures), and reward-related

brain function—this study begins to illuminate interrelationships between three physiological processes that have major health implications in adolescence. By focusing on a narrow age range with considerable variability in pubertal development, this study addresses questions about sleep and reward-related brain function that are likely to be directly linked to puberty rather than age or social-setting differences (e.g., high school vs. middle school). Because of mounting concerns about adolescent sleep [4] and because sleep may be related to both depression and risk-taking [19,21,40], it will be important to promote a better understanding of the relationship between sleep and reward in young adolescents.

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Figure 1. Actigraphy-Measured Sleep

a. Regions of blood-oxygenation-level-dependent (BOLD) activation negatively correlated with onset time of sleep during reward anticipation are shown. The scatter plot depicts correlation between onset time of sleep and caudate activation during reward anticipation at the voxel of maximum intensity (Talairach coordinates: -4, -1, 17; cluster size = 125 voxels, $p < 0.001$, $t = 3.55$). R^2 represents the fit of the entire model (including development and gender), not just the relationship between sleep and brain activation.

b. Regions of BOLD activation positively correlated with offset time of sleep during reward outcome are shown. The scatter plot depicts correlation between offset time of sleep and caudate activation during reward outcome at the voxel of maximum intensity (2, 8, 11;

cluster size = 100 voxels, $p < 0.001$, $t = 3.53$). R^2 represents the fit of the entire model (including development and gender), not just the relationship between sleep and brain activation.

Figure 2. Self-Reported Sleep

Regions of BOLD activation positively correlated with sleep quality during reward outcome are shown. The scatter plot depicts correlation between sleep quality and caudate activation during reward outcome is graphed at the voxel of maximum intensity (-6, 6, 11; cluster size = 214 voxels, $p < 0.005$, $t = 2.86$). R^2 represents the fit of the entire model (including development and gender), not just the relationship between sleep and brain activation.

Table 1

Description of Sample Used in Study.

		Developmental Group		
		Pre/Early (n=20)	Mid/Late (n=38)	<u>Total</u>
Age		M= 11.76 yrs SD=0.17	M= 12.64 yrs SD= 0.12	M=12.34 yrs SD=0.86
Race	European American	16 (80%)	25 (65.8%)	41 (70.7%)
	African-American	1 (5%)	7 (18.4%)	8 (13.8%)
	Multi-Racial	3 (15%)	6 (15.8%)	9 (15.5%)
Gender		13 female (65%)	19 female (50%)	32 female (55%)

Note: Fisher exact analyses found no significant difference in race by development group within each gender.

Table 2

Comparison of Sleep Variables Between Groups.

Developmental Group	Gender	Actigraphy-measured Sleep			Self-Reported Sleep	
		Sleep Onset	Sleep Offset (hh:mm:ss)	Minutes Asleep	Sleep Rating	
Early	Male	M=23:29:47 SD=00:52:42	M=09:19:04 SD=00:30:27	M=538.8 SD=34.9	M=78.6 SD=14.8	
	Female	M=22:56:06 SD=00:50:07	M=08:41:13 SD=00:45:14	M=544.0 SD=41.1	M=76.9 SD=15.3	
	Both	M=23:07:53 SD=00:52:18	M=08:54:28 SD=00:43:55	M=542.2 SD=38.2	M=77.6 SD=14.7	
	Male	M=23:36:36 SD=00:26:44	M=08:43:07 SD=00:59:34	M=501.8 SD=72.2	M=75.8 SD=16.0	
	Female	M=23:38:32 SD=00:56:54	M=08:45:00 SD=00:58:16	M=505.2 SD=50.1	M=78.6 SD=16.6	
	Both	M=23:37:33 SD=00:12:21	M=08:44:04 SD=00:58:08	M=503.5 SD=61.3	M=77.1 SD=16.1	
Both	Male	M=23:34:46 SD=00:18:03	M=08:52:48 SD=00:55:09	M=511.8 SD=65.8	M=76.6 SD=15.4	
	Female	M=23:21:17 SD=00:57:27	M=08:43:28 SD=00:52:36	M=520.9 SD=49.9	M=77.9 SD=15.8	
	Both	M=23:27:20 SD=00:07:10	M=08:47:39 SD=00:53:29	M=516.8 SD=57.2	M=77.3 SD=15.5	

Table 3

Association of Sleep Measures with Reward-Related Striatal Function

Talairach coordinates of maximum voxel in cluster											
Brain Region	+ or - Correlation	Hemisphere	x	Y	z	Cluster size	t	p <	R ²		
Reward Anticipation											
Minutes Asleep											
Caudate	positive	Left	0	1	18	22	2.67	0.010	0.15		
Sleep Onset Time											
Caudate	negative	Left	-4	-1	17	125	3.55	0.001	0.21		
Caudate	negative	Left	-4	13	-2	48	2.79	0.005	0.18		
Sleep Offset Time											
(no suprathreshold clusters)											
Subjective Sleep Quality											
Caudate	positive	Left	-4	2	11	13	2.44	0.010	0.19		
Reward Outcome											
Minutes Asleep											
(no suprathreshold clusters)											
Sleep Onset Time											
Caudate	negative	Left	-10	5	15	53	2.66	0.010	0.16		
Caudate	positive	Left	0	10	9	36	2.64	0.010	0.12		
Caudate	negative	Left	-4	13	-2	40	2.64	0.010	0.14		
Caudate	negative	Right	10	5	15	40	2.56	0.010	0.12		
Sleep Offset Time											
Caudate	positive	Right	2	8	11	100	3.53	0.001	0.22		
Subjective Sleep Quality											
Caudate	positive	Left	-6	6	11	214	2.86	0.005	0.16		

Note: Results are from region of interest analyses focusing on the effects of task in the striatum. Development (i.e., early or late pubertal stage) and gender were included in the model as covariates. Reward anticipation and reward outcome were fMRI task conditions. Cluster threshold was 10 voxels, $p < 0.05$ with False Discovery Rate correction. $df = (1, 54)$ for objective sleep variables and (1, 49) for subjective sleep variables. The square of the correlation coefficient (R^2) was calculated for the full model (i.e. using gender, development and sleep to predict brain function).