J. Behav. Ther. & Exp. Psychiat. 50 (2016) 23–32

Contents lists available at ScienceDirect

Journal of Behavior Therapy and Experimental Psychiatry

journal homepage: www.elsevier.com/locate/jbtep

Sleep disruption is related to poor response inhibition in individuals with obsessive–compulsive and repetitive negative thought symptoms

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ARTICLE INFO

Article history:
Received 23 December 2014
Received in revised form
22 April 2015
Accepted 25 April 2015
Available online 6 May 2015

Keywords:
Obsessive–compulsive
Repetitive negative thinking
Sleep
Bedtimes
Response inhibition

ABSTRACT

Background and objectives: Obsessive–compulsive (OC) symptoms and repetitive negative thinking (RNT) are associated with poor inhibitory control. Sleep disruptions may partially mediate these relations and/or act as a "second hit" to individuals with OC symptoms and RNT. Models including habitual (past month) hours slept and bedtimes were tested.
Methods: We employed a go/no-go task that allowed us to examine the relation between sleep and inhibition with various task contingencies. Sixty-seven unselected individuals were recruited from the participant pool at a public university.
Results: Bias-corrected bootstrap estimates did not show that sleep disruption mediated the relation between OC symptoms and response inhibition nor the relation between RNT and response inhibition. Multiple linear regression analyses found significant interactions between hours slept and OC symptom severity and between RNT and hours slept to predict poor response inhibition. Hours slept significantly negatively predicted commission errors when OC symptoms and RNT levels were relatively heightened but not when OC symptoms and RNT levels were relatively low. These effects were present in blocks where task contingencies were designed to shape a no-go bias. No significant relations were found with habitual bedtimes.
Limitations: The cross-sectional study design precludes testing the temporal precedence of symptoms in the "second hit" model. The unselected sample also limits generalization to clinical samples.
Conclusions: These findings support a "second hit" model of interaction between sleep disruption and perseverative thoughts and behaviors. Further research on the mechanisms of the relation between sleep disruption and perseverative thought symptoms (OC and RNT) is warranted.

1. Introduction

There is a growing literature showing that symptoms of obsessive–compulsive disorder (OCD) are often related to disruptions in sleep and circadian rhythms. A number of studies have now documented significant correlations between habitual delayed bedtimes and OC symptoms (Coles, Schubert, & Sharkey, 2012; Nota, Coles, & Sharkey, 2015; Schubert & Coles, 2013, 2015). For example, Nota and Coles (2015) found that distress associated with OC symptoms was correlated with shorter sleep duration and delayed bedtimes in a sample of undergraduate students. Similarly, Coles et al. (2012) found that the frequency of OC symptoms was significantly higher in individuals with delayed bedtimes compared to those without delayed bedtimes in a non-clinical sample. Recent meta-analytic findings document a significant reduction in total sleep duration and increase in the prevalence of delayed sleep phase disorder (DSPD; Weitzman et al., 1981) in individuals with an OCD diagnosis compared to healthy controls (Nota et al., 2015). Indeed, several studies suggest that individuals with OCD and sleep disruption may have particularly severe symptoms and may benefit less from treatment (Mukhopadhyay et al., 2008; Turner et al., 2007).

Previous studies have focused primarily on documenting the relationship between OCD and sleep disruption, but have not addressed the potential underlying mechanisms and moderators.

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http://dx.doi.org/10.1016/j.jbtep.2015.04.011
0005-7916/

Since approximately 1/3 of patients do not respond to current treatments and even "responders" are left with residual symptoms after treatment (Simpson, Huppert, Petkova, Foa, & Liebowitz, 2006), efforts to understand core features underlying OCD symptoms (e.g., deficits in response inhibition) and to identify modifiable contributors (e.g., sleep disruption) are important avenues for potentially improving our interventions. Given that OCD is associated with impairments in certain cognitive functions (e.g., inhibitory control) and sleep disruption is known to negatively impact these functions, these represent logical candidate processes to understand the relation between OCD and sleep disruption.

1.1. Inhibitory control and OC symptoms

OC symptoms are associated with both deficits in inhibitory control of responses and the ability to adapt responses to changes in environmental contingencies (Chamberlain et al., 2007, 2008; Menzies et al., 2007). Individuals with OCD also show differences in neural correlates of response inhibition and flexibility compared to healthy controls, including structural and functional changes in fronto-striatal circuits and orbitofrontal-striatal circuits (for review see: Menzies et al., 2008; Milad & Rauch, 2012). Indeed, the clinical presentation of OCD is consistent with the tasks used to evaluate ones ability to inhibit responses. In the experimental tasks used to assess response inhibition the participant is typically presented with a series of trials where they respond by pressing a key, but are then signaled to not respond due to a change in the appearance of the stimuli (e.g., shape, color, etc.); this is difficult as the participant has built up a habit of responding. Individuals with OCD may have particular difficulty altering their response patterns (Chamberlain et al., 2007; Nielen, den Boer, & Smid, 2009; Watkins et al., 2005) and inhibiting responses (Morein-Zamir et al., 2013) when given punishing feedback (Gillan et al., 2011). Indeed, Gillan et al. (2011) found that individuals with OCD were more prone to maintain habitual responding compared to healthy controls in response to outcome devaluations in an experimental task. Generally, these findings are taken to support a model of OCD vulnerability whereby impairments in inhibitory control give rise to the more complex behaviors that define the disorder (i.e., intrusive repetitive thoughts and excessive perseverative behaviors).

1.2. Inhibitory control and repetitive negative thinking

In fact, deficits in inhibitory control may be related to the process of perseverative thoughts and behaviors across many disorders (Amodio, Master, Yee, & Taylor, 2008; Eagle, Bari, & Robbins, 2008; Linville, 1996). Individuals with a number of mood and anxiety disorders have been found to show deficits in inhibitory control (Derryberry & Reed, 2002; Joormann & Gotlib, 2010). These findings support the possibility of a shared vulnerability across disorders. Repetitive negative thinking (RNT), defined as a perseverative and abstract focus on negative aspects of one's experience that is experienced as difficult to control (Watkins, 2008), is increasingly understood to be a transdiagnostic process associated with poor inhibitory control and anxiety and mood psychopathology (Bird, Mansell, Dickens, & Tai, 2013; Hallion, Ruscio, & Jha, 2014; Moore et al., 2013). Indeed, the content of symptoms across individuals may differ (e.g., OCD, depression, anxiety) but this more general process may be more closely associated with impairments in inhibitory control, representing a basic vulnerability that may be activated in individuals who manifest symptoms.

1.3. Inhibitory control and sleep disruptions

Previous studies have found that response inhibition and flexibility are impaired by sleep disruption (Anderson & Platten, 2011; Drummond, Paulus, & Tapert, 2006). For example, Drummond et al. (2006) found that individuals were more likely to make commission errors during a go/no-go task after 22 h of wakefulness compared to well-rested baseline. The same level of impairment in response inhibition was found after up to 55 h of total sleep deprivation (Drummond et al., 2006). However, some studies have failed to find this relation in less extreme circumstances of sleep disruption (Fallone, Acebo, Arnedt, Seifer, & Carskadon, 2001; Schubert & Coles, 2013). Evidence suggests that prefrontal cortical areas of the brain are particularly sensitive to disruptions in sleep and circadian rhythms (Borbely & Achermann, 2005; Cajochen, Blatter, & Wallach, 2004; Muzur, Pace-Schott, & Hobson, 2002), perhaps as a consequence of their high metabolic load during conscious functioning (Borbely & Achermann, 2005; Finelli, Borbély, & Achermann, 2001). Across electroencephalographic and molecular measures, prefrontal cortical areas show the greatest drive to sleep after a period of wakefulness (Cajochen, Foy, & Dijk, 1999; Muzur et al., 2002; de Sanchez et al., 1993). These include prefrontal, inhibitory, areas of fronto-striatal circuits implicated in response inhibition and flexibility.

1.4. The current study

Given the effects of sleep and circadian rhythms on inhibitory performance and prefrontal cortical function, it is expected that disruptions in these bioregulatory systems would interact with existing deficits in individuals with OC symptoms and RNT. In other words, disruptions in sleep may act as a "second hit" activating vulnerabilities present in individuals and thus maintaining their perseverative symptoms by negatively affecting their ability to inhibit and respond flexibly. Such models relating sleep and other forms of psychopathology are emerging in the literature (cf. Nofzinger et al., 2005; Pritchett et al., 2012; Voigt et al., 2014). This may be evident if disruptions in sleep partially account for the relation between symptoms and measures of inhibitory control; or alternatively, if symptoms moderate the strength of relation between sleep disruption and inhibitory control. A recent study demonstrated that RNT is associated with shorter habitual sleep duration and delayed bedtimes (Nota & Coles, 2015), however, there is a need to extend from these findings by better understanding the mechanisms and moderators of this relation.

In this study we treated inhibitory control as a potential "shared substrate" between OC and RNT symptoms and sleep disruption. Even though response inhibition is commonly thought to "precede" symptoms, we chose to treat it as our "outcome" variable in relation to sleep disruption and symptoms for the sake of greater ease in interpretation in this initial cross-sectional study. This choice was based on our working model that a tendency toward perseverative thought and behavior increases vulnerability to the negative effects of sleep disruption on response inhibition. We employed the go/no-go task used by Morein-Zamir et al. (2013) to measure response inhibition. This task was selected to facilitate comparisons to prior OCD studies (Chamberlain, Fineberg, Blackwell, Robbins, & Sahakian, 2006; Chamberlain et al., 2007, 2008; Menzies et al., 2007) and because it relies on sustained effort and the inhibition of pre-potent responses, which is known to be impacted by sleep disruption (Anderson & Platten, 2011; Fallone et al., 2001; Zhou et al., 2011). Further, this task allowed us to evaluate response inhibition under different contingencies (e.g., punish inhibition, reward response, punish response, and reward inhibition).