Slow-Wave Sleep Disruption in Adolescence: Brain Responses to Monetary Reward and Loss

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A Thesis Submitted in Partial Fulfillment of the Requirements for the Degree of Bachelor of Science in BCN from the University of Michigan 2018

Author Note

This work was supported by the National Institute on Alcohol Abuse and Alcoholism (R21 AA022339).

Abstract

Among adolescents, there is an association between sleep deprivation and reward seeking, but more work is needed to better understand this association. The goal of this study was to investigate slow-wave sleep disruption (SWD) and activity in the nucleus accumbens (NAcc) during a task measuring reward processing. Participants were 28 healthy adolescents aged 15–17 years (50% female; 57.1% White) who underwent functional magnetic resonance imaging while performing a monetary incentive delay task to measure reward anticipation and feedback after a baseline (BL) night and a SWD night. There was greater activation of the NAcc during *feedback* of large loss and less activation of the NAcc during *anticipation* of large loss after the SWD night relative to the BL night. These results support an association between SWD and reward processing among adolescents. Knowing more about SWD and reward responsivity may provide enhanced treatment and support to adolescents exhibiting sleep problems and risky reward-related behaviors.

Keywords: monetary incentive delay task; nucleus accumbens; functional magnetic resonance imaging (fMRI); reward feedback; reward anticipation

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Adolescence is a developmental period associated with increased risky and reward-seeking behaviors, which are large contributors to adolescent mortality and substance use problems (CDC, 2014). These behaviors are controlled in part by the brain's reward system, which is highly sensitive during adolescence relative to childhood and adulthood. Due to high levels of risky behavior and sensation seeking typically displayed by adolescents, reward processing during adolescence is important to study.

Extensive research has identified a dual systems model of adolescent brain development that underlies heightened reward-seeking behavior in adolescents (Casey, Getz, & Galvan, 2008; Duckworth & Steinberg, 2015; Metcalfe & Mischel, 1999; Steinberg, Albert, Cauffman, Banich, Graham, & Woolard, 2008). This theoretical framework posits a developmental mismatch between the bottom-up reward system and the top-down cognitive control system. That is, the reward system—localized primarily in the ventral striatum (VS) (including the nucleus accumbens [NAcc]) and the medial and orbital prefrontal cortices—begins substantial development at puberty leading to heightened sensitivity during adolescence, whereas the cognitive control system—localized primarily in the lateral prefrontal, lateral parietal, and anterior cingulate cortices—undergoes more gradual development throughout the teens and twenties (Casey et al., 2008; Duckworth & Steinberg, 2015; Metcalfe & Mischel, 1999; Steinberg et al., 2008). This mismatch can lead to an increase in sensation seeking that influences risky behavior from early adolescence to early adulthood (Harden & Tucker-Drob, 2011; Steinberg et al., 2008).

Researchers have studied the reward system by looking for activation of the VS, particularly the NAcc. Specifically, functional magnetic resonance imaging (fMRI) studies have

shown that the NAcc responds to the anticipation and feedback of monetary reward (Bjork, Knutson, Fong, Caggiano, Bennet & Hommer, 2004; Bjork, Smith, Chen & Hommer, 2010). The monetary incentive delay (MID) task has been used as a tool to study brain activation associated with reward anticipation and feedback and consists of cues indicating the potential to win or to lose varying amounts of money based on the speed of the participant's response (Knutson, Adams, Fong & Hommer, 2001).

There are developmental differences in adolescence relative to childhood and adulthood that may alter brain activation and processing of reward and loss. For example, adolescents have been found to show less VS activation in response to reward anticipation compared to young adults ages 22–28 (Bjork et al., 2004). However, this reduced activation is not uniform across all aspects of reward response. Compared to young adults, adolescents also show greater activation in the NAcc during reward feedback (Bjork et al., 2010; Haber, 2011). This developmental difference provides important insight into why adolescents are such a high-risk population. The gap in the maturation of the adolescent brain leaves the reward processing system in a state of heightened sensitivity during a time when the cognitive processing system is not yet mature enough to compensate for the heightened reward response. This creates a period of vulnerability to rewards that encourage risky and reward seeking behavior (Blum, Febo, Panavotis, Baron, Fratantonio, & Gold, 2015).

In addition to these differences in brain function, adolescents' social lives and levels of activity may undergo various changes relative to childhood (Dijk, 2009; Kurth, Jenni, Riedner, Tononi, Carskadon & Huber, 2010; Wong, Brower, Fitzgerald & Zucker, 2004). These changes may be due to sports and other extracurricular activities, as well as school schedules starting

earlier in the day. These environmental changes can lead to a reduction in sleep during this important developmental period.

Effects of Sleep on Reward Processing

Sleep is universally known to restore and facilitate day-to-day cognitive functions. Sleep is made up of rapid-eye movement (REM) sleep and non-REM sleep. Slow-wave sleep consists of stages three and four of non-REM sleep, the deepest part of sleep as the brain becomes less responsive to external stimuli, as recorded by electroencephalography (EEG; Dijk, 2009; National Sleep Foundation, 2000; Roth, 2009;). This overall reduction and change in sleep during adolescence is also thought to decrease the brain's metabolic rate and plasticity (Feinberg & Carlson, 1968; Kanwal, Jung & Zhang, 2015). Because of this decrease in metabolic rate and plasticity, disruption of slow-wave sleep may be detrimental to overall health and cognitive functioning (Kanwal et al., 2015). As shown in previous research (Armitage, Béné, Charles, Johnson & Allison, 2012), the initial accumulation of slow-wave EEG activity after non-REM sleep onset is reduced and the rate of decay across the night is significantly slower in adolescents, meaning adolescents are initiating sleep later compared to other age groups (Armitage et al., 2012). As adolescents experience a heightened responsiveness to rewards, a change in the architecture of slow-wave sleep may increase reward seeking and risky decision-making in adolescents more than other age groups.

Given that adolescents may be primed toward reward seeking behavior due to the developmental mismatch between brain systems involved in reward processing and cognitive control (Dijk, 2009; Kurth et al., 2010; Wong et al., 2004)—and considering that sleep deficits may contribute to increased sensation seeking (Krause, Simon, Mander, Greer, Saletin, Goldstein-Piekarski & Walker, 2017; Mullin, Phillips, Siegle, Buysse, Forbes & Franzen, 2013;

Venkatraman, Chuah, Huettel & Chee, 2007)—it is important to better understand how sleep changes may impact reward processing. A combination of the biological changes of the brain and environmental changes of adolescence may lead to overall sleep deprivation leading to more susceptibility for risk-taking such as seeking out alcohol, drugs, and other risky and dangerous rewards.

There is a lack of literature on the association between slow-wave sleep disruption and risk-taking among adolescents. This gap is problematic because this knowledge could lead to important preventions that target sleep disruption and impairments in adolescents. The reward system shows sensitivity to sleep deprivation that may lead to risk taking behaviors and reward seeking among adolescents (Casey et al., 2008; Dijk, 2009; Roth, 2009), suggesting that disruption in the deepest stage of sleep, slow-wave sleep, may alter the reward system (Mullin et al., 2013). Previous literature has used the MID task to examine the association between sleep deprivation and NAcc activity during the anticipation and receipt of reward (Krause et al., 2017; Mullin et al., 2013; Venkatraman et al., 2007). However, very little is known about the association between slow-wave sleep disruption and reward in adolescents.

Present Study

To study the relation between reward responsivity and slow-wave sleep disruption, the present study examined neurobiological changes among a community sample of adolescents during the MID task after experiencing slow-wave sleep disruption (SWD). fMRI scans were conducted twice to study brain function during reward processing after both non-disrupted and slow-wave-disrupted sleep. Consistent with prior evidence suggesting that total sleep disruption is associated with changes in reward processing among adolescents, it is hypothesized that SWD

is related to heightened sensitivity of the reward system and can be seen by heightened activation of the NAcc during monetary gain and loss.

Method

Participants

Participants were 28 right-handed healthy adolescents from the community between the ages of 15 and 17 (M = 16.32 years old; SD = 0.84; 50% female). See Table 1 for demographic information.

Inclusionary criteria for this study included low levels of alcohol and other substance use (i.e., ≤ 15 lifetime alcoholic drinks, ≤ 3 alcoholic drinks on a single occasion, ≤ 9 lifetime uses of marijuana and none in the past month, no nicotine use in the past month, and no other lifetime illegal drug uses). In addition, participants had to have reached Tanner Stage 4 or 5 of physical development, indicating physical maturity (Tanner, 1962). Exclusionary criteria consisted of any substance use disorder diagnosis, acute or chronic medical/neurological illness, history of psychosis in self or first-degree relatives, current treatment with centrally active medications, history of head trauma with loss of consciousness greater than 5 minutes, pregnancy, or magnetic resonance imaging (MRI) contraindications such as metal implants or claustrophobia.

Each participant spoke fluent English and was medically and physically able to consent. All participants gave written consent/assent after explanation of the experimental protocol. Because participants were under the age of 18, at least one parent gave written informed consent. The study was approved by the University of Michigan Medical School IRB.

Assessment

All participants were tested for alcohol and drug use prior to each in-person session. In addition, an in-house substance use form was used to investigate frequency, quantity, and

duration of alcohol and other drug use. Example items include: “When was the first time you tried alcohol?”; “Have you ever tried nicotine?”; “What is your pattern of cannabis use?”; and “Have you ever used another drug on a regular basis, meaning 3 or more times per week for at least 4 weeks?”. Finally, the Kiddie-SADS Present and Lifetime Version (K-SADS) is a clinical structured interview and was used to assess psychiatric history (Kaufman, Birmaher, Brent, Rao, Flynn & Moreci, 1997).

Sleep Protocol

Participants completed three nights at the University of Michigan Sleep Lab: an adaptation sleep night, a baseline sleep night (BL), and a SWD night. Specifically, participants slept in the lab the first night to adapt to the sleep clinic and environment. The next night served as either the BL night or the SWD night (counterbalanced). One week later, participants completed their third night in the sleep lab (BL or SWD). Figure 1is an illustration and timeline of the study. The BL night allowed baseline information during uninterrupted sleep. During the BL and SWD nights, EEG was recorded from left and right frontal, central, occipital, and parietal electrode sites. The SWD night consisted of tones of 1000Hz delivered at an intensity of 40–100dB (intensity starting at 40 and increased by increments of 5dB) delivered through a speaker attached to the headboard of the bed. These tones were emitted when two delta waves appeared within 15 seconds according to the EEG. Participants also kept a sleep diary and wore an actigraph (Actiwatch-AW2, Respironics) during the study to record daily sleep habits and bed times.

fMRI paradigm

The mornings after the BL and SWD nights, participants were scanned with fMRI while performing a monetary incentive delay task (MID; Knutson et al., 2001) to assess the brain’s

response to the anticipation and feedback of monetary reward. The task consisted of two runs, each lasting 5 minutes. Each trial was 6 seconds and consisted of four events. During the first event (*incentive cue*), participants were presented with a monetary incentive cue (2000 ms) of five potential values (gain of $0.20, gain of $5.00, loss of $0.20, loss of $5.00, or no money at stake). During the second event (*anticipation cue*), participants were presented with a crosshair lasting 2000, 4000, or 6000 ms. The third event (*response target*) consisted of a target (200–300 ms) cuing the participants to press a button to receive or avoid losing money. During the fourth event (*feedback*), participants received the outcome of that trial informing them whether they received money, did not receive money, lost money, avoided losing money, or that no money was at stake. Gains and losses of $5.00 were designated as *large*, gains and losses of $0.20 were designated as *small*, and no money at stake was designated as *neutral*. The task is illustrated in Figure 2.

Trials presenting the opportunity to win or lose monetary incentives were presented in random order. Each condition (small reward; large reward; no money at stake; small loss; large loss) was presented twenty times for each participant. For each individual, reaction time was calculated during a practice run before the first fMRI scan and used for calibration to achieve an approximate success rate of 60%. Each participant kept the money that they earned during the task.

fMRI Data Acquisition and Analysis

Whole-brain blood oxygen level-dependent (BOLD) images were acquired on a 3.0T GE Signa scanner (GE Healthcare) using a T2*-weighted gradient echo sequence (repetition time [TR]=2000 ms; echo time [TE]=30 ms; flip angle=90°; field of view [FOV]=20 cm; 64×64 matrix; in plane resolution=3.12×3.12 mm; slice thickness=4 mm). In addition, a high-resolution

anatomical T1-weighted scan was obtained (TR=25 ms; minimum TE; FOV=26 cm; 256×256 matrix; slice thickness=1.2 mm). To minimize motion, a foam padding around the head was used and participants were instructed before the scan and between blocks on the importance of remaining still.

Preprocessing. An iterative algorithm reconstructed functional images, and motion was corrected using FSL v5.0.2.2 (FMRIB, Oxford, UK; Noll, Fessler, & Sutton, 2005; Sutton, Noll, & Fessler, 2003). Trials that exceeded 3 mm translation or 3° rotation were excluded. Statistical Parametric Mapping (Wellcome Institute of Cognitive Neurology, Oxford, UK) was used to preprocess images. Functional images were spatially normalized to the Montreal Neurological Institute (MNI) template and smoothed with a 6 mm full-width at half-maximum (FWHM) smoothing kernel. Low-frequency noise was removed with a high-pass filter (128 s).

First-level analysis. A general linear model was used to conduct individual-level analyses. Regressors for each condition type were convolved with the canonical hemodynamic response function: 1) incentive cue + anticipation cue (*anticipation*); 2) response target; and 3) feedback cue (*feedback*). Contrasts of interest were created to probe reward and loss anticipation and feedback: 1) large reward anticipation vs. neutral anticipation; 2) small reward anticipation vs. neutral anticipation; 3) large loss anticipation vs. neutral anticipation; 4) small loss anticipation vs. neutral anticipation; 5) large reward positive feedback vs. negative feedback; 6) small reward positive feedback vs. negative feedback; 7) large loss positive feedback vs. negative feedback; and 8) small loss positive feedback vs. negative feedback. In order to capture background noise and remove residual motion artifacts, white matter signal intensity and six motion parameters were modeled as nuisance regressors.

Region of interest. Given the issues associated with the circularity of statistical analysis when defining volumes of interest based on observed contrast activation, and considering the present study's focus in the NAcc, anatomic masks of the left and right NAcc were created as described previously (Bjork, Smith & Hommer, 2008; Yau, Zubieta, Weiland, Samudra, Zucker & Heitzeg, 2012) using the Build ROI function in MarsBaR (Brett, Anton, Valabregue & Poline, 2002). Mean beta weights for the left and right NAcc (separately) from each contrast were then extracted and exported into SPSS v22 for further analysis. Finally, for each contrast, the mean of left and right NAcc was calculated, giving one value for each participant for each contrast.

Analytic Plan

Paired-samples *t*-tests were used to compare NAcc activation at BL and SWD sessions for the following contrasts (see First-Level Analysis): large reward anticipation, small reward anticipation, large loss anticipation, small loss anticipation, large reward feedback, small reward feedback, large loss feedback, and small loss feedback.

Results

Correlations for anticipation variables in the MID task for both BL and SWD nights are displayed in Table 2. Specifically, sex was negatively correlated with nucleus accumbens (NAcc) activity during SWD small reward anticipation ($r = -.59$, $p < .001$) as well as SWD small loss anticipation ($r = -.40$, $p = .038$). NAcc activity during BL small reward anticipation was significantly positively correlated with BL large reward ($r = .48$, $p = .009$), BL small loss ($r = .79$, $p < .001$), and BL large loss ($r = .53$, $p = .004$). NAcc activity during SWD small reward anticipation was positively correlated with SWD small loss anticipation ($r = .63$, $p < .001$) and SWD large loss anticipation ($r = .62$, $p = .001$). NAcc activity during BL and SWD large reward anticipation were positively correlated ($r = .44$, $p = .020$). In addition, NAcc activity during BL

large reward anticipation was positively correlated with NAcc activity during BL large loss anticipation (r = .55, p = .003) and SWD large loss anticipation (r = .46, p = .017). NAcc activity during SWD large reward anticipation was positively correlated with SWD small loss anticipation (r = .46, p = .016) and SWD large loss anticipation (r = .69, p < .001). NAcc activity during BL and SWD small loss anticipation were not significantly correlated.

Correlations for feedback variables are displayed in Table 3. There was a positive correlation between NAcc activity during BL feedback of large reward and age (r = .40, p = .039). In addition, NAcc activity during BL feedback of large reward was positively correlated with SWD feedback of small reward (r = .58, p = .001). Activity during BL feedback of large loss and BL feedback of small reward were also significantly positively correlated (r = .40, p = .037). Finally, there was a negative correlation between activity during BL feedback of large loss and BL feedback of large reward (r = -.58, p = .002). There were no significant associations between BL and SWD feedback of large reward or between BL and SWD feedback of large loss.

Paired-samples t-tests for mean differences in NAcc activity for both BL and SWD nights are displayed in Table 4. Large loss anticipation values were significantly greater after BL than after SWD, $t(26) = 2.17$, p = .039. However, large loss feedback values were significantly greater after SWD than after BL, $t(25) = -2.86$, p = .008. There were no other significant mean differences between BL and SWD activations.

Discussion

This study examined NAcc activity measured during the MID task at BL and after SWD in a sample of healthy adolescents using fMRI. Overall, findings suggest an impact of SWD that affects activity in the NAcc during anticipation and feedback of monetary losses. In addition, this is the first study to show a significant change in NAcc activation after *slow-wave sleep*

disruption among adolescents, not just overall sleep deprivation as previous studies have revealed (Dijk, 2009; Roth, 2009). This work suggests that SWD in healthy adolescents may lead to altered responsivity to losses.

Loss Anticipation and Feedback

In the current study, we found that adolescents who had histories of low substance use differed in neurobiological activity between BL and SWD nights. Specifically, disruption of slow-wave sleep was associated with an increase in NAcc activity during loss feedback and a decrease in NAcc activity for loss anticipation. This increase in NAcc activity suggests sensitivity to the receipt of large loss, whereas the decrease NAcc activity suggests decreased sensitivity to the anticipation of a certain amount of loss. An increase in NAcc activity during loss feedback shows that adolescents who undergo SWD may be sensitive to losing a large amount of money. Therefore, SWD could contribute to higher sensitivity to losses and risk-taking in adolescence. However, a decrease in NAcc activity during loss anticipation shows that adolescents who undergo SWD may not be as motivated to respond to avoid loss. This sensitive response to loss has been found to be associated with altered motivation and inhibition (Patel, Stevens, Meda, Muska, Thomas, Potenza & Pearlson, 2013). Thus, altered responsivity to loss could perhaps be an early indicator of risky behavior and sensation seeking among adolescence.

Reward Anticipation and Feedback

In contrast to loss, participants did not differ in NAcc activation during reward anticipation or feedback after SWD versus BL nights. These findings are surprising, because both anticipation and feedback of reward have been shown to be sensitive to sleep deprivation (Mullin et al., 2013; Venkatraman et al., 2007). In a previous study, Venkatraman et al. (2007) found that 24 hours of sleep deprivation resulted in increased NAcc activation for anticipated

rewards and decreased activation in the NAcc following losses. They concluded that sleep deprivation might diminish the ability to learn from the negative consequences of risky behavior (Venkatraman et al., 2007). However, as demonstrated in the present study, SWD may play a different role compared to sleep deprivation in relation to reward anticipation and feedback.

While interpreting these results, it is important to note that participants were required to have low levels of substance use. According to Romer, Reyna, and Satterthwaite, (2017), youth who have experience with drugs and alcohol were more likely to be impulsive and sensation seeking than those who did not begin to use drugs between ages 13–15. Although the use of drugs and alcohol, as well as other risky behaviors, lead to dangerous consequences, adolescents tend to learn from these experiences as they mature (Romer et al., 2017). This pattern may suggest that those who do not use drugs or alcohol at an early age may be more sensitive to losses because of such a low history of alcohol use. Previous findings suggest that individual differences in lifetime alcohol or drug use may be predictors of risk-taking and reward-seeking (Davey, Yucel & Allen, 2008; Hankin, Mermelstein & Roesch, 2007). Adolescents with lower use of substances throughout their lifetime may continue to engage in low risk-taking and decreased sensation-seeking in their life (Romer et al., 2017), which may contribute to sensitivity toward losses rather than rewards.

Strengths and Limitations

A major strength of the present study was that participants were measured twice for the same task (i.e., after BL and SWD nights), which accounts for within-person variance. Because there was an fMRI scan conducted twice for the same individual, we have more confidence that individual differences in unmeasured variables such as anxiety or stress did not substantially impact the results (Keulers, Stiers, Nicolson & Jolles, 2015). Second, this study was, to our

knowledge, the first to observe SWD in relation to in NAcc activity, instead of overall sleep deprivation. Third, in order to reduce the effects of confounding factors that might affect neurobiological activity measured during fMRI scanning and increase validity of study results, none of the participants were currently taking any psychoactive medications or had any substance use disorders or sleep disorders. Psychoactive medications, heavy substance use, or sleep impairments could affect brain regions involving reward and loss, leading to results that may not be a result of the manipulation. Lastly, a strength of this study was that the study design allowed us to specifically disrupt the deepest stage of non-REM sleep, slow-wave sleep. This, in turn, allowed us to draw more specific conclusions about slow-wave sleep as well as how slow-wave sleep may affect reward processing in adolescents.

In light of these strengths, limitations of the study should be addressed. First, findings may not be generalizable, because the current sample came from a highly educated area and was majority White with no regular substance use, sleeps impairments, or mental illness. However, this study purposely required a healthy sample and screened for low substance use and no sleep impairments in order to avoid confounding variables that may affect sleep and brain function. Second, the present study did not control for covariates such as sex and race/ethnicity. However, the sex and race/ethnicity of the sample reflected the characteristics of the surrounding community.

Future Directions

Although it is beyond the scope of the current study, the high prevalence of alcohol use (Johnston, Miech, O'Malley, Bachman, Schulenberg & Patrick, 2018) and sleep impairments (CDC, 2017) in adolescence suggests that future research is needed to address potential associations between sleep and alcohol. For example, a longitudinal study following youth from

adolescence through early adulthood would be useful to track impacts of alcohol on sleep disruption and vice versa. Future studies should also examine whether these associations differ across samples of adolescents who have had history of substance use and compare to adolescents who have had minimal experiences of substance use in order to test dose effects from heavy alcohol use.

Conclusion

In sum, results from the present study indicate a link between SWD and NAcc response to both the anticipation and feedback of losses during a monetary reward task. Due to adolescent vulnerability to risk-taking, coupled with susceptibility to sleep disorders, this study offers important information on the association between SWD and reward processing during adolescence. Studies on slow-wave sleep and adolescents are needed to inform the medical community about the proper treatments and prevention strategies (e.g., how many hours of sleep should one get per night and how to best facilitate schedules) to provide to adolescents with sleep disorders. Educating youth and families about the importance of sleep may reduce the potential harms associated with risk-taking and sleep disorders.

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Figures

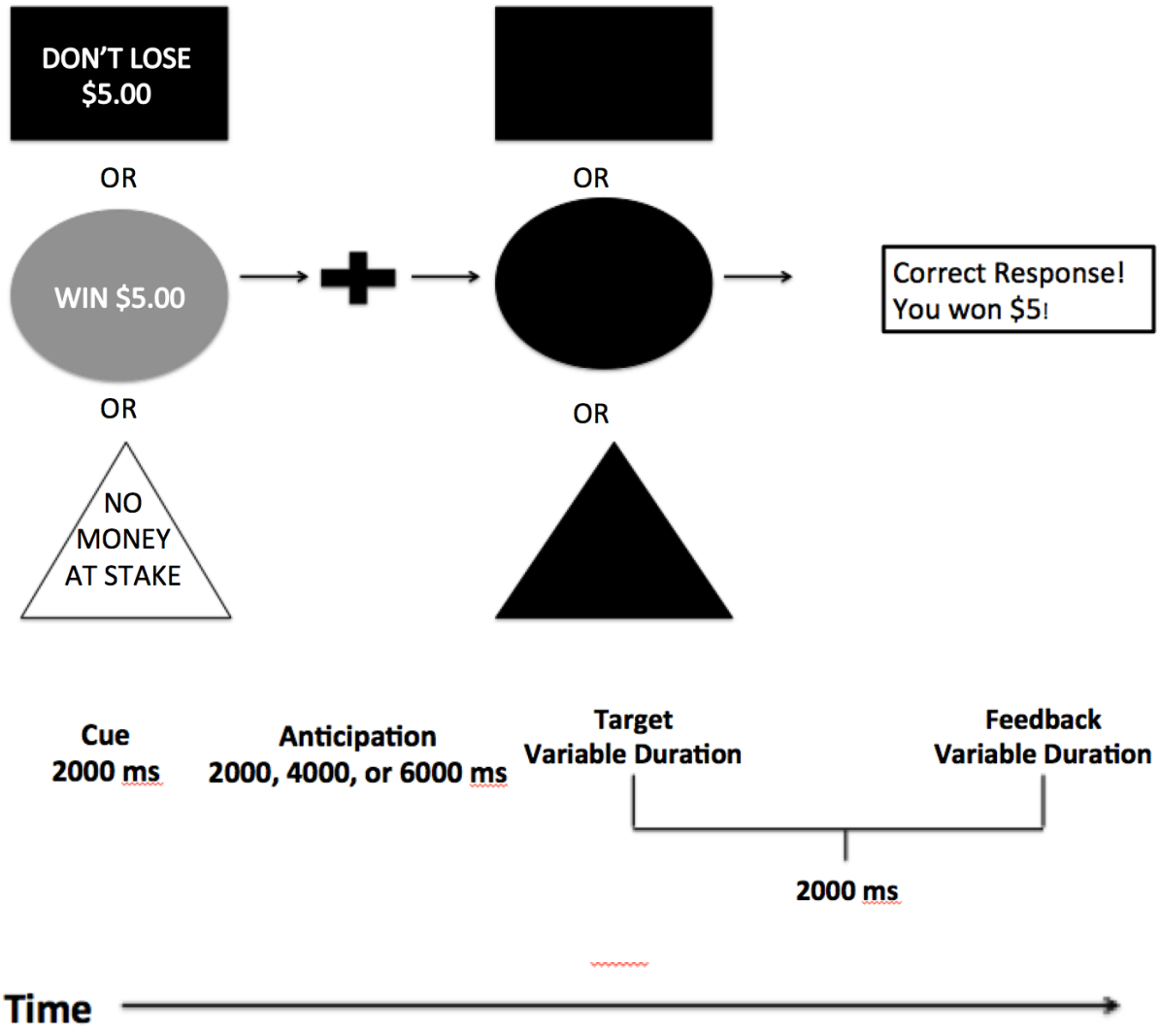

Figure 1. Monetary Incentive Delay Task

Figure 1: An illustration of the monetary incentive delay task performed by subjects in the functional magnetic resonance imaging scanner (adapted from Heitzeg, Zucker, Samudra, Weiland, Zubieta & Yau, 2012).

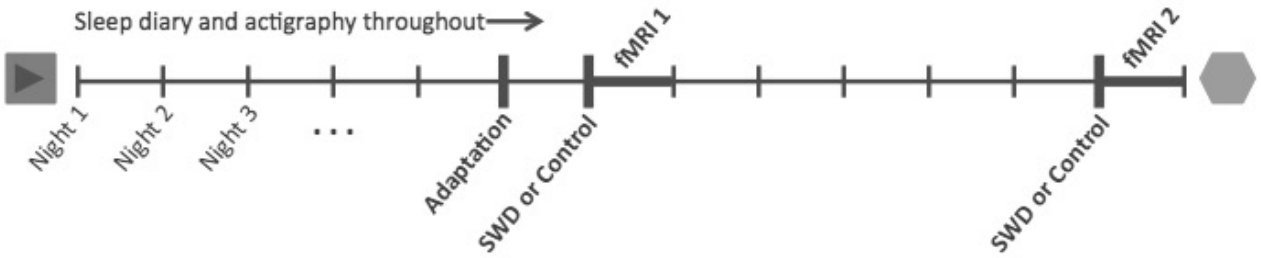

Figure 2. Sleep Lab Nights

Figure 2: An illustration of the sleep lab nights that each subject went through. Adaptation nights involved no experimental manipulation, which allowed subjects time to acclimate to the sleep lab environment and electroencephalography (EEG) equipment. Slow-wave disruption (SWD) nights involved disruption of slow-wave sleep only (counter-balanced with the baseline [BL] night). BL nights involved no experimental manipulation (adapted from Cope, Martz, Conroy, Cheng, Hoffman, Arnedt & Heitzeg, 2017). fMRI = functional magnetic resonance imaging.

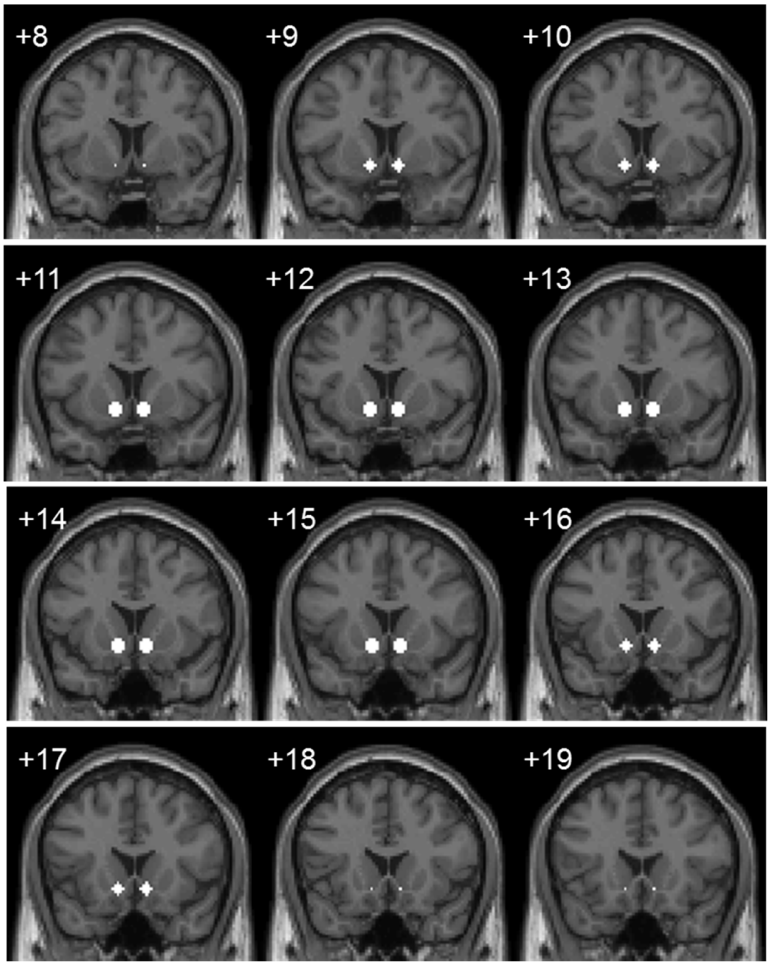

Figure 3. Left and Right Nucleus Accumbens (NAcc)

Figure 3: Statistical Parametric Mapping (SPM8) canonical single subject T1 coronal slices, with NAcc masks (5mm radius spheres centered around x = -10, y = 13, z = -8, and x = 10, y = 13, z = -8) overlaid. The numbers 8–19 indicate the Montreal Neurological Institute (MNI) y-coordinate of the slice (adapted from Martz, Trucco, Cope, Hardee, Jester, Zucker & Heitzeg, 2016).

Tables

Table 1
Participant Characteristics

	N
	28
Sex	
Females	14
Males	14
Age, mean (SD)	16.32 (0.84)
Ethnicity	
Hispanic/Latino	2
Not Hispanic/Latino	23
Race	
White	16
Black or African American	8
Asian American	1
Multiracial	2

Note. Three participants did not report ethnicity and one participant did not report race.

Table 2

Correlations Between Monetary Incentive Delay Task Anticipation and Demographic Variables

	1	2	3	4	5	6	7	8	9	10
1. Age (years)	1									
2. Sex (1=Female; 2=Male)	.05	1								
3. BL Small Reward	.02	-.27	1							
4. SWD Small Reward	.00	-.59***	.30	1						
5. BL Large Reward	.16	-.07	.48**	.54**	1					
6. SWD Large Reward	.18	-.24	-.01	.63**	.44*	1				
7. BL Small Loss	-.14	-.25	.79***	.08	.22	-.20	1			
8. SWD Small Loss	-.08	-.40*	.03	.63***	.12	.46*	.00	1		
9. BL Large Loss	.31	-.15	.53**	.24	.55**	.07	.47*	.02	1	
10. SWD Large Loss	-.12	-.23	.32	.62***	.46*	.69***	.21	.45*	.25	1

Note. Monetary incentive delay task variables (i.e., variables 3–10) reflect the mean of left and right nucleus accumbens activity for the given condition relative to a neutral condition. BL = baseline, SWD = slow-wave disruption.

*$p < .05$, **$p < .01$, ***$p < .001$

Table 3

Correlations Between Monetary Incentive Delay Task Feedback and Demographic Variables

	1	2	3	4	5	6	7	8	9	10
1. Age (years)	1									
2. Sex (1=Female; 2=Male)	.05	1								
3. BL Small Reward	.04	.02	1							
4. SWD Small Reward	.09	.03	-.01	1						
5. BL Large Reward	.40*	.08	.58**	.03	1					
6. SWD Large Reward	-.23	.17	-.36	.29	-.19	1				
7. BL Small Loss	.27	-.11	.19	.13	.08	-.10	1			
8. SWD Small Loss	.02	.07	.31	-.20	-.08	.04	.12	1		
9. BL Large Loss	.12	.02	.40*	-.34	.37	-.58**	-.21	.01	1	
10. SWD Large Loss	-.05	-.15	.27	.32	.18	.20	.02	.06	-.09	1

Note. Monetary incentive delay task variables (i.e., variables 3–10) reflect the mean of left and right nucleus accumbens activity for positive feedback of the given condition relative to negative feedback. BL = baseline, SWD = slow-wave disruption.

*$p < .05$, **$p < .01$

Table 4

Mean Differences in Nucleus Accumbens Activity During the Monetary Incentive Delay Task: Baseline versus Slow-Wave Disruption Nights

Anticipation

	BL	SWD	95% CI	*t*	df	Sig (2-tailed)
Small Reward	.28 (.70)	.30 (.67)	-.34, .31	-0.11	26	.914
Large Reward	.88 (.84)	.93 (.74)	-.38, .28	-0.29	26	.772
Small Loss	.43 (.87)	.20 (.70)	-.21, .67	1.07	26	.296
Large Loss	.70 (.75)	.35 (.62)	.02, .68	2.17	26	.039*

Feedback

	BL	SWD	95% CI	*t*	df	Sig (2-tailed)
Small Reward	.96 (2.24)	1.15 (1.26)	-1.21, .82	-0.40	26	.696
Large Reward	2.24 (2.40)	1.65 (1.61)	-.67, 1.86	0.97	25	.343
Small Loss	.97 (1.72)	1.42 (1.97)	-1.43, .51	-0.97	26	.343
Large Loss	.45 (1.19)	1.54 (1.42)	-1.87, -.31	-2.86	25	.008**

Note. Means (standard deviations) and the results of paired samples *t*-tests are given for the mean of left and right nucleus accumbens activity for each contrast (e.g., BL anticipation of small reward [relative to BL neutral anticipation]; SWD large loss positive feedback [relative to SWD negative feedback]). BL = baseline, SWD = slow-wave disruption, CI = confidence interval, *t* = *t*-statistic, df = degrees of freedom, Sig = significance.

*$p < 0.05$, **$p < 0.01$