



## Disruptions in the Amount and Timing of Sleep and Repetitive Negative Thinking in Adolescents

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### ABSTRACT

*Objective/Background:* Numerous psychiatric conditions characterized by repetitive negative thinking (RNT) are also frequently associated with disruptions in the duration and timing of sleep. The emerging literature supports that these types of sleep disruptions may be associated with negative psychological consequences such as depressed mood, anxiety, and poor emotion regulation, all of which have features of RNT. There is a paucity of research on the association between RNT and disruptions in sleep duration and timing in adolescents. The aim of the current study was to examine if sleep duration and timing in an adolescent sample would be predictive of RNT. *Participants:* Participants included 1,021 adolescents (ages 11 to 17) from a public school district in upstate New York. *Methods:* Participants completed a survey about their sleep practices, symptoms of psychopathology, and RNT. *Results:* Results indicated that sleep timing was predictive of RNT, but sleep duration was not. This result remained even after controlling for symptoms of psychopathology. Further, sleep onset latency was also predictive of RNT. *Conclusions:* These results indicate that it may be important to make the distinction between sleep duration and sleep timing. Sleep timing may uniquely impact RNT in adolescents.

Consistent with prominent theories of psychopathology, research has reliably shown repetitive negative thinking (RNT) to be a central characteristic of many psychiatric conditions (Beck & Clark, 1988; Borkovec, Alcaine, & Behar, 2004; Rachman, 1997). RNT can manifest in many ways including worry, rumination, and obsessing, but has the shared feature that it is exhibited as frequent negative thoughts that are difficult to control (Ehring & Watkins, 2008). In addition to being characterized by elevated RNT, many psychiatric disorders are also characterized by disruptions in the duration and timing of sleep (Apa, 2013; Boivin, 2000; Harvey, 2008, 2011; Wulff, Gatti, Wettstein, & Foster, 2010). Harvey, Murray, Chandler, and Soehner (2011) argue that sleep disturbance should be viewed as a transdiagnostic process, as it is a feature of most psychiatric conditions. For example, sleep disruptions are reported by up to 90% of those experiencing an acute depressive episode (Wulff et al., 2010). The high prevalence of sleep disruptions in individuals with elevated anxiety or mood dysregulation is consistent with increasing evidence that sleep disruptions are associated with impairment in cognitive, emotional, and somatic functioning (Wulff et al., 2010). This highlights the importance of appropriate sleep duration and timing for optimal psychological well-being. Some studies suggest that sleep duration and sleep timing may even play a role in the etiology of psychiatric conditions based on findings that sleep difficulties occurred before the onset of anxiety and depressive symptoms (Monti & Monti, 2000; Posmontier,

2008). Regardless of the etiology, sleep disturbances are likely to exacerbate psychological difficulties, which may make sleep a possible target for intervention and prevention.

There is growing evidence for relations between sleep disruption and RNT in both adults and adolescents. Sleep is an important factor to consider in developmental psychopathology (Meltzer, 2016). In adults, higher levels of RNT are associated with longer sleep onset latency and poorer sleep quality (Guastella & Moulds, 2007; Nota & Coles, 2015; Zoccola, Dickerson, & Lam, 2009). Further, an initial study in adults that incorporated both sleep duration and sleep timing components in one study found that sleep timing uniquely impacted RNT. Specifically, delayed sleep timing was associated with higher levels of RNT (Nota & Coles, 2015). There has been much less work investigating the relation between RNT and sleep timing in adolescents. A longitudinal study of adolescents found that later bedtimes, and not sleep duration, were associated with heightened emotional distress and worse academic performance (Asarnow, McGlinchey, & Harvey, 2014). Other preliminary studies in youth have reported associations with RNT and sleep disruption, specifically inadequate sleep duration, and negative psychological consequences such as depressed mood, poor emotion regulation, and increased anxiety (Baum et al., 2014; Short & Louca, 2015). However, these adolescent studies have been focused exclusively on sleep duration and did not address sleep timing (Baum et al., 2014; Roberts, Roberts, & Duong, 2009; Short & Louca, 2015; Talbot, McGlinchey, Kaplan, Dahl, & Harvey, 2010). Future research in youth would likely benefit from the examination of both sleep timing and sleep duration components.

Examining the link between RNT and disruptions in sleep duration and sleep timing in younger samples is particularly important because there are substantial changes in sleep during adolescence. Specifically, older adolescents demonstrate a large shift in their sleep patterns – delaying their bedtimes and wake times compared to adults and younger children (Carskadon, Acebo, & Jenni, 2004). Assessing a sleep timing component in addition to sleep duration may be especially relevant in research with adolescents because this is a time period of increased anxiety and mood disruptions (Compas, Orosan, & Grant, 1993). These anxiety and mood disruptions are often characterized by difficulty controlling one's thoughts. Adolescence is also a period of rapid structural and functional changes in the prefrontal cortex, which is important for cognitive control (Crews, He, & Hodge, 2007). Studies have demonstrated that disrupted sleep can hinder youth's cognitive processes, such as their ability to learn new information, memory, and executive functioning (Baum et al., 2014; Beebe, 2011; Kahn, Sheppes, & Sadeh, 2013; Sadeh, Gruber, & Raviv, 2003). Later chronotypes (i.e. preference for later bedtimes and later optimal functioning times) in adolescents have been associated with impairments in attention and memory (Goldstein, Hahn, Hasher, Wiprzycka, & Zelazo, 2007; Vollmer, Pötsch, & Randler, 2013) and in response inhibition (Kang et al., 2015; Telzer, Fuligni, Lieberman, & Galván, 2013). Attention and response inhibition problems are commonly associated with anxiety disorders, obsessive compulsive disorder (OCD), and other disorders that are characterized by RNT (Bannon, Gonsalvez, Croft, & Boyce, 2002; Shin & Liberzon, 2010). There is increasing recognition that the specific cognitions related to the breakdown in control processes found across disorders (e.g., worry, rumination, obsessions) have many shared features and can therefore be viewed collectively as forms of RNT (Ehring et al., 2011).

The aim of the current study was to examine if sleep timing and sleep duration as predictors of RNT during the significant developmental period of adolescence. The World Health Organization defines adolescence between the ages of 10 and 19 (Sacks, 2003). Given this definition, our sample of middle school and high school students, ages 11–17, were an ideal sample to test this link. Based on previous research on the negative consequence of disruptions in sleep timing and sleep duration, it was hypothesized that these variables would significantly predict levels of RNT, in that those with greater sleep disruptions (i.e. fewer hours of sleep, and later bedtimes) would experience higher levels of RNT. Further, as an exploratory aim, this study investigated the additional sleep variables of sleep onset latency, sleep quality, sleep efficiency, and daytime sleepiness as potential predictors of RNT.

## Method

### Participants

Participants included 1,021 adolescents attending public middle and high schools in upstate New York. Participants ranged from 11 to 17 years old ( $M = 14.12$  years,  $SD = 1.94$ ) and 51.8% were male. Data was collected in December of 2015. Based on self-report, this sample was 68.9% Caucasian, 8.9% African American, 2.7% Asian or Pacific Islander, 3.3% American Indian or Native Alaskan, 4.6% Hispanic, 10.1% multiple races/ethnicities, and 1.5% did not report their race/ethnicity. A letter was sent home to all students' parents/legal guardians, which provided an overview of the study and invited their child to participate. A copy of the survey was available for parents to view at the school if they were interested. At that time, parents were provided with the opportunity to decline their child's participation. After a teacher explained the study survey to students, the students were given the option to decline participation. Students who were absent on the day of the study were asked to complete the survey within one week from the original survey collection date.

### Procedure

The study procedures were approved by Binghamton University's Human Subjects Research Review Committee, the superintendent of the school district, and the school board. Assent was obtained by all students who participated in the study. The survey was conducted during regular school hours and completed in computer labs within the school. The survey was administered online through the Survey Monkey website.

### Measures

#### Perseverative Thoughts Questionnaire – Child Version (PTQ-C; Ehring et al., 2011)

The PTQ-C is used to assess RNT transdiagnostically in youth. It is a self-report measure containing 15 items. The questions on the PTQ-C specifically ask about the frequency, control, and distress associated with RNT on average. The measure was originally developed for adults and has since been revised to be developmentally appropriate for its administration with younger populations. There is emerging evidence that the PTQ-C has good psychometric properties, demonstrating excellent internal consistency and support for its validity (Bijttebier, Raes, Vasey, Bastin, & Ehring, 2015). In this sample, the PTQ-C items demonstrated very high internal consistency with a Cronbach's alpha of .99. The overall mean level of RNT measured by the PTQ-C in the current sample was 22.46 (19.56), ranging from scores of zero to 60. This mean is consistent with the findings of Bijttebier et al. (2015) who had a mean PTQ-C score of 27.98 ( $SD = 12.80$ ). Mean substitution was implemented for those cases who were missing less than 20% of items on the PTQ-C.

#### Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) and the School Sleep Habits Survey (SSHS; Shahid, Wilkinson, Marcu, & Shapiro, 2011)

Sleep was assessed using items from both the PSQI and the SSHS. Both measures contain self-report items that assess for sleep timing, sleep onset latency, and duration over the past month. Reports of sleep duration were utilized as a proxy for the homeostatic component of sleep and reports of bedtime were utilized as a proxy for the circadian component. The PSQI has adequate reliability and good validity in adult samples (Buysse et al., 1989) and has been used successfully in adolescents (Megdal & Schernhammer, 2007; Mesquita & Reimão, 2007; Tagaya et al., 2004). A potential limitation of the PSQI is that it can be more reflective of weekday nights than weekends. The SSHS has been validated in a large sample of adolescents (Shahid et al., 2011). Consistent with prior studies from our lab, we used the bedtime item from the PSQI (Coles, Schubert, & Sharkey, 2012; Nota & Coles, 2015; Schubert & Coles, 2013). Comparison of bedtimes reported on this item were consistent with published estimates from actigraphy data in adolescents (Crowley et al., 2014).

For the analyses of this study, the bedtime variable was taken from PSQI item one, asking generally what time the participants have gone to bed over the past month. To investigate the reliability of this item, a weighted bedtime was calculated using the school day and weekend bedtimes reported on the SSHS (i.e. ((school day bedtime\*5)+(weekend bedtime\*2)/7)). A paired samples *t*-test indicated that there were no significant

differences in reports for these variables ( $t(1007) = 1.00, p = .32$ ), meaning that the general bedtime report from PSQI item one was consistent with participants' reports of school day and weekend bedtimes. The hours slept variable was taken from PSQI item 4, which asks participants generally how many hours of sleep they have had over the past month. Again, a weighted variable was calculated to address the reliability of participants' reporting of this item (i.e.  $((\text{school day hours slept} * 5) + (\text{weekend hour slept} * 2) / 7)$ ). Hours slept for the weighted item was calculated using the bedtimes and wake times for schooldays and weekends reported on the SSSH.

### Strengths and Difficulties Questionnaire–Child Report (SDQ; Goodman, Meltzer, & Bailey, 1998)

The child-report SDQ is a youth (ages 4–17) 25-item self-report measure of psychological attributes including: emotional problems, conduct problems, hyperactivity, peer problems, and prosocial behavior. Scores of internalizing and externalizing problems can be generated in addition to a total score. The SDQ is widely used to assess youth mental health and has established retest reliability, internal consistency, and validity (Goodman, 2001). For the current study, the SDQ internalizing subscale was used as a measure of psychological symptoms. This subscale was used because RNT is primarily discussed in relation to disruptions in mood and anxiety (Goodman, 2001). In this sample, the SDQ internalizing subscale had adequate internal consistency with a Cronbach's alpha of .74. Mean substitution was implemented for those cases who were missing less than 20% of items on that subscale.

### Data analysis plan

Linear regressions were conducted to test the hypothesis that bedtime and hours slept would be predictive of RNT. Given the large sample size, the alpha level was set to .001. Several separate linear regressions were run. The first regression tested if demographic variables were predictors of RNT, the second regression tested if sleep variables were predictors of RNT, and the third regression tested if the sleep variable of bedtime remained a significant predictor of RNT after controlling for symptoms of psychopathology. Additional regressions were run to investigate whether discrepancies between weekends versus weekdays were contributors to this relation, and to investigate the possible contribution of sleep onset latency, sleep quality, daytime sleepiness, and sleep efficiency.

To address the possibility of multicollinearity of bedtime and hours slept items, a Pearson correlation was run. It revealed that the variables of bedtime and hours slept were not significantly correlated with each other ( $r = -.05, p = .12$ ). Correlation tables in the regression analyses were checked for sign flips and the variance inflation factors (VIFs) were checked to ensure that they were within an acceptable range (i.e. between .20 and 4.0; Hair, Anderson, Tatham, & Black, 1995).

Results Differences in sleep variables based on demographics were investigated. Independent *t*-tests revealed that males and females did not differ on their average self-reported bedtimes, hours slept, daytime sleepiness, sleep efficiency, or sleep quality. However, females had a significantly longer sleep onset latency than males ( $t(1003) = -2.14, p = .03$ ; males = 33.04 (37.86) min, females = 38.27 (39.80) min). Additionally, participants who identified as a racial minority had significantly longer sleep onset latency than participants who identified as Caucasian ( $t(1001) = -1.95, p = .05$ ; racial minority = 39.39 (42.48) min, Caucasian = 33.89 (37.16) min). Racial minorities and Caucasians did not differ on any other self-reported sleep variables. Further, there was a significant positive correlation between bedtime and age ( $r(1021) = .34, p < .001$ ) but not with any other sleep variables.

The demographic variables of sex, age, and race were entered into a linear regression analysis to test if they significantly predicted PTQ-C scores. The overall model was significant (adjusted  $R^2 = .024, F(3, 989) = 9.10, p < .001$ ). The results further indicated that sex was the only significant predictor of PTQ-C ( $\beta = .152, p < .001$ ). Specifically, females were more likely to have higher PTQ-C scores than males ( $t(1006) = -4.97, p < .001$ ; females = 25.52 (19.38), males = 19.47 (19.20)). The sleep indices of overall self-reported hours slept and bedtime were then added to the model in addition to the significant demographic variable of sex. The overall model was significant (adjusted  $R^2 = .055, F(3, 1007) = 20.41, p < .001$ ) and sex (i.e. females) remained a predictor ( $\beta = .153, p < .001$ ). For the sleep variables, self-reported bedtime was a significant predictor of PTQ-C (in that

**Table 1.** Primary regression models of later average bedtimes predicting higher PTQ-C scores.

Source	<i>B</i>	<i>SE B</i>	<i>B</i>	<i>t</i>	<i>p</i>
Model 1					
Sex	5.92	1.22	.15	4.84	.00
Age	.58	.32	.06	1.83	.07
Race	.35	.36	.03	.96	.33
Adjusted $R^2 = .02$ ( $p < .001$ )					
<sup>a</sup> Model 2.a.					
Sex	5.95	1.20	.15	4.98	.00
Bedtime	2.01	.34	.18	5.97	.00
Hours Slept	-.01	.26	-.01	-.04	.97
Adjusted $R^2 = .05$ ( $p < .001$ )					
<sup>a</sup> Model 2.b.					
Sex	5.95	1.20	.15	4.98	.00
Bedtime	2.01	.34	.18	5.98	.00
Adjusted $R^2 = .05$ ( $p < .001$ )					
Model 3					
Sex	2.04	1.19	0.05	1.95	.05
Bedtime	0.984	0.294	0.09	3.35	.00
SDQ Internalizing	2.45	0.129	0.52	18.99	.00
Adjusted $R^2 = .30$ ( $p < .001$ )					

<sup>a</sup>Models 2.a. and 2.b. were ran again using the average of all the bedtime sleep items as a composite score and the same pattern of significance occurred.

later bedtimes predicted greater PTQ-C scores;  $\beta = .183$ ,  $p < .001$ ), but self-reported hours slept was not significant ( $\beta = -.001$ ,  $p = .968$ ).<sup>1</sup>

Next, in order to control for symptoms of psychopathology, SDQ internalizing scores were added to the model along with sex and bedtime (see Table 1). Again, the overall model was significant (adjusted  $R^2 = .30$ ,  $F(3, 1007) = 147.98$ ,  $p < .0001$ ). SDQ internalizing scores were significant (in that greater SDQ internalizing scores predicted greater PTQ-C scores;  $\beta = .518$ ,  $p < .001$ ) and sex (i.e. females) was trending toward significance ( $\beta = .052$ ,  $p = .051$ ). Self-reported later bedtimes remained a significant predictor of greater PTQ-C scores ( $\beta = .089$ ,  $p = .001$ ) even after controlling for symptoms of psychopathology. A sex-bedtime interaction variable was added to this model and revealed that sex did not moderate the relation between later bedtimes and higher PTQ-C scores ( $\beta = .01$ ,  $p = .66$ ).

Further analyses were run investigating differences in weekday and weekend sleep patterns, given that bedtimes and hours slept can vary from weekends to weekdays for adolescents. In our sample the mean discrepancy score from weekend to weekday bedtimes and hours slept were 1.81 (1.73) hours and 1.10 (6.76) hours, respectively. Additional regressions were run investigating the difference score between weekend and weekday bedtimes and hours slept as possible predictors of PTQ-C, while continuing to control for internalizing symptoms. The regressions indicated that there was a trend for greater differences between weekday and weekend hours slept to predict higher PTQ-C scores ( $\beta = .05$ ,  $p = .07$ ), but not greater differences for weekday and weekend bedtimes ( $\beta = -.02$ ,  $p = .57$ ).

The sleep variables of sleep onset latency, sleep efficiency, and sleep quality as predictors of RNT were then investigated in further regression models, while controlling for SDQ internalizing scores. Neither sleep efficiency, nor sleep quality, nor daytime sleepiness were predictors of PTQ-C. However, the overall model with sleep onset latency was significant ( $F(2) = 219.89$ ,  $p < .001$ ). Longer sleep onset latency ( $\beta = .10$ ,  $p < .001$ ) was a significant predictor of higher PTQ-C scores.

The significant predictors of bedtime, sex, and sleep onset latency were then entered into a final linear regression model. The overall model was significant ( $F(3) = 29.64$ ,  $p < .001$ ). Bedtime ( $\beta = .13$ ,

<sup>1</sup>These analyses were ran again using the average of all the bedtime sleep items as a composite score and the same pattern of significance occurred.

$p < .001$ ), sex ( $\beta = .14, p < .001$ ), and sleep onset latency ( $\beta = .16, p < .001$ ) continued to remain unique predictors of RNT.

## Discussion

The aim of this study was to investigate sleep duration and sleep timing as predictors of RNT in an adolescent sample. Extending prior work, we found that self-reported later bedtimes were predictive of higher levels of RNT. Further, bedtimes continued to significantly predict levels of RNT even when controlling for internalizing symptoms. The overall number of hours slept was not found to significantly predict levels of RNT. However, when investigating the discrepancy between weekend and weekday sleep durations, there was a trend for greater discrepancies in sleep duration to predict higher levels of RNT. In other words, larger changes in the number of hours slept on weekend nights compared to weeknights were predictive of higher levels of RNT. Results from this study also indicated that longer sleep onset latencies were predictive of higher RNT. Further, a sex difference emerged in that females were more susceptible to higher levels of RNT.

These results suggest that it may be worthwhile to consider disruptions in both the amount and timing of sleep, and their variability from weekdays to weekends. Previous studies in adolescents ages 14 to 18 (Baum et al., 2014; Short & Louca, 2015) found a relation between overall hours slept and RNT, but the present study did not. This may be due to differences in study procedures, as those studies induced sleep deprivation whereas the current study was a naturalistic reporting of typical adolescent sleep habits. Furthermore, wake times, as determined by school start times, likely influenced the sleep duration that our participants achieved on average. This notion is consistent with our finding of a trend for greater differences in weekday versus weekend hours slept predicting higher RNT. Consequently, sleep duration and sleep timing may be more strongly related due to these stricter wake times that are dictated by school start times. The finding of overall later bedtimes being associated with higher RNT is a replication of the effect found by Nota and Coles (Nota & Coles, 2015) in an adult sample. These results are also consistent with the findings of Asarnow et al. (2014), who found later bedtimes, and not sleep duration, in adolescents to be associated with greater emotional distress. The results of the current study suggest that timing of sleep is its own contributor to the increase in RNT in adolescents. This finding is further strengthened by the additional study result of longer sleep onset latency predicting higher levels of RNT. It is possible that the timing of sleep may uniquely impact cognitive control processes responsible for the regulation of RNT, as sleep disruptions have been associated with cognitive impairments (Wulff et al., 2010). Specifically, studies have demonstrated that disruptions in circadian rhythms, a larger process connected to sleep timing, are linked to deficits in memory, learning, and response inhibition (Goldstein et al., 2007; Kang et al., 2015; Telzer et al., 2013; Vollmer et al., 2013). These cognitive deficits have also been linked to psychiatric conditions characterized by RNT, such as, depression, anxiety disorders, and OCD (Bannon et al., 2002; Shin & Liberzon, 2010). Consistent with our study findings, females are at a particular risk during adolescence to develop disorders characterized by RNT (Natsuaki et al., 2009). It is possible that disrupted sleep, such as a longer sleep onset latency, later overall bedtimes, and greater weekend versus weekday discrepancies, may increase adolescent females' risk of developing such disorders.

At present there is much emphasis on total sleep duration, but this emerging research suggests that bedtime and sleep variability are also important factors. There is a natural shift to later bedtimes during adolescence (Carskadon et al., 2004). It is unclear whether this natural bedtime shift is a contributing factor to increased levels of RNT. Sleep timing may serve as an important area of intervention and prevention of excessive RNT. For example, providers may want to be aware of their clients' sleep habits as targeting RNT through cognitive therapy techniques alone may be particularly challenging given that sleep difficulties are associated with cognitive impairments (Wulff et al., 2010). It may be beneficial for clinicians to help foster environments and habits that promote earlier bedtimes and consistency in total sleep durations to alleviate symptoms of RNT. Since adolescence is



associated with a natural shift to later bedtimes, clinicians can offer advice, such as reducing electronic usage at night, so as not to further exacerbate the effect of late bedtimes.

The current study would have benefited from multiple measures of sleep timing and sleep duration. Additionally, it could have benefited from the use of validated measures to assess factors, such as, chronotype, sleep hygiene, and other sleep problems, as these factors many have a direct impact on sleep timing and sleep duration. The cross-sectional nature of this study is a further limitation. To truly capture the causality of the relation between sleep timing and RNT, future studies would benefit from longitudinal data collection methods. This study utilized a primarily Caucasian and younger sample, so further research is warranted in samples reflecting greater diversity of age and race.

Future research can also extend the findings of this initial study through the use of additional methods such as sleep diaries and saliva collection to measure dim-light melatonin onset as a more precise estimate of circadian rhythms. Additional research should look deeper at the influence of sleep timing and sleep duration, including sleep quality and sleep onset latency. Other possible domains of impact – such as emotional, cognitive, and somatic functioning – should also be examined.

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## Disclosure statement

No potential conflict of interest was reported by the authors.

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