



# Executive Dysfunction and Emotion Dysregulation Explain the Effects of Insomnia Symptoms on Repetitive Negative Thinking

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## Abstract

Although research has linked insomnia symptoms to repetitive negative thinking (RNT), few studies have examined how insomnia symptoms are associated with RNT over time or specific factors that may account for this relationship. The present study addressed this gap in the literature by examining executive function and emotion regulation as mediators of the relationship between insomnia symptoms and RNT over 3 months. A final sample of 357 unselected community participants completed measures of insomnia symptoms and RNT at time 1, executive function 1 month later (time 2), emotion regulation 2 months later (time 3), and RNT again 3 months later (time 4). Results revealed that insomnia symptoms were associated with increased RNT over 3 months through an indirect effect of executive function on emotion regulation. An alternate model in which emotion regulation at time 2 and executive function at time 3 mediated the effect of insomnia symptoms on RNT was also significant; however, the effect size was relatively reduced. These findings implicate executive function and emotion regulation as factors that may explain the role of insomnia symptoms in the development of RNT observed in many psychiatric disorders.

**Keywords** Insomnia · Sleep · Repetitive thinking · Executive function · Emotion regulation

Accumulating evidence links insomnia symptoms to repetitive negative thinking (RNT), a transdiagnostic process involving thoughts that are recurrent and excessive, include negative content, and are perceived as difficult to control (Ehring and Watkins 2008). Indeed, recent findings indicate that insomnia symptoms are associated with RNT cross-sectionally (Baker et al. 2015) and longitudinally (Cox et al. 2018). Poor sleep is also linked to specific forms of RNT. For example, rumination, or RNT related to perceived threats to the self and/or negative past experiences (Trapnell and Campbell 1999), is associated with insomnia symptoms in both unselected (Cox et al. 2016) and sleep-disordered samples (Carney et al. 2010). Likewise, sleep disturbance predicts increased worry, or RNT about negative future

events (Borkovec et al. 1998), in both unselected adolescents (Danielsson et al. 2013) and adults with generalized anxiety disorder (GAD; Thielsch et al. 2015). Finally, obsessions, or repetitive intrusive, distressing thoughts (Julien et al. 2007), are uniquely linked with insomnia symptoms, controlling for depressive symptoms (Timpano et al. 2014).

Though extant research links insomnia symptoms to RNT, the majority of these studies have relied on cross-sectional methods, which precludes examining change in RNT over time. Further, limited research has examined mechanisms of this relationship. One potential mechanism is impaired executive function, which consists of higher-order cognitive control processes that regulate cognition and includes flexible shifting, updating information, and inhibition (Miyake et al. 2000). Considerable evidence indicates that sleep loss and impaired sleep continuity negatively impact executive function (Wilckens et al. 2014), including deficits in inhibition (Bocca et al. 2014) and switching (Plessow et al. 2011). Likewise, compared to healthy controls, individuals with insomnia demonstrate difficulties with switching, and individuals with insomnia and short sleep duration demonstrate additional difficulties with working memory (Khasawneh et al. 2018).

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Deficits in executive function are also linked to disorders characterized by RNT, including obsessive–compulsive disorder (OCD; Chamberlain et al. 2005) and posttraumatic stress disorder (PTSD; Aupperle et al. 2012). Further, decreased executive function may contribute to RNT, as one study found that deficits in inhibition and switching are linked to rumination (Whitmer and Banich 2007). Importantly, previous research examining the reverse relationship (i.e., the impact of RNT on executive function) indicates that induced RNT decreases executive function only among disordered populations, while no such effect is found in healthy individuals (Philippot and Brutoux 2008; Watkins and Brown 2002). Thus, the predictors of RNT in the general population remain unclear. Extant findings from the sleep literature suggest that the downstream consequences of insomnia symptoms on executive function may play a role in the development of RNT. This view is consistent with recent findings linking decreased sleep duration to decreased executive function among those high in RNT (Nota and Coles 2018) and showing that the prospective relationship between insomnia symptoms and RNT is mediated by decreased attentional control (Cox et al. 2018). Thus, insomnia symptoms may impair executive function, which may then result in increased RNT. However, few studies have examined these relationships over time, limiting the ability to determine how these processes impact change in RNT.

It is also unclear whether affective processes intervene in the relationship between executive function and RNT. That is, does decreased executive function as a result of insomnia symptoms directly impact RNT, or does this effect occur through the influence of executive function on emotional processes? One candidate affective mediator is emotion regulation, or monitoring and modulating affective reactions to promote adaptive responding (Bjureberg et al. 2016). Poor emotion regulation is a transdiagnostic process (Fernandez et al. 2016) that may increase RNT by promoting maladaptive responses (i.e., worry, rumination) to stressful events. Extant research also links decreased executive function to poor emotion regulation and increased RNT (Joormann and Gotlib 2010). Further, poor sleep quality is associated with decreased emotion regulation ability (Mauss et al. 2013; but see also Reddy et al. 2017), and emotion dysregulation mediates the relationship between insomnia and negative reinforcement in a sample of smokers (Kauffman et al. 2017).

Taken together, current research suggests that insomnia symptoms may disrupt executive function, which may then impair the ability to effectively regulate emotion, thus conferring vulnerability for RNT. However, no study has examined the relationships between these processes. Thus, the present study assessed insomnia symptoms, executive function, emotion regulation, and RNT over 3 months. It

was hypothesized that insomnia symptoms at time 1 would predict increased RNT at time 4. It was also hypothesized that this relationship would be mediated by executive function at time 2 and emotion regulation at time 3.

## Methods

### Participants

1613 participants enrolled in the study through Research-Match, an online volunteer registry. 1206 participants had complete data and met validity criteria at time 1, 750 participants at time 2, 536 participants at time 3, and 516 participants at time 4. A total of 357 participants (86.6% female) had complete data and met validity criteria at all time points. Participants who scored over 2 standard deviations above the sample mean on the Lie Scale (see Measures) and participants who completed a given survey more than a week after survey receipt were considered invalid and were excluded from analysis.

The mean age of the participants was 45.88 years ( $SD = 13.63$ ), ranging from 19 to 66 years. The ethnicity composition was as follows: White ( $n = 323$ ; 90.5%), African American ( $n = 13$ ; 3.6%), Asian ( $n = 6$ ; 1.7%), Hispanic/Latino ( $n = 4$ ; 1.1%), Other ( $n = 8$ ; 2.2%). Those who did not complete subsequent surveys after Time 1 did not differ from those who completed subsequent time points on gender, race, or baseline insomnia symptoms or diagnostic status. However, there was a significant difference in age between those who completed subsequent time points ( $M = 44.01$ ) and those who did not ( $M = 38.02$ ),  $t(1258) = -6.86$ ,  $p < .01$ .

### Measures

#### Difficulties with Emotion Regulation Scale-16 (DERS-16; Bjureberg et al. 2016)

The DERS-16 is a 16-item self-report scale of emotion regulation problems (e.g., *When I am upset, my emotions feel overwhelming*). Items are rated on a Likert scale from 1 (*almost never*) to 5 (*almost always*), and higher scores indicate poorer emotion regulation. The DERS-16 demonstrated good internal consistency at time 3 ( $\alpha = .96$ ) in the present sample. Sample mean on the DERS-16 was similar to that found in previous community samples (see Table 1; Bjureberg et al. 2016).

#### Insomnia Severity Index (ISI; Bastien et al. 2001)

The ISI is a 7-item self-report measure of insomnia symptoms (e.g., *Difficulty falling asleep*). Items are rated on a

**Table 1** Associations between study variables ( $N=357$ )

| Measures     | ISI<br>T1 | WEX<br>T2 | DERS T3 | PTQ<br>T1 | PTQ<br>T4 | Gender       |
|--------------|-----------|-----------|---------|-----------|-----------|--------------|
| ISI.T1       | –         |           |         |           |           |              |
| WEX.T2       | .39*      | –         |         |           |           |              |
| DERS.T3      | .35*      | .50*      | –       |           |           |              |
| PTQ.T1       | .40*      | .55*      | .64*    | –         |           |              |
| PTQ.T4       | .35*      | .55*      | .69*    | .75*      | –         |              |
| Gender       | –.02      | .03       | .01     | .02       | .04       | –            |
| <i>M</i>     | 11.35     | 12.28     | 29.15   | 23.04     | 20.01     | 86.6% female |
| <i>SD</i>    | 6.20      | 4.22      | 14.00   | 13.29     | 14.13     |              |
| <i>Range</i> | 0–28      | 6–24      | 16–78   | 0–59      | 0–60      |              |

*ISI.T1* Insomnia Severity Index, Time 1, *WEX.T2* Webexec, Time 2, *DERS.T3* Difficulties with Emotion Regulation, Time 3, *PTQ.T1* Perseverative Thinking Questionnaire, Time 1, *PTQ.T4* Perseverative Thinking Questionnaire, Time 4

\* $p < .01$

Likert scale from 1 (*none*) to 4 (*very severe*), and higher scores indicate increased insomnia symptoms. 29.9% of the sample exceeded the cutoff for clinically significant insomnia symptoms (i.e., scores > 14). The ISI demonstrated adequate internal consistency at time 1 ( $\alpha = .88$ ) in the present sample. Sample mean on the ISI was similar to that found in previous community samples (see Table 1; Raines et al. 2015).

### The Abbreviated Eysenck Personality Questionnaire Revised-Lie Scale (Francis et al. 1992)

The Lie Scale is a 12-item self-report measure of socially desirable responding (e.g., *Have you ever taken advantage of someone?*). Items are rated dichotomously (*Yes* or *No*), and higher scores indicate higher socially desirable responding. The Lie Scale demonstrated marginal internal consistency at time 1 ( $\alpha = .67$ ) in the present sample.

### The Perseverative Thinking Questionnaire (PTQ; Ehring et al. 2011)

The PTQ is a 15-item self-report measure of RNT (e.g., *Thoughts intrude into my mind*). Items are rated on a Likert scale from 1 (*never*) to 4 (*almost always*), and higher scores indicate higher RNT. The PTQ demonstrated good internal consistency at time 1 and time 4 ( $\alpha$ 's = .97) in the present sample. Sample mean on the PTQ was similar to that found in previous community samples (see Table 1; Teismann and Forkmann 2017).

### The Webexec (WEX; Buchanan et al. 2010)

The WEX is a 6-item self-report measure of executive function (e.g., *Do you find it difficult to keep your attention on a particular task?*). Items are rated on a Likert scale from

1 (*no problems experienced*) to 4 (*a great many problems experienced*), and higher scores indicate more problems with executive function. Previous research suggests that scores on the WEX are correlated with objective measures of working memory and inhibitory control (Buchanan et al. 2010), although studies have yielded mixed results (see Buchanan 2016). The WEX demonstrated adequate internal consistency at time 2 ( $\alpha = .88$ ) in the present sample. Sample mean on the WEX was similar to that found in previous community samples (see Table 1; Buchanan et al. 2010).

### Procedure

Participants were recruited online through ResearchMatch, a national health volunteer registry created by several academic institutions and supported by the U.S. National Institutes of Health as part of the Clinical Translational Science Award (CTSA) program. ResearchMatch has a large population of volunteers who have consented to be contacted by researchers about health studies. Volunteers who wish to participate in ResearchMatch provide demographic and health-related information (e.g., mental health diagnoses, preexisting medical conditions) during the registration process, and the system later matches potentially eligible volunteers with specific research projects (Harris et al. 2012). Previous research indicates that crowdsourcing platforms are a valid and reliable method for data collection (Sheehan, 2018), and previous studies utilizing ResearchMatch indicate high rates of data validity (Hoerger et al. 2016). To increase validity in the present study, participants who scored over 2 standard deviations above the sample mean on the Lie Scale were excluded from analyses. Participants received four identical surveys via email over 3 months and had the opportunity to enter into a \$25 gift card drawing after completing each survey. Participants who completed

the first survey (time 1) received the second, third, and fourth surveys one (time 2), two (time 3), and three months (time 4), respectively, after first survey completion. Participants were instructed to respond to each survey considering only their experiences over the past month. Given limited previous research in this area, the study period was selected to limit participant burden and attrition. Previous research suggests that results from online data collection are psychometrically comparable to paper and pencil methods (Coles et al. 2007).

Study data were collected and managed using REDCap (Research Electronic Data Capture) hosted at Vanderbilt University (Harris et al. 2009). REDCap is a secure, web-based application designed to support data capture for research studies and is supported by UL1 TR000445 from NCATS/NIH. Review and approval for this study and all procedures was obtained from the Vanderbilt University Institutional Review Board, and informed consent was obtained from all participants.

## Data Analytic Strategy

Data analysis was conducted with SPSS 25. A serial mediation model was tested to examine the indirect effect of insomnia symptoms at time 1 on RNT at time 4 through the association between executive function at time 2 and emotion regulation at time 3. Baseline RNT at time 1 and gender were included as covariates. Mediation analysis was conducted with the PROCESS macro, and bias-corrected bootstrap confidence intervals were calculated to test the significance of the indirect effects (Hayes 2013). Missing

data was addressed with two methods. Prior to data analysis, for measures with one item missing, mean imputation was used to replace the missing item. Measures with more than one missing item were considered missing. Consistent with the PROCESS macro (Hayes 2013), listwise deletion was utilized for missing measures.

## Results

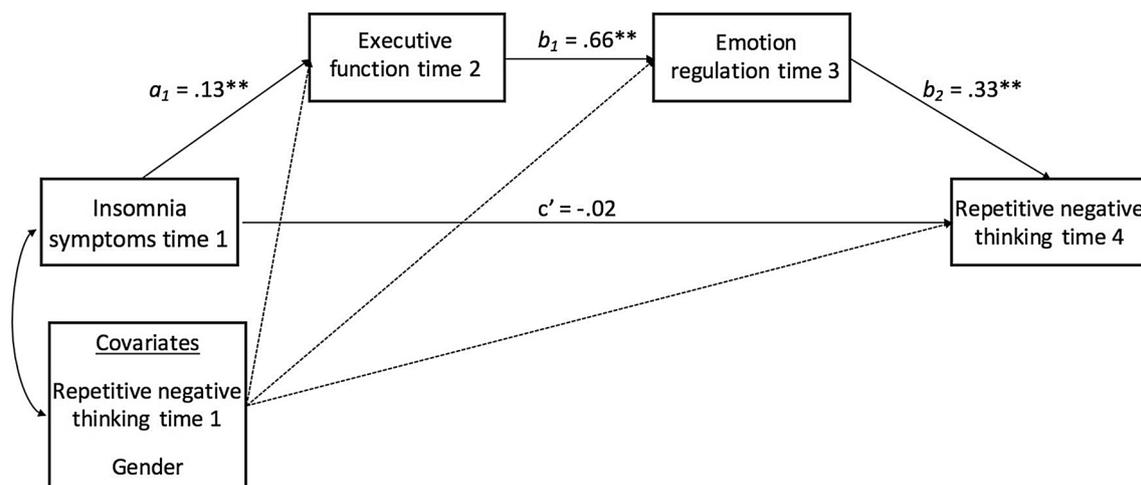
### Associations Between Study Variables

Means and standard deviations for study variables are shown in Table 1. All study variables were significantly correlated,  $p < .01$ , with the exception of gender.

### Mediation Model

As shown in Fig. 1 and Table 2, results of a mediation analysis using ordinary least squares regression revealed that insomnia symptoms at time 1 (baseline) significantly influenced change in RNT at time 4 (month 3) through its effect on executive function at time 2 (month 1) and emotion regulation at time 3 (month 2). Increased insomnia symptoms at time 1 were significantly associated with increased executive function problems at time 2 ( $a_1 = .13$ ), which were significantly associated with increased emotion regulation difficulties at time 3 ( $d_{21} = .66$ ), which were significantly associated with increased RNT at time 4 ( $b_2 = .33$ ).

A 95% bias-corrected bootstrap confidence interval for the serial indirect effect of executive function and emotion regulation ( $a_1 d_{21} b_2 = .03$ ) based on 10,000 bootstrap samples



**Fig. 1** Depiction of the unstandardized beta values of the hypothesized serial mediation model in the serial effect of executive function on emotion regulation mediates the relationship between insomnia symptoms and repetitive negative thinking, controlling for baseline

repetitive negative thinking and gender ( $n = 357$ ). Solid lines represent hypothesized model paths. Dotted lines represent covaried paths;  $**p < .001$

**Table 2** Unstandardized model coefficients for the hypothesized model predicting change in repetitive negative thinking ( $N=357$ )

| Predictor       | Outcome        |                           |      |                 |          |                           |              |       |       |                            |       |       |
|-----------------|----------------|---------------------------|------|-----------------|----------|---------------------------|--------------|-------|-------|----------------------------|-------|-------|
|                 | $M_1$ (WEX.T2) |                           |      | $M_2$ (DERS.T3) |          |                           | $Y$ (PTQ.T4) |       |       |                            |       |       |
|                 | Coeff.         | SE                        | $p$  | Coeff.          | SE       | $p$                       | Coeff.       | SE    | $p$   |                            |       |       |
| $X$ (ISI.T1)    | $a_1$          | .13                       | .03  | <.001           | $a_2$    | .16                       | .10          | .11   | $c'$  | -.02                       | .08   | .81   |
| $M_1$ (WEX.T2)  | –              | –                         | –    | $d_{21}$        | .66      | .16                       | <.001        | $b_1$ | .40   | .13                        | <.01  |       |
| $M_2$ (DERS.T3) | –              | –                         | –    | –               | –        | –                         | –            | $b_2$ | .33   | .04                        | <.001 |       |
| $Cov$ (PTQ.T1)  |                | .15                       | .01  | <.001           |          | .52                       | .05          | <.001 |       | .51                        | .05   | <.001 |
| $Cov$ (Gender)  |                | .28                       | .53  | .69             |          | -.32                      | 1.62         | .84   |       | .89                        | 1.30  | .50   |
| Constant        | $i_{M1}$       | 6.76                      | 1.08 | <.001           | $i_{M2}$ | 7.68                      | 3.47         | <.05  | $i_Y$ | -7.60                      | 2.80  | <.01  |
|                 |                | $R^2=.34$                 |      |                 |          | $R^2=.44$                 |              |       |       | $R^2=.65$                  |       |       |
|                 |                | $F(3, 353)=60.16, p<.001$ |      |                 |          | $F(4, 352)=70.29, p<.001$ |              |       |       | $F(5, 351)=128.62, p<.001$ |       |       |

ISI.T1 Insomnia Severity Index at Time 1, WEX.T2 Webexec at Time 2, DERS.T3 Difficulties with Emotion Regulation-16 at Time 3, PTQ.T1 Perseverative Thinking Questionnaire at Time 1, PTQ.T4 Perseverative Thinking Questionnaire at Time 4

did not contain zero (.01 to .06), suggesting a significant mediation effect through the association between executive function at time 2 and emotion regulation at time 3. Effect size calculations estimating the ratio of the indirect effect to the total effect (Alwin and Hauser 1975) indicate that 25% of the effect of insomnia symptoms at time 1 on RNT at time 4 occurred through the indirect effect. With executive function at time 2 and emotion regulation at time 3 included in the model, there was not a significant direct effect of sleep disturbance at time 1 on RNT at time 4 ( $c' = -.02, p = .816$ ). Contrary to prediction, the total effect of insomnia symptoms at time 1 on RNT at time 4 was also not significant ( $c = .12, p = .176$ ).

Examination of the specific indirect effects revealed that the 95% bias-corrected bootstrap confidence interval for the singular indirect effect of executive function at time 2 ( $a_1b_1 = .05$ ) did not contain zero (.02 to .11), suggesting a significant mediating effect through executive function

alone. In contrast, the 95% bias-corrected bootstrap confidence interval for the singular indirect effect of emotion regulation at time 3 ( $a_2b_2 = .05$ ) did contain zero (-.004 to .12), suggesting that emotion regulation alone did not mediate the relationship between insomnia symptoms and RNT.

**Model Controlling for Baseline Mediators**

To examine whether the indirect effect in the hypothesized model remained significant when controlling for baseline levels of all variables modeled, the model was re-tested with executive function and emotion regulation at time 1 included as covariates (see Table 3). A 95% bias-corrected bootstrap confidence interval for the serial indirect effect of executive function and emotion regulation ( $a_1d_{21}b_2 = .01$ ) based on 10,000 bootstrap samples contained zero (-.002 to .03), indicating a nonsignificant mediation effect through the association between executive

**Table 3** Unstandardized model coefficients for the model predicting change in repetitive negative thinking ( $N=355$ ), controlling for baseline levels of the mediators

| Predictor       | Outcome        |                            |     |                 |          |                            |              |       |       |                           |       |       |
|-----------------|----------------|----------------------------|-----|-----------------|----------|----------------------------|--------------|-------|-------|---------------------------|-------|-------|
|                 | $M_1$ (WEX.T2) |                            |     | $M_2$ (DERS.T3) |          |                            | $Y$ (PTQ.T4) |       |       |                           |       |       |
|                 | Coeff.         | SE                         | $p$ | Coeff.          | SE       | $p$                        | Coeff.       | SE    | $p$   |                           |       |       |
| $X$ (ISI.T1)    | $a_1$          | .03                        | .02 | .22             | $a_2$    | .06                        | .08          | .43   | $c'$  | .002                      | .08   | .98   |
| $M_1$ (WEX.T2)  | –              | –                          | –   | $d_{21}$        | .45      | .20                        | .02          | $b_1$ | .52   | .19                       | .01   |       |
| $M_2$ (DERS.T3) | –              | –                          | –   | –               | –        | –                          | –            | $b_2$ | .45   | .05                       | <.001 |       |
| $Cov$ (Gender)  |                | .26                        | .36 | .46             |          | .15                        | 1.32         | .91   |       | .74                       | 1.23  | .56   |
| $Cov$ (WEX.T1)  |                | .74                        | .04 | <.001           |          | -.20                       | .20          | .32   |       | -.09                      | .19   | .64   |
| $Cov$ (DERS.T1) |                | .01                        | .01 | .63             |          | .67                        | .05          | <.001 |       | -.24                      | .06   | <.001 |
| $Cov$ (PTQ.T1)  |                | .03                        | .01 | .02             |          | .10                        | .05          | <.001 |       | .60                       | .05   | <.001 |
| Constant        | $i_{M1}$       | 1.38                       | .78 | .08             | $i_{M2}$ | 1.89                       | 2.87         | .51   | $i_Y$ | -6.13                     | 2.79  | .03   |
|                 |                | $R^2=.71$                  |     |                 |          | $R^2=.64$                  |              |       |       | $R^2=.66$                 |       |       |
|                 |                | $F(5, 349)=167.17, p<.001$ |     |                 |          | $F(6, 348)=101.96, p<.001$ |              |       |       | $F(7, 347)=97.93, p<.001$ |       |       |

function at time 2 and emotion regulation at time 3 with baseline levels of the mediators included as covariates. Likewise, insomnia symptoms at time 1 were no longer associated with executive function at time 2; however, executive function at time 2 remained significantly associated with emotion regulation at time 3, which remained significantly associated with RNT at time 4 (see Table 3).

### Alternate Mediation Model

To clarify the temporal order of the mediators, an alternate model was tested to examine the indirect effect of emotion regulation at time 2 and executive function at time 3 (see Table 4). A 95% bias-corrected bootstrap confidence interval for the indirect effect of emotion regulation and executive function ( $a_1d_{21}b_2 = .01$ ) based on 10,000 bootstrap samples did not contain zero (.002 to .02), suggesting a significant mediation effect through the association between emotion regulation at time 2 and executive function at time 3. Effect size calculations indicated that 7% of the effect of insomnia symptoms at time 1 on RNT at time 4 occurred through the indirect effect of emotion regulation at time 2 and executive function at time 3. With emotion regulation at time 2 and executive function at time 3 included in the model, there was not a significant direct effect of insomnia symptoms at time 1 on RNT at time 4 ( $c' = -.001, p = .992$ ).

Examination of the specific indirect effects revealed that the 95% bias-corrected bootstrap confidence interval for the singular indirect effect of emotion regulation at time 2 ( $a_1b_1 = .05$ ) did not contain zero (.01 to .11). Likewise, the 95% bias-corrected bootstrap confidence interval for the singular indirect effect of executive function at time 3 ( $a_2b_2 = .06$ ) did not contain zero (.02 to .12).

### Discussion

The present study found partial support for a mediation effect in which executive dysfunction and emotion dysregulation mediated the relationship between insomnia symptoms and RNT over 4 months and account for 25% of this effect. More specifically, these findings suggest that insomnia symptoms are associated with decreased executive function over one month. This is consistent with previous research indicating negative effects of both sleep loss and insomnia symptoms on executive function (Bocca et al. 2014; Khassawneh et al. 2018) and extends these findings by showing an association between insomnia symptoms and executive function from time 1 to time 2; however, this relationship was no longer significant when executive function at time 1 was included in the model. Previous findings suggest that sleep deprivation is linked to deficits in executive function (Drummond et al. 2006) and individuals with insomnia exhibit decreased executive function compared to controls (Ballesio et al. 2019). The present findings suggest that the insomnia-executive function link may be better detected at a closer time lag than 1 month. Although a significant zero order correlation was observed between insomnia symptoms and emotion regulation, insomnia symptoms were not associated with emotion regulation in the full model. Previous research suggests that poor sleep quality may impair individuals' ability to engage in effective emotion regulation (Mauss et al. 2013). However, this finding has not been consistently replicated (Reddy et al. 2017). The present results suggest that an observed link between poor sleep and emotion regulation is likely to be accounted for by other factors (i.e., executive function).

Consistent with previous cross-sectional research (Hendricks and Buchanan 2016), problems with executive function were associated with worse emotion regulation from time 2 to time 3. Given the role of executive function in

**Table 4** Unstandardized model coefficients for the alternate model predicting repetitive negative thinking ( $N = 360$ )

| Predictor       | Outcome  | $M_1$ (DERS.T2)                |      |       | $M_2$ (WEX.T3) |                               |      | $Y$ (PTQ.T4) |       |                                |      |       |
|-----------------|----------|--------------------------------|------|-------|----------------|-------------------------------|------|--------------|-------|--------------------------------|------|-------|
|                 |          | Coeff.                         | SE   | $p$   | Coeff.         | SE                            | $p$  | Coeff.       | SE    | $p$                            |      |       |
| $X$ (ISL.T1)    | $a_1$    | .21                            | .09  | <.05  | $a_2$          | .11                           | .03  | <.001        | $c'$  | -.001                          | .08  | .99   |
| $M_1$ (DERS.T2) |          | –                              | –    | –     | $d_{21}$       | .07                           | .02  | <.001        | $b_1$ | .25                            | .05  | <.001 |
| $M_2$ (WEX.T3)  |          | –                              | –    | –     | –              | –                             | –    | –            | $b_2$ | .56                            | .14  | <.001 |
| $Cov$ (PTQ.T1)  |          | .71                            | .04  | <.001 |                | .08                           | .02  | <.001        |       | .51                            | .05  | <.001 |
| $Cov$ (Gender)  |          | -.54                           | 1.49 | .72   |                | .23                           | .52  | .66          |       | 1.05                           | 1.35 | .44   |
| Constant        | $i_{M1}$ | 12.44                          | 3.01 | <.001 | $i_{M2}$       | 6.41                          | 1.09 | <.001        | $i_Y$ | -8.22                          | 2.93 | <.01  |
|                 |          | $R^2 = .52$                    |      |       |                | $R^2 = .31$                   |      |              |       | $R^2 = .61$                    |      |       |
|                 |          | $F(3, 356) = 128.04, p < .001$ |      |       |                | $F(4, 355) = 39.49, p < .001$ |      |              |       | $F(5, 354) = 112.97, p < .001$ |      |       |

ISL.T1 Insomnia Severity Index at Time 1, DERS.T2 Difficulties with Emotion Regulation-16 at Time 2, WEX.T3 Webexec at Time 3, PTQ.T1 Perseverative Thinking Questionnaire at Time 1, PTQ.T4 Perseverative Thinking Questionnaire at Time 4

regulating cognitive processes (Miyake et al. 2000), effective emotion regulation may rely on intact executive function. For example, shifting abilities may support reappraisal, while impaired inhibition may limit the ability to direct attention away from negative stimuli. Indeed, recent evidence suggests that cognitive control interventions positively impact emotion regulation (Hoorelbeke et al. 2016). In addition to emotion regulation, the present study also found that decreased executive function was associated with increased RNT. This finding is consistent with previous research indicating a prospective relationship between executive processes and RNT (Cox et al. 2018). Problems with emotion regulation were also associated with increased RNT over 1 month, which is consistent with previous research implicating emotion regulation deficits in disorders characterized by RNT (Berking et al. 2014). Over time, diminished ability to adaptively and flexibly respond to negative experiences may result in excessive worry and rumination.

Consistent with predictions, the relationship between insomnia symptoms at time 1 and RNT at time 4 was significantly mediated by the indirect effect of executive function at time 2 and emotion regulation at time 3. That is, poor executive function was associated with poor emotion regulation, and this effect accounted for 25% of the relationship between insomnia symptoms and RNT. This finding is consistent with research implicating deficits in higher-order cognitive function in the link between sleep disturbance and RNT (Nota and Coles 2018), and suggests that emotion regulation is one affective process by which decreased executive function impacts RNT. Poor sleep may impair executive processes, which may then limit one's ability to effectively regulate emotions. In the absence of adaptive emotion regulation, individuals may respond to emotion-inducing events with maladaptive strategies, such as RNT. For example, when stressed, a poor sleeper may lack the shifting and/or inhibitory abilities necessary to utilize reappraisal, leaving them vulnerable to intrusive, repetitive cognition. This interpretation is supported by research suggesting the brain regions that support the executive function necessary to engage in emotion regulation are also those impacted by sleep loss (Ochsner and Gross 2005; Yoo et al. 2010), as well as findings from behavioral studies linking decreased cognitive control to RNT (Whitmer and Banich 2007). Similarly, given detrimental effects of poor sleep on positive affect (Reddy et al. 2017), deficient positive affect may also contribute to emotion regulatory deficiencies and symptoms of emotional disorders (Hofmann et al. 2012), such as RNT.

Closer examination of the mediators revealed a significant specific indirect effect of executive function at time 2 on the relationship between insomnia symptoms at time 1 and RNT at time 4, but a nonsignificant specific indirect effect of emotion regulation at time 3. This suggests that the impact of poor emotion regulation on the relationship between

insomnia symptoms and RNT may be due to diminished shifting, updating, and/or inhibitory abilities, which is consistent with previous research suggesting that effective emotion regulation is supported by cognitive control processes, and impaired cognitive control is linked to RNT (Joormann and Gotlib 2010). These differential indirect effects also suggest that executive function is more relevant than emotion regulation in explaining the relationship between insomnia symptoms and RNT. However, it is also important to consider that both specific indirect effects had a similar effect size, accounting for 46 and 45%, respectively of the effect of insomnia at time 1 on RNT at time 4. Thus, it is possible that the null finding for the specific indirect effect of emotion regulation at time 3 is due to characteristics of the model tested or sample.

Notably, the total effect of insomnia symptoms at time 1 on RNT at time 4 was not significant. However, current mediation theory proposes that a significant total effect is not necessary in order to test a mediation model (Hayes, 2013). Otherwise stated, the degree to which an independent variable influences a change in a dependent variable through a mediator is not predicated on the ability to observe a relationship between the independent and dependent variables without considering the mediator. These results highlight the importance of testing potential mediators when studying the prospective relationship between sleep disturbance and symptoms of psychopathology, as singular regression coefficients may be misleading.

However, it is important to note that the indirect effect was no longer significant with baseline levels of the mediators included as covariates. One interpretation of these discrepant indirect effects is that without inclusion of the mediators at time 1, the observed significant indirect effect is actually reflecting cross-sectional associations between variables that do not exist within a causal pathway. Alternatively, given that the only individual pathway that became nonsignificant in the controlled model was the link between time 1 insomnia symptoms and time 2 executive function, the nonsignificant indirect effect may be driven by an inappropriate lag selection at which to detect the hypothesized  $a$  pathway (e.g., Cole and Maxwell 2003). A third possible explanation regards measurement error. That is, all relationships in the model may be underestimated due to the use of single indicators of constructs of interest, and a proliferation of measurement error may limit the ability to detect small effects in a highly controlled model.

Although the indirect effect in the alternate model in which emotion regulation at time 2 and executive function at time 3 mediated effect of insomnia symptoms on RNT was also significant, the reduction in effect size suggests that this model may be inferior to the hypothesized model. Whereas 25% of the effect of insomnia symptoms on RNT occurred through the indirect effect in the hypothesized model, 7%

of this effect occurred through the alternate indirect effect. However, it is important to note that both models represent small effects. This model also revealed significant specific indirect effects for emotion regulation at time 2 and executive function at time 3, in contrast to the differential specific indirect effects in the hypothesized model. These findings further support the interpretation that the effect of emotion regulation in the relationship between insomnia symptoms and RNT is largely dependent on executive function. That is, the unique effect of emotion regulation is only observable when prior levels of executive function are not accounted for. Additional research utilizing experimental paradigms is necessary to better understand the role of executive function in the relationship between insomnia symptoms and emotion regulation.

The associations in the present study suggest that insomnia symptoms may predict change in RNT over time through an effect on executive function and emotion regulation. However, future prospective studies are needed to determine this. Identifying cognitive and affective mechanisms may clarify etiological models of disorders characterized by sleep disturbance and generate novel treatment targets. Indeed, these findings may have important clinical implications for disorders characterized by RNT. Previous research suggests cognitive behavior therapy for insomnia (CBTI) is effective in reducing insomnia symptoms, as well as symptoms of PTSD and depression, two disorders characterized by RNT (e.g., Manber et al. 2011; Ho et al. 2016). Thus, clinicians may benefit from using CBTI as an adjunctive intervention in the treatment of RNT-related disorders. Additionally, treatments that target executive function deficits (e.g., attentional bias modification; MacLeod and Clarke 2015) may yield better results when combined with CBTI.

These results should be considered within the context of the study limitations. First, the sample included unselected participants, precluding the ability to generalize these findings to clinical samples. Additional research is necessary to determine if the present results extend to clinical populations. Second, the sample was predominantly white and female, limiting the ability to generalize this model to various demographic groups. Similarly, given increased attrition among younger participants, the present findings may not generalize to younger adults. Likewise, the large attrition rate also diminishes generalizability. Thus, replication in representative samples is necessary. Third, the present study utilized self-report instruments that may be incomplete measures of the desired constructs, and participants' subjective responses may differ from objective data. Further, single-measure methods are vulnerable to estimate inflation due to shared method variance (Cole and Maxwell 2003). Future studies should expand upon previous research utilizing multi-method ecological momentary assessment indicating relations between daily sleep and anxiety-related

outcomes (Short et al. 2017; Cox et al. 2018). Fourth, it is unclear if this model would yield the same results over different time intervals. Additional research is necessary to continue to probe these relationships at various lags. Fifth, the present study does not examine potential bidirectional relationships between the study variables (see Takano et al. 2014). Further research is necessary to elucidate potential feedback loops between these processes. Finally, the lack of manipulation of sleep limits causal interpretations. While this study offers a first step towards determining causality, causal conclusions cannot be drawn without experimental investigation. Future research utilizing experimental sleep restriction is necessary to fully examine the causal effect of insomnia symptoms on the relationships between cognitive and affective function.

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## Compliance with Ethical Standards

**Conflict of Interest** Rebecca C. Cox, Sarah C. Jessup, and Bunmi O. Olatunji declare that they have no conflict of interest.

**Informed Consent** All procedures in this study were in accordance with the ethical standards of the institutional review board, and informed consent was obtained from all individual subjects who participated in the study.

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

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