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Sleep Disturbance in Obsessive-Compulsive Disorder: Preliminary Evidence for a Mechanistic Relationship

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Abstract

Purpose of Review Sleep disturbance has received growing attention as a transdiagnostic factor that may contribute to multiple forms of psychopathology, including obsessive-compulsive disorder (OCD). The extant literature regarding the role of sleep disturbance in OCD is rapidly developing. This review integrates the findings related to sleep and OCD over the past year and highlights important areas for future research.

Recent Findings Results indicate impaired sleep in those with OCD versus healthy controls and link sleep disturbance to OCD symptom severity in multiple samples. Findings from the past year also implicate cognitive control as a potential mechanism in this relationship. Finally, a recent treatment outcome study suggests that sleep disturbance may limit treatment response in pediatric OCD.

Summary Findings from the past year contribute to the robustness of the small, but growing body of literature linking sleep disturbance to OCD. These results highlight the importance of incorporating sleep disturbance into extant models of OCD and suggest that consideration of the biopsychosocial effects of sleep disturbance may offer novel insight into the etiology and treatment of OCD.

Keywords Sleep · OCD · Cognitive control · Treatment response

Introduction

Sleep disturbance has been consistently linked to psychopathology [1, 2]. Despite this robust relationship, sleep disturbance has largely been considered an epiphenomenon of psychopathology and has thus remained understudied. However, given the cascade of downstream effects of sleep loss and overlap between neurobiological systems related to sleep and psychopathology, sleep disturbance has recently been proposed as a mechanistic transdiagnostic factor [3]. That is, sleep disturbance may contribute to the development of various forms of psychopathology through its negative impact on functioning across multiple systems.

Though extant research examining sleep and psychopathology has largely focused on depression and postraumatic stress disorder (PTSD), sleep disturbance has received recent attention within the context of obsessive-compulsive disorder (OCD). Indeed, a recent meta-analysis revealed alterations in multiple sleep parameters among those with OCD compared to controls, even when excluding those with comorbid depression [4]. Further, multiple sleep parameters are associated with OCD symptom severity, including slow-wave sleep percentage [5], rapid eye movement (REM) density [5, 6], and total number of subjective sleep problems [7]. Finally, sleep disturbance may interfere with the treatment of OCD. Preliminary evidence in pediatric OCD samples suggests that baseline sleep disturbance predicts worse treatment outcome, and those who do not respond to cognitive behavior therapy (CBT) are more likely to report residual sleep disturbance than responders [8].

Taken together, these findings highlight the role of sleep disturbance in OCD. Given increasing interest in this relationship in recent years, as well as the relatively small body of extant research, integration of the most up to date findings on sleep disturbance and OCD is necessary to identify the most

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fruitful areas for future research. As such, the purpose of the present review is to synthesize findings related to sleep disturbance and OCD over the past year. Conclusions and future directions for the role of sleep disturbance in OCD will be discussed, including ways in which sleep disturbance might be incorporated into existing models of OCD.

Recent Findings on Sleep and OCD

Recent research continues to link sleep disturbance to OCD, and this relationship is evident across a range of indices, including group differences, relation to symptom severity, and treatment outcome. In the only recent study of sleep in an OCD sample, those with OCD exhibited subjective and objective sleep disturbance compared to controls [9•]. For example, the OCD group reported increased global sleep disturbance, with evidence for specific disturbance in sleep quality, sleep onset latency (SOL), sleep efficiency, and daytime impairment [9•]. Evidence for subjective sleep disturbance in the OCD group was consistent with actigraphy assessment of sleep, which indicated decreased total sleep time (TST) and sleep efficiency and increased SOL compared to controls, with medium effect sizes [9•]. The concordance between subjective and objective sleep deficits suggests that the sleep disturbance reported by those with OCD represent genuine deficits in sleep rather than an overestimation of ancillary symptoms. Interestingly, this study found divergent results for TST, such that those with OCD reported comparable TST to controls, but objectively exhibited decreased TST. One interpretation of these findings is that individuals with OCD may experience restless sleep that accelerometers interpret as increased wakefulness. Such restlessness may then contribute to the interpretation of the sleep as poor quality.

Recent findings have also linked sleep disturbance to OCD symptoms in multiple nonclinical samples. In a sample of unselected college students, subjective sleep disturbance was associated with OCD symptoms [10]. A similar relationship was found in a nationally representative sample, even when controlling for depression [11]. This study also found that those who reported sleep disturbance reported increased OCD symptoms compared to those without sleep disturbance [11]. These findings highlight the relationship between sleep disturbance severity and OCD symptom severity. In contrast, one recent study found no relationship between subjective TST and OCD symptoms in an unselected sample [12]. This finding is consistent with Donse et al. [9•] and provides additional evidence that perceived sleep duration may not be a sensitive indicator of sleep disturbance in OCD.

While recent studies largely link sleep disturbance severity to OCD symptom severity, such cross-sectional approaches offer limited ability to examine temporality. That is, it is unclear from these findings whether sleep disturbance contributes to OCD symptoms or vice versa. However, one recent study utilizing a longitudinal design found that sleep disturbance predicted obsessions over 6 months [13•], suggesting that sleep disturbance may precede OCD symptoms. This finding is consistent with previous research indicating that insomnia predicts the development of anxiety disorders in children [14] and adults [15], but contradicts a recent study which found a unidirectional relationship between insomnia symptoms and OC symptoms in adolescents over 6 months [16]. Thus, the temporal link between sleep and OCD symptoms remains unclear, though extant findings may suggest a bidirectional relationship where sleep disturbance exacerbates OCD symptoms and vice versa.

Despite the consistent link between sleep disturbance and OCD, few studies have examined mechanisms that may account for this relationship. However, one recent study found that the relationship between sleep disturbance and obsessions over 6 months was mediated by focusing attentional control [13•], suggesting that the negative effects of sleep disturbance on cognitive function contribute to increased obsessions. This finding provides preliminary evidence for a potential mechanism that may link sleep disturbance and OCD; however, given the relative dearth of mechanistic studies in this field, considerable additional research is necessary to replicate this finding and identify other candidate mechanisms. Similarly, few studies have examined the downstream effects of this relationship on other processes. However, one recent study found that sleep disturbance moderated the effect of OCD symptoms on inhibitory control [12], such that those with high OCD symptoms and decreased TST exhibited increased commission errors on a Go/NoGo task. Importantly, findings from this study revealed no main effect of OCD symptoms on inhibitory control, suggesting the importance of considering sleep disturbance when examining the relationship between OCD and higher-order cognitive function. However, both of these studies are limited by reliance on unselected samples. Future research in this area will benefit from the utilization of OCD populations.

Finally, recent evidence suggests that sleep disturbance may play a role in OCD treatment. Indeed, one study examining the efficacy of repetitive transcranial magnetic stimulation (rTMS) found that treatment non-responders reported increased pre-treatment sleep disturbance compared to responders [9•], suggesting that sleep disturbance may interfere with treatment efficacy. Alternatively, elevated sleep disturbance may be a marker of more severe, treatment-refractory OCD. This interpretation is consistent with previous evidence that baseline sleep disturbance predicted diminished response to CBT for pediatric OCD [8]. However, in discriminant analyses, Donse et al. [9•] found that sleep disturbance could not predict responders vs non-responders, though circadian rhythm disturbances did. Given recent attention to the role of circadian rhythms in psychiatric disorders [17], including

OCD [4], sleep disturbance in OCD may represent a biomarker of underlying circadian rhythm abnormality.

Conclusions and Future Directions

Consistent with extant research, findings from the past year link sleep disturbance to OCD. Those with OCD exhibit both subjective and objective sleep disturbance, indicating that sleep problems among those with OCD represent a true physiological deficit rather than a misperception. Further, subjective sleep disturbance is linked to OCD symptoms in multiple samples both cross-sectionally and prospectively, suggesting that sleep disturbance is not only concurrent with but also precedes OCD symptoms. Recent attention has also been given to the role of cognitive control within the context of sleep and OCD, with results highlighting decreased cognitive control as a candidate mechanism in this relationship. Finally, findings from the past year replicate previous research linking sleep disturbance to diminished treatment response. Taken together, the results from the past year continue to implicate sleep disturbance in OCD. Further, these findings provide additional specificity to this relationship by highlighting the divergence of subjective and objective TST, providing preliminary evidence for the role of cognitive function, and extending treatment outcome results beyond CBT to rTMS.

Given accumulating evidence for a role of sleep in OCD, an important next question is how to incorporate sleep into etiological conceptualizations of OCD? Current models of OCD posit a diathesis-stress model in which the interaction of genetic vulnerabilities and environmental stressors results in hyperactivity in cortico-striato-thalamo-cortical circuitry [18, 19]. That is, the combination of certain genes and environmental factors is thought to cause abnormal brain activity, which then manifests as the intrusive cognition and repetitive behavior characteristic of OCD. How then might sleep disturbance function in such a model? It may be that sleep disturbance is an environmental stressor that interacts with genetic vulnerabilities to influence symptom expression. Indeed, sleep disturbance has recently been proposed as a stressor that can contribute to allostatic overload, or the hypo/hyperactivity of systems that maintain physiological homeostasis [20].

This hypothesis is supported by considerable research delineating the negative consequences of sleep disturbance for whole system function, including processes linked to OCD. For example, sleep loss is associated with alterations in immune system function [21], including increased inflammatory cytokines [22]. Similarly, dysregulated immune function has been implicated in OCD and related disorders, most clearly in the case of pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections [PANDAS; [23]]. Further, sleep loss results in blunted cortisol reactivity to an acute stressor [24], and decreased sleep and sleep variability are linked to alterations in the diurnal cortisol rhythm [25]. Dysregulated cortisol has likewise been implicated in OCD [26]. Thus, immune and cortisol responses are two candidate stress responses to sleep disturbance that may interact with genetic predispositions to confer vulnerability for OCD.

Sleep disturbance and OCD also share linkages to deficits in cognitive function. Sleep loss and disturbance results in diminished executive function [27], including deficits in inhibitory control [28] and working memory [29]. These deficits are further linked to altered activity in frontal and thalamic regions [29, 30]. Importantly, these regions are also implicated in the neurocircuitry of OCD [19], and considerable research has identified higher-order cognitive deficits in OCD, particularly regarding inhibitory control and working memory [18]. Taken together, these findings suggest that sleep disturbance impairs multiple processes necessary for adaptive function in a similar fashion to a major stressor. When experienced in an individual with a genetic predisposition, sleep disturbance may contribute to the development of OCD.

Further, recent attention has been given to the role of epigenetic factors in OCD; that is, environmental influences may modify the expression of certain genes, and these alterations may have downstream effects on neural activity that contributes to OCD [19]. Indeed, recent research has linked epigenetic processes to OCD [31, 32]. One possibility is that sleep disturbance functions as an epigenetic factor that alters the expression of genes related to OCD. Indeed, recent findings suggest that both acute sleep deprivation and insomnia symptoms are associated with epigenetic indicators [33, 34]. Though these findings are preliminary, they suggest that examining sleep disturbance as an epigenetic factor in OCD may be fruitful.

Future research would benefit from beginning to test specific mechanisms that may account for the relationship between sleep disturbance and OCD. Further, extant research in this area is limited by an overreliance on correlational and cross-sectional methods. Additional research utilizing experimental sleep restriction paradigms and longitudinal designs are necessary to move beyond associating sleep disturbance with OCD toward determining whether sleep disturbance causally contributes to OCD. Finally, future research on the role of sleep disturbance in treatment outcomes is necessary to clarify how sleep disturbance may contribute to treatment nonresponse. OCD is a highly heterogeneous disorder; thus, OCD characterized by sleep disturbance may represent a unique variant of OCD that is less responsive to current treatments. Further, future research is necessary to examine the role of sleep disturbance in OCD treatment outcome among adult samples. These gaps in the literature represent important areas for future research to further clarify the relationship between sleep disturbance and OCD.

Compliance with Ethical Standards

Conflict of Interest Rebecca Cox, Sarah Jessup, and Bunmi Olatunji each declare no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:
• Of importance

1. Baglioni C, Nanovska S, Regen W, Spiegelhalder K, Feige B, Nissen C, et al. Sleep and mental disorders: a meta-analysis of polysomnographic research. Psychol Bull. 2017;142:969–90. https://doi.org/10.1037/bul0000053.
2. Benca RM, Obermeyer WH, Thisted RA, Gillin JC. Sleep and psychiatric disorders: a meta-analysis. Arch Gen Psychiatry. 1992;49:651–68.
3. Harvey AG, Murray G, Chandler RA, Soehner A. Sleep disturbance as transdiagnostic: consideration of neurobiological mechanisms. Clin Psychol Rev. 2011;31:225–35.
4. Nota JA, Sharkey KM, Coles ME. Sleep, arousal, and circadian rhythms in adults with obsessive–compulsive disorder: a meta-analysis. Neurosci Biobehav R. 2015;51:100–7. https://doi.org/10.1016/j.neubiorev.2015.01.002.
5. Insel TR, Gillin JC, Moore A, Mendelson WB, Loewenstein RJ, Murphy DL. The sleep of patients with obsessive-compulsive disorder. Arch Gen Psychiatry. 1982;39:1372–7.
6. Voderholzer U, Riemann D, Huwig-Poppe C, Kuelz AK, Kordon A, Bruestle K, et al. Sleep in obsessive compulsive disorder: polysomnographic studies under baseline conditions and after experimentally induced serotonin deficiency. Eur Arch Psychiatry Clin Neurosci. 2007;257:172–82. https://doi.org/10.1007/s00406-006-0708-9.
7. Storch EA, Murphy TK, Lack CW, Geffken GR, Jacob ML, Goodman WK. Sleep-related problems in pediatric obsessive-compulsive disorder. J Anxiety Disord. 2008;22:877–85. https://doi.org/10.1016/j.janxdis.2007.09.003.
8. Ivarsson T, Skarphedinsson G. Sleep problems and cognitive behavior therapy in pediatric obsessive-compulsive disorder have bidirectional effects. J Anxiety Disord. 2015;30:28–33. https://doi.org/10.1016/j.janxdis.2014.12.009.
9.• Donse L, Sack AT, Fitzgerald PB, Arns M. Sleep disturbances in obsessive-compulsive disorder: association with non-response to repetitive transcranial magnetic stimulation (rTMS). J Anxiety Disord. 2017;49:31–9. https://doi.org/10.1016/j.janxdis.2017.03.006. **Those with OCD report and exhibit increased sleep disturbance compared to healthy controls. rTMS non-responders reported increased baseline sleep disturbance compared to treatment responders**.
10. Yu CKC. Obsessions at night: dream experiences, emotional attributes, and personality traits as indicators of sleep problems. Sleep Hypn. 2016;18:30–40. https://doi.org/10.5350/Sleep.Hypn.2016.18.0106.
11. Cox RC, Olatunji BO. Sleep disturbance and obsessive-compulsive symptoms: results from the national comorbidity survey replication. J Psychiatr Res. 2016;75:41–5. https://doi.org/10.1016/j.jpsychires.2016.01.007.
12. Nota JA, Schubert JR, Coles ME. Sleep disruption is related to poor response inhibition in individuals with obsessive-compulsive and repetitive negative thought symptoms. J Behav Ther & Exp Psychiatr. 2016;50:23–32. https://doi.org/10.1016/j.jbtep.2015.04.011.
13.• Cox RC, Cole DA, Kramer EL, Olatunji BO. Prospective associations between sleep disturbance and repetitive negative thinking: the mediating roles of focusing and shifting attentional control. Behav Ther. 2017; https://doi.org/10.1016/j.jbtep.2015.04.011. **Subjective sleep disturbance predicts obsessions over 6-months, controlling for baseline obsessions, and this relationship is mediated by focusing attentional control.**
14. Gregory AM, Caspi A, Eley TC, Moffitt TE, O'Connor TG, Poulton R. Prospective longitudinal associations between persistent sleep problems in childhood and anxiety and depression disorders in adulthood. J Abnorm Child Psych. 2005;33:157–63. https://doi.org/10.1007/s10802-005-1824-0.
15. Neckelmann D, Mykletun A, Dahl AA. Chronic insomnia as a risk factor for developing anxiety and depression. Sleep. 2007;30:873–80.
16. Alvaro PK, Roberts RM, Harris JK, Bruni O. The direction of the relationship between symptoms of insomnia and psychiatric disorders in adolescents. J Affect Disorders. 2017;207:167–74. https://doi.org/10.1016/j.jad.2016.08.032.
17. McClung CA. How might circadian rhythms control mood? Let me count the ways. Biol Psychiatry. 2013;74:242–9. https://doi.org/10.1016/j.biopsych.2013.02.019.
18. Chamberlain SR, Blackwell AD, Fineberg NA, Robbins TW, Sahakian BJ. The neuropsychology of obsessive compulsive disorder: the importance of failures in cognitive and behavioural inhibition as candidate endophenotypic markers. Neurosci Biobehav R. 2005;29:399–419. https://doi.org/10.1016/j.neubiorev.2004.11.006.
19. Pauls DL, Abramovitch A, Rauch SL, Geller DA. Obsessive–compulsive disorder: an integrative genetic and neurobiological perspective. Nat Rev Neurosci. 2014;15:410–24. https://doi.org/10.1038/nrn3746.
20. McEwen BS, Karatsoreos IN. Sleep deprivation and circadian disruption: Stress, allostasis, and allostatic load. Sleep Med Clin 2015;10:1–10. doi:https://doi.org/10.1016/j.jsmc.2014.11.007.
21. Ingiosi AM, Opp MR, Krueger JM. Sleep and immune function: glial contributions and consequences of aging. Curr Opin Neurobiol. 2013;23:806–11. https://doi.org/10.1016/j.conb.2013.02.003.
22. Vgontzas AN, Zoumakis E, Bixler EO, Lin HM, Follett H, Kales A, et al. Adverse effects of modest sleep restriction on sleepiness, performance, and inflammatory cytokines. J Clin Endocrinol Metab. 2004;89:2119–26. https://doi.org/10.1210/jc.2003-031562.
23. Frick L, Pittenger C. Microglial dysregulation in OCD, Tourette syndrome, and PANDAS. J Immunol Res. 2016;2016:1–8. https://doi.org/10.1155/2016/8606057.
24. Vargas I, Lopez-Duran N. Investigating the effect of acute sleep deprivation on hypothalamic-pituitary-adrenal-axis response to a psychosocial stressor. Psychoneuroendocrino. 2017;79:1–8. https://doi.org/10.1016/j.psyneuen.2017.01.030.
25. van Lenten SA, Doane LD. Examining multiple sleep behaviors and diurnal salivary cortisol and alpha-amylase: within- and between-person associations. Psychoneuroendocrino. 2016;68:100–10. https://doi.org/10.1016/j.psyneuen.2016.02.017.
26. Gustafsson PE, Gustafsson PA, Ivarsson T, Nelson N. Diurnal cortisol levels and cortisol response in youths with obsessive-compulsive disorder. Neuropsychobiology. 2008;57:14–21. https://doi.org/10.1159/000123117.
27. Wilckens KA, Woo SG, Erickson KI, Wheeler ME. Sleep continuity and total sleep time are associated with task-switching and

preparation in young and older adults. J Sleep Res. 2014;23:508–16. https://doi.org/10.1111/jsr.12148.
28. Drummond SPA, Paulus MP, Tapert SF. Effects of two nights sleep deprivation and two nights recovery sleep on response inhibition. J Sleep Res. 2003;15:261–5. https://doi.org/10.1111/j.1365-2869.2006.00535.x.
29. Chee MWL, Choo WC. Functional imaging of working memory after 24 hr of total sleep deprivation. J Neurosci. 2004;24:4560–7. https://doi.org/10.1523/JNEUROSCI.0007-04.2004.
30. Ma N, Dinges DF, Basner M, Rao H. How acute total sleep loss affects the attending brain: a meta-analysis of neuroimaging studies. Sleep. 2015;38:233–40. https://doi.org/10.5665/sleep.4404.
31. Grunblatt E, Marinova Z, Roth A, Gardini E, Ball J, Geissler J, et al. Combining genetic and epigenetic parameters of the serotonin transporter gene in obsessive-compulsive disorder. J Psychiatr Res. 2018;96:209–17. https://doi.org/10.1016/j.jpsychires.2017.10.010.
32. Yue W, Cheng W, Liu Z, Tang Y, Lu T, Zhang D, et al. Genome-wide DNA methylation analysis in obsessive-compulsive disorder patients. Sci Rep. 2016;6:31333. https://doi.org/10.1038/srep31333.
33. Trivedi MS, Holger D, Bui AT, Craddock TJA, Tartar JL. Short-term sleep deprivation leads to decreased systemic redox metabolites and altered epigenetic status. PLoS One. 2017;12:30181978. https://doi.org/10.1371/journal.pone.0181978.
34. Carroll JE, Irwin MR, Levine M, Seeman TE, Absher D, Assimes T, et al. Epigenetic aging and immune senescence in women with insomnia symptoms: findings from the Women's Health Initiative Study. Biol Psychiatry. 2016;81:136–44. https://doi.org/10.1016/j.biopsych.2016.07.008.