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BRIEF REPORT

Duration and Timing of Sleep are Associated with Repetitive Negative Thinking

Jacob A. Nota · Meredith E. Coles

Abstract Higher levels of repetitive negative thinking (RNT; a perseverative and abstract focus on negative aspects of one's experience) are associated with reduced sleep duration. This information is already informing theory and clinical practice. However, we are not aware of any studies examining the relation between RNT and the timing of sleep. We examined both disorder specific measures of RNT and a transdiagnostic measure of the RNT process in relation to sleep duration and timing in a sample of 100 unselected undergraduates. Replicating prior findings, shorter sleep duration was cross-sectionally associated with more rumination and delayed sleep timing was associated with more obsessive–compulsive symptoms. Further, extending this prior work, the transdiagnostic measure of RNT was associated with shorter sleep duration *and* delayed sleep timing. Individuals who endorsed a preference for later sleep and activity times also reported more RNT. These findings suggest that RNT may be uniquely related to both sleep duration and timing.

Keywords Repetitive negative thinking · Worry · Rumination · OCD symptoms · Sleep · Bedtimes

Introduction

Repetitive Negative Thinking (RNT) is defined as an abstract, perseverative, negative focus on one's problems and experiences that is difficult to control (Ehring and Watkins 2008; Harvey et al. 2004). This process is associated with several disorders, including generalized anxiety disorder (GAD), major depressive disorder (MDD), post-traumatic stress disorder (PTSD), obsessive–compulsive disorder (OCD), and social anxiety disorder (for review see Ehring and Watkins 2008; Watkins 2008). Across these disorders, there are topographical differences in RNT content (e.g., worry about the future in GAD, rumination about the past in MDD, compulsive repetitive thoughts in OCD) but a core construct also seems to be shared (Ehring et al. 2011; McEvoy et al. 2010). Disorder specific forms of RNT (e.g., worry and rumination) are associated with greater distress (Demeyer et al. 2012; McEvoy and Brans 2013; Starr and Davila 2012), future depressive symptoms (Abela et al. 2002; Nolen-Hoeksema 1991), and increased anxious symptoms (Blagden and Craske 1996; Calmes and Roberts 2007; Hong 2007; McEvoy and Brans 2013; Wahl et al. 2011) in clinical and non-clinical samples. Further, inducing RNT in non-clinical samples has been shown to prolong negative affect and negatively impact cognitive, behavioral, and interpersonal performance (for review see Watkins 2008).

The literature supporting RNT as a transdiagnostic factor in psychopathology suggests that understanding how RNT itself comes about and is maintained may be of theoretical and clinical interest. There are a number of lines of research examining this question. Psychosocial (e.g., overcontrolling parenting, modeling of emotional expression and rumination; Cox et al. 2010; Gibb et al. 2012; Hilt et al. 2012) and genetic (Clasen et al. 2011; Gibb et al. 2012; Hilt et al. 2007) research has identified some putative risk factors for RNT, however further research is needed (Moore et al. 2013). Many researchers hypothesize that RNT may contribute to the maintenance of symptoms by serving as a form of cognitive avoidance (Borkovec 1994; Ehring and Watkins 2008; Giorgio et al. 2010;

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Lyubomirsky and Nolen-Hoeksema 1995). Cognitive avoidance strategies are thought to have a paradoxical effect: despite being aimed at reducing distressing thoughts, the importance assigned to suppressing thoughts can lead to increased attention towards these thoughts and thereby unintentionally increases the likelihood of their reoccurrence while reducing experience in effectively handling these thoughts (Freeston et al. 1995; Freeston and Ladouceur 1997). Working memory, attention and inhibition appear to be involved in the control of RNT as behavioral measures of these are negatively impacted by manipulations of RNT (Hallion et al. 2014; Hayes et al. 2008; Joormann and Gotlib 2008; Krebs et al. 2010; Wells 1994). For example, Hallion and colleagues found that asking individuals to worry about a personally relevant thought and then attempt to control their worry resulted in impairments for working memory accuracy and inhibition reaction times compared to doing the same with a neutral thought (Hallion et al. 2014). Moreover, psychophysiological measures related to the control of attention and working memory [e.g., activity in the default mode network, alpha wave EEG band and dorsolateral prefrontal cortex (DLPFC)] are heightened during conditions of both activity and rest in individuals with heightened levels of worry and rumination compared to healthy controls (Hamilton et al. 2013; Borkovec et al. 1998).

Individuals with a number of the disorders characterized by RNT are likely to report sleep disturbances. For example, difficulty falling and staying asleep are diagnostic criteria for several internalizing disorders (American Psychiatric Association 2013). In addition, a number of studies document a relationship between RNT and reductions in sleep duration or quality (Fairholme et al. 2013; Guastella and Moulds 2007; Harvey 2002; Thomsen et al. 2003; Zoccola et al. 2009). For example, sleep deprivation has been linked to increased rumination and decreased mood (Baglioni et al. 2010; Pilcher and Huffcutt 1996; Walker 2009). However, there is still more to be learned about the relation between RNT and sleep duration. Experimental studies that induce rumination or worry prior to sleep have been shown to reduce sleep quality (Gross and Borkovec 1982; Vandekerckhove et al. 2012) and naturally occurring levels of RNT have been shown to prospectively predict sleep quality (Takano et al. 2012). Indeed, baseline reports of both rumination and worry interactively predicted reductions in self-reported sleep quality 3 weeks later in an unselected sample (Takano et al. 2012). Thus, the relation between RNT and sleep disturbance may be bidirectional in nature. Further, there is a known relation between the negative valence of RNT content and sleep restriction (Pilcher and Huffcutt 1996).

While the relation between RNT and sleep duration is gaining empirical support there is limited information on RNT's relation with the timing of sleep. Incorporating the timing of sleep is important given that it represents another primary component of sleep regulation (for review see Borbely and Achermann 2005). Alterations in sleep timing and circadian rhythms could be independently related to RNT (Wulff et al. 2010). Indeed, many disorders characterized by RNT tend to onset during young adulthood (Kessler et al. 2007; Kessler et al. 2005), a period also associated with delays in sleep timing (Carskadon 2011; Pelayo et al. 1988). Recent studies have documented heightened rates of OCD, depression, and generalized anxiety disorder in individuals with delayed bedtimes (Abe et al. 2011; Reid et al. 2012; Schubert and Coles 2013). In addition, eveningness (Horne and Ostberg 1976), a trait preference for a sleep/wake schedule that is skewed later in the day, has also been found to be related to measures of mental health (Chelminski et al. 1999; Hidalgo et al. 2009; Randler 2011). Interestingly, there is evidence that clinically addressing delayed sleep timing may improve comorbid psychopathology symptoms (Coles and Sharkey 2011). Despite this, we are not aware of any investigations of the relation between sleep timing and levels of RNT.

Similarities in the neurocognitive functions involved in RNT and those affected by sleep disturbance further suggest the benefits of continuing this line of work. Experimental reductions of sleep duration produce impairments in executive functioning (Harrison and Horne 1999, 2000; Nilsson et al. 2005) as does misalignment of the sleep-wake cycle with the light–dark cycle (Kohyama 2011; Wright et al. 2006). Further, evidence suggests that prefrontal cortical areas of the brain (recruited in the allocation of attention, working memory, and inhibition) are particularly sensitive to sleep deprivation and circadian rhythm disruption (Borbely and Achermann 2005; Cajochen et al. 2004; Muzur et al. 2002). Given this, it is plausible that sleep disturbances could maintain heightened levels of RNT by impeding the ability to control RNT vis-à-vis these neuropsychological pathways.

This study was designed to replicate and extend previous research linking RNT to reduced sleep duration. First, consistent with previous research, it was hypothesized that measures of RNT framed within disorder specific content (i.e., worry about the future, rumination about the past, repetitive obsessive thoughts) would be negatively related to sleep duration. Extending from previous research, we hypothesized that a transdiagnostic measure of the RNT process (i.e., perseverative abstract thinking) free from disorder specific content would also be negatively related to sleep duration. This would be consistent with the hypothesis that sleep duration affects an individual's ability to control RNT and is not simply negatively affecting mood. We further hypothesized that both disorder specific and transdiagnostic measures of RNT would be positively related to sleep timing (i.e.

later bed times would be associated with higher levels of RNT). Indeed, we expected that sleep duration and sleep timing would each independently predict RNT when controlling for variance shared between them. Given the documented relations between negative affect and sleep disruptions and negative affect and RNT (Baglioni et al. 2010; Pilcher and Huffcutt 1996; Walker 2009) we hypothesized that the relations between sleep and RNT would be partially mediated by negative affect. However, given the literature linking disruptions of sleep and deficits in working memory, inhibition, and attention, we posited that these relations would not be totally accounted for by covariation with negative affect. Finally, to test the convergent validity of our assessment of sleep timing we included a measure of circadian preference (i.e., morningness-eveningness). Based on research relating an evening-type circadian preference to greater sleep disruption (Kang et al. 2012; Tzischinsky and Shochat 2009) and poorer mental health (Chelminski et al. 1999; Hidalgo et al. 2009; Randler 2011), we hypothesized that individuals who were evening-types (i.e., those who's preference is to go to bed and wake later) would report higher levels of RNT compared to those who are morning-types.

Methods

Participants and Procedures

One hundred young adults were recruited from the research participation pool at a large public university. The sample was approximately half females (58 %) with an average age of 19.4 years (SD = 1.9, range 17–33). Participants were predominantly White (56 %), with smaller proportions of Asian (26 %), Latino (8 %), biracial (4 %), and Black (3 %) individuals. The population from which we drew this sample was well suited to the study aims, as emerging adults are more likely to provide adequate variability necessary to detect relations in both sleep and psychopathology variables compared to other age groups (Carskadon 2011; Carskadon and Acebo 2002). It was deemed most prudent to employ a cross-sectional design in the current study given the novel nature of our hypotheses regarding the relation between sleep timing and RNT. This would minimize participant burden, and be a more time and cost-effective manner of determining whether further study was warranted. Participants were given an overview of the study and provided written informed consent. Participants then completed a battery of self-report questionnaires (as outlined below) and two computerized tasks (not reported herein).

Measures

Consistent with previous studies, we included three diagnosis specific measures of symptoms related to RNT (i.e., worry, rumination, and obsessions). These measures were included to document their relations with the higher order construct of RNT and to assess their independent relations with sleep. Worry was assessed using the *Worry Domain Questionnaire* (WDQ; Tallis et al. 1992). Twenty-five items assess five common domains of worry (relationships, lack of confidence, aimless future, work, and financial). The WDQ is highly correlated with other measures of worry (Davey 1993) and has displayed good retest reliability ($r = .85$; Stöber 1998). In the current sample the WDQ displayed strong internal consistency ($\alpha = .95$). Depressive rumination was assessed by the *Ruminative Response Scale of the Response Style Questionnaire* (RRS; Nolen-Hoeksema and Morrow 1991). Twenty-two items assess responses to depressed mood that focus on the self, symptoms, and the causes and consequences of depressed mood. Only the brooding subscale (5 item) was examined herein (Treynor et al. 2003) because this scale has been found to be more strongly associated with depression (Armey et al. 2009; Gibb et al. 2012; Treynor et al. 2003). In previous studies, the RRS-brooding subscale has displayed sufficient retest reliability over two months ($r = 0.71$) and good concurrent validity with related scales assessing depressive symptoms (Sakamoto et al. 2001). In the current sample it displayed adequate internal consistency ($\alpha = .85$). OCD symptoms were assessed with the *Obsessive–Compulsive Inventory* (OCI; Foa et al. 1998). Forty-two items measure severity across symptom domains (washing, checking, doubting, ordering, obsessing, hoarding, and neutralizing) during the past month. OCI scores are strongly correlated with other measures of OCD symptoms in clinical and control samples (r's = .65 to .81), are less correlated with symptoms of depression than the more brief OCI-revised (Foa et al. 2002), and the scale has previously demonstrated strong retest reliability ($r = .84$; Foa et al. 1998). In the current sample, the OCI displayed strong internal consistency ($\alpha = .95$). Although the OCI measures both frequency and distress associated with OCD symptoms, our analyses focused on the distress data.[1]

We also included a measure of the transdiagnostic process thought to underlie RNT; the *Perseverative Thinking Questionnaire* (PTQ; Ehring et al. 2011). The PTQ was designed to capture RNT independent of specific thought content that might vary depending on diagnosis

[1] Though we focused on Obsessive–Compulsive Inventory distress scores, the findings were consistent when utilizing Obsessive–Compulsive Inventory frequency scores.

(e.g., thoughts about the past in individuals with depression and worries about the future in individuals with anxiety disorders). The PTQ consists of 15 items assessing the repetitiveness, intrusiveness, difficulties disengaging, and unproductiveness of RNT as well as the degree to which this response to intrusive thoughts captures mental capacity. Higher scores indicate greater levels of RNT. Past research has demonstrated good psychometric properties for this measure (Ehring et al. 2011). In the current sample, the PTQ displayed strong internal consistency ($\alpha = .96$).

Negative affect (NA) was assessed by the *Positive and Negative Affective Schedule Negative Affect* scale (PANAS-NA; Watson et al. 1988). NA is a general dimension of subjective distress that is related to a variety of aversive mood states, including guilt and sadness. In the PANAS-NA, ten items assess trait NA. Given the high overlap between intrusive thought disorders and mood disruption it would be unrealistic to expect complete independence from negative affect (Clark 2002; Clark and Watson 1991). This subscale was included to test the hypothesis that the relations between sleep disruption and RNT would be partially mediated by NA. This will serve as a test of robustness of the observed relation between sleep disruption and RNT. In the current sample, the PANAS-NA displayed adequate internal consistency ($\alpha = .81$).

Four items from the *Pittsburgh Sleep Quality Index* (PSQI; Buysse et al. 1989) were used to evaluate sleep duration (item 4), onset latency (i.e., how long it took to fall asleep; item 2)] and bed and wake timing (items 1 and 3, respectively) over the past month. Sleep duration and timing are variables of interest for our primary aims in this study, while sleep onset latency was included to describe our sample's sleep behavior. Studies have demonstrated that data gathered from the PSQI produce similar results to those obtained when using longitudinal sleep diaries (Grandner et al. 2006). In the current sample there were small intercorrelations between sleep onset latency and bedtime ($r = .07$, $p = .48$), sleep duration and bedtime ($r = -.11$, $p = .28$), and slightly larger correlations of sleep onset latency and sleep duration ($r = -.26$, $p = .01$) supporting the separability of these constructs.

Morningness-eveningness was determined by means of the *Horne Ostberg Morningness-Eveningness Questionnaire* (MEQ; Horne and Ostberg 1976). Nineteen forced choice and time scale items assessed personal preferences over the past several weeks and item scores were converted to a five-point morningness-eveningness scale (i.e., definitely evening type, moderately evening type, neither type, moderately morning type, and definitely morning type). We created two groups to compare habitual morning and evening types. These groups were designed to reflect the morning and evening type distinction and to accommodate the distribution of morning and evening types in our sample (i.e., skew toward evening types). Our groups consisted of (a) those who were moderately morning types or neither type ($n = 34$) and (b) those who were moderately evening type or definitely evening type ($n = 41$).

This study represents a portion of a larger project. As a result, all study measures were not available for every participant. Specifically, the WDQ, RRS-brooding, and the MEQ were not available for 25 participants. However, since these measures were not required for the primary aims of the study, we include them herein as a means of obtaining supplementary information on the relation between RNT and sleep. Further, independent samples t tests demonstrated that the 25 subjects without these measures did not differ from those before them on the PTQ ($t(97) = -.48$, $p = .64$), OCI ($t(94) = .75$, $p = .46$), PANAS-NA ($t(96) = -.09$, $p = .93$), sleep duration ($t(95) = -.30$, $p = .77$), sleep onset latency ($t(95) = .27$, $p = .79$), or bedtime ($t(96) = -.67$, $p = .51$).

Statistical Analysis

Pearson correlations and multiple linear regressions were utilized to investigate the relations between measures of sleep and RNT. The SPSS INDIRECT macro (Preacher and Hayes 2008) was used to test whether NA partially mediated the relations between sleep duration and sleep timing in predicting levels of transdiagnostic RNT. The INDIRECT macro uses a regression-based model to produce coefficients for both direct and indirect effects for relations between continuous variables and a bias-corrected 95 % bootstrap confidence interval for the indirect effect using bootstrap samples (1,000 bootstrap samples were used herein). One participant was removed from all analyses as they were an outlier (>3 SD above overall mean) on the OCI and PANAS-NA. Based on their MEQ, this individual fell in the morning or neither type, making the final n = 33 for this group during analyses. Regarding normality, the OCI-D, PANAS-NA, and sleep onset latency scores were positively skewed and therefore square root transformed. Findings were effectively identical using both transformed and untransformed values. Therefore, untransformed values are reported herein for the sake of interpretability. Finally, a two-tailed significance level of $\alpha = .05$ was used for all analyses. Missing values were excluded pairwise; therefore degrees of freedom in inferential tests vary.

Results

Sample Characteristics

Descriptive statistics for the study measures are presented in Table 1. Reports on the items assessing sleep, diagnosis

Table 1 Descriptive statistics

	Mean (SD)	Min–Max
RNT/affect		
PTQ	20.97 (12.73)	0–53
RRS-brooding[a]	9.95 (3.76)	5–20
WDQ[a]	34.27 (20.64)	1–80
OCI	20.65 (18.00)	0–97
PANAS-NA	18.84 (5.59)	10–38
Sleep		
Bedtime	1:01 AM (1 h:13 m)	10 PM–5 AM
Onset latency	21 m:39 s (15 m:21 s)	0 m–90 m
Duration	6 h:59 m (1 h:15 m)	4 h–11 h
Mid sleep	4:54 AM (1 h:22 m)	11:37 PM–8AM
MEQ[a]	Median = moderately evening	Moderately morning–definitely evening

RRS Ruminative Response Scale, *WDQ* Worry Domain Questionnaire, *OCI* Obsessive–Compulsive Inventory, *PTQ* Perseverative Thought Questionnaire, *PANAS* Positive and Negative Affective Scale, *NA* negative affect, *PA* positive affect, *Mid sleep* middle point between bedtime and wake time, *MEQ* Morningness-Eveningness Questionnaire

[a] n = 74

Table 2 Correlations between sleep measures, RNT, and Positive and Negative Affect and mean scores by morningness-eveningnessRNT and Sleep

	PTQ	RRS-brooding[a]	WDQ[a]	OCI	PANAS-NA
Bed time	.28*	.17	.20[T]	.26*	.24*
Sleep onset latency	.03	−.05	.15	.07	.10
Sleep duration	−.24*	−.31*	−.22[T]	−.16	−.15
MEQ[a]					
Morning	17.00 (11.26)	8.85 (3.60)	15.97 (14.60)	29.30 (20.97)	17.38 (4.86)
Evening	24.80 (13.78)	10.83 (3.69)	23.00 (18.36)	38.27 (19.72)	20.07 (5.95)

* $p < .05$. [T] $p \leq .10$; *PTQ* Perseverative Thought Questionnaire, *RRS* Ruminative Response Scale, *WDQ* Worry Domain Questionnaire, *OCI* Obsessive–Compulsive Inventory, *PANAS-NA* Positive and Negative Affective Scale negative affect

[a] n = 74

specific forms of RNT, and transdiagnostic RNT were similar to those from previous young adult samples (Buboltz et al. 2001; Ehring et al. 2012; Foa et al. 1998; Lund et al. 2010) and demonstrate sufficient variability for further examination.

RNT and Sleep

We first examined the relations between the measures of sleep and disorder specific forms of RNT (rumination, worry, and OC symptoms). Zero-order correlations revealed a significant *negative* relation between sleep duration and rumination and a significant *positive* relation between bedtime and OC symptoms (see Table 2). We then examined the zero-order correlations of transdiagnostic RNT and the measures of sleep. Findings showed that transdiagnostic RNT was significantly *negatively* correlated with sleep duration and transdiagnostic RNT was significantly *positively* correlated with bedtimes (see Table 2). In other words, sleeping for shorter periods of time and going to bed later were associated with experiencing more RNT overall.

Next, a multiple linear regression was carried out to assess the independence of the indices of sleep as predictors of transdiagnostic RNT. We entered sleep duration and bedtimes as step-wise predictors of RNT.[2] Results indicated that when levels of RNT were simultaneously predicted by sleep duration and bedtimes ($F(2,94) = 6.93$, $p < .01$), both sleep duration ($t(1,94) = -2.21$, $\beta = -.21$, $p = .03$) and bedtimes ($t(1,94) = 2.72$, $\beta = .27$, $p < .01$) contributed independently to the prediction of transdiagnostic RNT.

Next, we used INDIRECT to test two mediation models, examining both the direct effects of sleep variables on transdiagnostic RNT, and the indirect effects when NA is inserted between the sleep variables and RNT. First, including NA, we did not find a significant indirect effect of sleep duration on RNT through NA ($\beta = -0.85$, 95 % CI [−2.04, 0.31], $\kappa^2 = .09$). Further, after accounting for the indirect effect through NA, there was only a marginally significant direct effect of sleep duration on RNT ($t(95) = -1.85$, $p = .07$, $r_{es} = .19$). In the second model, sleep duration was replaced with bedtime. In contrast to the first model, there was a significant positive indirect effect of bedtime on RNT through NA ($\beta = 1.36$, 95 % CI [0.30, 2.54], $\kappa^2 = .14$). However, mimicking the first model, there was only a marginally significant direct effect of bedtime on RNT after accounting for the indirect effect ($t(96) = 1.91$, $p = .06$, $r_{es} = .19$).

Circadian Preference and RNT

Finally, we conducted an independent samples *t* test to address our hypothesis that levels of transdiagnostic RNT would be higher in those who were characterized as

[2] We did not include sleep onset latency in the regression given the lack of a significant zero-order correlation between this index and RNT.

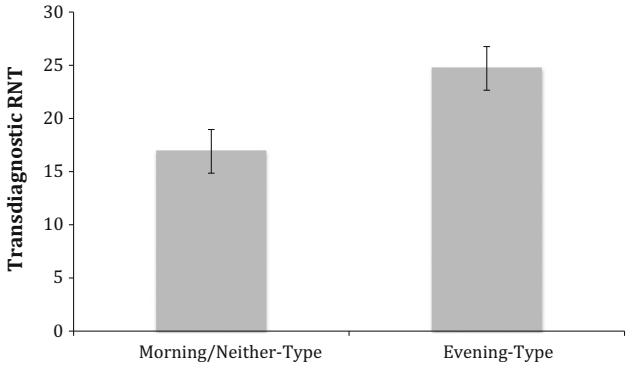

Fig. 1 Comparison of morning/neither and evening types on levels of repetitive negative thought. RNT was measured by the Perseverative Thought Questionnaire. *Error bars* represent standard error. $N = 74$

evening types compared to those who were morning type or neither morning nor evening type. As predicted, those individuals classified as moderately or definitely evening type ($M = 24.80$, $SD = 13.78$) reported significantly higher levels of transdiagnostic RNT than those who were moderately morning-types or neither type ($M = 17.00$, $SD = 11.26$; $t(72) = -2.62$, $p = .01$; See Fig. 1).

Discussion

The current study replicated and extended previous work by examining relations between RNT and sleep duration using disorder specific and a transdiagnostic measure of the RNT process and then also examining relations between RNT and sleep timing. When we differentiated sleep duration and sleep timing, we found evidence of unique relations between both these constructs and transdiagnostic RNT thereby supporting our proposal that distinguishing between these two sleep measures is useful for understanding RNT. Further, our findings that the timing of sleep and eveningness, a characteristic of individuals associated with their circadian rhythm biology, are associated with heightened levels of transdiagnostic RNT are notable as we are not aware of any previous studies demonstrating such relations. The relation between these measures of sleep and a transdiagnostic measure of RNT is also notable as these findings suggest that sleep disruption may be related to the process of RNT, regardless of the content of intrusive repetitive thoughts.

More evidence linking sleep duration and/or timing to the experience of repetitive unwanted thoughts holds promise for creating more integrated models of psychopathology and informing interventions. For example, it may be the case that deficits in working memory, attention, and inhibition related to disruptions in homeostatic (sleep duration; Harrison and Horne 1999, 2000; Nilsson et al. 2005) and circadian (sleep timing; Kohyama 2011; Wright et al. 2006) sleep processes may increase the probability of RNT occurring and decrease the ability of individuals to inhibit RNT once it does occur. Therefore, individuals who are at risk for developing a disorder characterized by intrusive thoughts may benefit from focusing on obtaining adequate sleep. Ultimately, individuals that are chronically sleep deprived and experience repetitive intrusive thoughts may have the option of using psychotropic medication, engaging in cognitive or cognitive-behavioral therapy, *or* focusing on increasing their sleep duration. Similarly, pending replication, making sure that sleep is obtained during the right time of day may be an inexpensive and easily disseminable intervention for individuals bothered by intrusive thoughts. Such interventions would build from evidence that individuals with OCD, depression, and anxiety disorders are at heightened odds for circadian disruptions characterized by an inability to go to sleep until late (delayed sleep phase disorder; DSPD; Mukhopadhyay et al. 2008; Turner et al. 2007; Abe et al. 2011; Reid et al. 2012; Schubert and Coles 2013).

The study findings need to be considered in the context of a number of limitations. First, the unselected sample precludes generalization to clinical samples. Second, the correlational and cross-sectional design of the current study did not allow for the determination of directionality in either the relations between RNT and sleep disruptions. This is an important question to address in future studies, as evidence thus far is equivocal for both directions of causality. Prospective studies examining the temporal relations between shifts in bed times and increases in RNT are needed to address questions regarding directions of effects unanswered herein. Further, the relations between RNT and bedtimes and RNT and sleep duration did not remain statistically significant when controlling for variance shared with negative affect. However, the magnitudes of the direct paths from sleep variables to RNT were only slightly reduced compared to the zero order correlations. Indeed, the direct effects of both sleep duration and timing to RNT were small (Cohen 1992), but notable ($r_{es} = .19$). Therefore the failure to detect these effects may have been an issue of power rather than a limitation of our working hypotheses regarding the relation between sleep disruption and RNT. In addition, given the high overlap between intrusive thought disorders and mood disruption it may be unrealistic to expect complete independence from negative affect (Clark 2002; Clark and Watson 1991). In the current study, the indirect effects of sleep duration and timing on RNT through NA were also relatively small (Hayes 2013), but notable, particularly for sleep timing ($\kappa^2 = .09$ and .13, respectively). Future studies should attempt to clarify the

complex relation between these constructs. Finally, in future studies, it may also be beneficial to document the rates of clinically significant shifts in sleep timing, DSPD diagnoses, and other sleep disruptions via clinician-administered interviews.

If further findings support the relation between sleep timing and RNT this could one day lead to a new avenue for treatment of individuals with internalizing disorders. Already, studying the relation between reductions in sleep duration and psychopathology has demonstrated that focusing on sleep in the clinic also lead to reductions in symptoms of psychopathology (Belleville et al. 2011; Manber et al. 2008). Well-developed, inexpensive, safe, and effective treatments aimed at addressing sleep timing disruptions (e.g. bright light chronotherapy; Wirz-Justice et al. 2009) could also potentially be employed to reduce RNT and increase individual's ability to control RNT across psychiatric diagnoses. As the direction of this relation is not yet known, it may also be possible that employing treatments aimed at reducing worry, rumination, and other forms of RNT will improve the timing and duration of sleep; an outcome that is rarely noted in the treatment literature but is likely to have an impact on daily functioning (Curcio et al. 2006; Meijer et al. 2000; Wittmann et al. 2006). The current study suggests that further examination of RNT and its relation to sleep duration, *and* sleep timing, may be fruitful.

Conflict of Interest Jacob A. Nota and Meredith E. Coles declares that they have no conflict of interest.

Informed Consent All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2000. Informed consent was obtained from all patients for being included in the study.

Animal Rights No animal studies were carried out by the authors for this article.

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