The role of eveningness in obsessive-compulsive symptoms: Cross-sectional and prospective approaches

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ABSTRACT

Background: Eveningness may be defined as the tendency to be most active and alert during the evening. Previous research has linked eveningness with maladaptive psychological outcomes, and recent evidence has highlighted circadian dysregulation as a novel factor in psychopathology, including obsessive-compulsive disorder (OCD). However, limited research has examined the unique relationship between eveningness and OC symptoms, while also considering the role of depression symptoms and sleep-related factors.

Methods: Using a cross-sectional approach, Study 1 examined the association between eveningness and OC symptoms when controlling for depression symptoms. Study 2 then employed a prospective approach to examine the extent to which the relationship between eveningness and change in OC symptoms over 4 months is mediated by change in sleep disturbance and total sleep time when controlling for depression symptoms.

Results: Results indicated that depression better accounts for the cross-sectional association between eveningness and OC symptoms. However, eveningness was found to be a more robust prospective predictor of change in OC symptoms in Study 2. Furthermore, sleep disturbance, but not total sleep time, partially mediated the relationship between eveningness and OC symptoms.


Conclusions: These findings suggest that eveningness may contribute to the development of OC symptoms over time, in part due to its effect on sleep disturbance. Future research examining the role of circadian dysregulation in OCD may uncover novel physiological mechanisms.

Obsessive-compulsive disorder (OCD) is a debilitating disorder characterized by obsessions, or intrusive, distressing cognitions, and compulsions, or repetitive behaviors performed to reduce the distress of the obsessions (American Psychiatric Association, 2013). Although much is known about the nature and structure of OCD (Ruscio et al., 2010), processes that may contribute to the development of OCD remain unclear. Current research approaches suggest that examining the role of arousal and regulatory systems may provide important insight into biopsychological processes that contribute to OCD (Nota et al., 2015). Disruptions in the circadian system, which drives the 24 h cycles of physiological and psychological processes through regulation by the suprachiasmatic nucleus (Czeisler et al., 1999; Weaver, 1998), are thought to be one such regulatory factor that may be relevant to OCD. Indeed, circadian dysregulation has recently been implicated in the development of psychopathology (Charrier et al., 2017; Wulff et al., 2011), including OCD (Lange et al., 2012). Given circadian regulation of important biopsychological processes, including the sleep/wake cycle and hormone secretion (Luik et al., 2015; McClung, 2013), disturbances in these processes among those with OCD may reflect underlying circadian dysregulation.

Evidence for circadian dysregulation in OCD is supported by recent findings that have implicated phase delay, or late sleep onset and offset, in OCD. Among unselected samples, delayed bedtimes are associated with increased OC symptoms (Coles et al., 2012; Schubert and Coles, 2015). Likewise, those with delayed phase sleep disorder (DPSD) report increased OC symptoms compared to healthy controls (Schubert and Coles, 2013). Further, rates of DPSD are elevated among those with severe, treatment resistant OCD (Drummond et al., 2012), and those with comorbid treatment resistant OCD and DPSD report increased OCD symptoms compared to those with treatment resistant OCD without DPSD (Turner et al., 2007). Such links between delayed sleep phase and OCD suggest an underlying circadian shift that may contribute to the development and/or maintenance of OCD.

Accumulating evidence has also implicated sleep disturbance in OCD. For example, those with OCD exhibit alterations in objective sleep parameters compared to healthy controls, including decreased total
Sleep time, increased wake after sleep onset (Alfano and Kim, 2011; Voderholzer et al., 2007), and decreased slow wave, or deep sleep (Kluge et al., 2007a). Objective sleep disturbance is also associated with increased OC symptoms (Robinson et al., 1998). Likewise, those with OCD report increased subjective sleep disturbance compared to healthy controls (Dose et al., 2017; Iverson and Larsson, 2009), and increased sleep disturbance is associated with increased OC symptoms among unselected adults (Raines et al., 2015). Importantly, studies have found a relationship between sleep disturbance and OC symptoms, even when controlling for depression (Cox and Olatunji, 2016a; Timpano et al., 2014; but see also Diaz-Roman et al., 2015).

The sleep disturbance observed in OCD may reflect underlying circadian dysregulation that could be the focus of novel interventions (de Koning et al., 2013). Similarly, alterations in circadian hormones, including cortisol and melatonin, have been linked to OCD (Kluge et al., 2007b; Monteleone et al., 1995; but see also Millet et al., 1998; Catapano et al., 1992). Such abnormalities in circadian hormone regulation in OCD may result from underlying deficits in the circadian system. In addition to circadian hormones, circadian variation may also be indexed by examination of circadian preference, or chronotype. Measures of individual differences in diurnal activity rhythms are thought to reflect the underlying circadian system (Roenneberg et al., 2007) and are associated with other circadian outcomes, including cortisol and body temperature rhythms (Bailey and Heitkemper, 2001), activity rhythms, and sleep (Vitale et al., 2015). Previous research also suggests that chronotype is a product of the intrinsic circadian period, rather than an environmental contributor to it (Duffy et al., 2001). Estimates from twin studies suggest that chronotype is approximately 50% heritable (Koskenvuo et al., 2007), and recent findings from genome wide association studies have identified multiple genes associated with chronotype (Kalmbach et al., 2017). Thus, measures of chronotype offer a low-burden, non-invasive estimate of the individual differences in the circadian system that has a genetic basis.

Chronotype can be measured as a continuous trait that spans from morningness to eveningness, with increased eveningness reflecting a later peak in diurnal activity and energy and delayed sleep/wake schedule (Roenneberg et al., 2007). Previous research suggests that eveningness is linked with a sleep/wake cycle that is “out of sync” with circadian peaks in performance and sleep propensity (Duffy et al., 2001). Thus, eveningness may reflect varying degrees of desynchrony in the circadian system that has been linked to mood variability (Jeong et al., 2015) and decreased positive affect (Hasler et al., 2010). Although the available literature suggests that eveningness may reflect underlying circadian variability that negatively impacts psychological outcomes, the extent to which eveningness may uniquely contribute to OC symptoms is unclear.

The purpose of the present study was to examine the relationship between eveningness and OC symptoms in large samples of community adults. Study 1 examined the cross-sectional relationship between eveningness and OC symptoms. It was hypothesized that eveningness would be associated with increased OC symptoms, even when controlling for depressive symptoms. Notably, the majority of extant research examining circadian rhythms in OCD has utilized cross-sectional designs, precluding the ability to determine the prospective effect of circadian dysregulation on OC symptoms over time. Likewise, no study to date has examined specific mechanisms that may account for the relationship between eveningness and OC symptoms. To address this gap in the literature, Study 2 examined the relationships between eveningness, sleep disturbance, total sleep time, and OC symptoms over 4 months. Given recent evidence that sleep disturbance partially accounts for the relationship between eveningness and negative affect in unselected samples (Simor et al., 2015), it was hypothesized that eveningness would predict increased OC symptoms, and this relationship would be partially mediated by sleep disturbance and total sleep time when controlling for symptoms of depression.

1. Study 1: methods

1.1. Participants

A total of 461 participants enrolled in the study, and 341 participants (81.5% female) completed the survey and comprised the present sample. The mean age of the participants was 33.18 years (SD = 13.49), ranging from 18 to 66 years. The ethnicity composition was as follows: African American (n = 26; 7.6%), Asian (n = 22; 6.5%), Caucasian (n = 272; 80%), Hispanic/Latino (n = 15; 4.4%), Other (n = 5; 1.5%).

1.2. Measures

The Depression, Anxiety, and Stress Scales-Short Form (DASS; Lovibond and Lovibond, 1995) is a 21-item self-report measure of symptoms of depression, anxiety, and stress over the past week. Only the 7-item depression subscale was used in the present analysis. Scores on the DASS short form can be multiplied by 2 to be converted to the full scale (Lovibond and Lovibond, 1995). Items on the DASS are rated on a Likert scale from 0 (did not apply to me at all) to 3 (applied to me very much, or most of the time), and higher scores indicate higher symptoms of stress and depression. A score of 10 or higher suggests severe depression (Lovibond and Lovibond, 1995). The depression subscale demonstrated adequate internal consistency (α = 0.88) in the Study 1 sample.

The Morningness-Eveningness Questionnaire (MEQ; Horne and Ostberg, 1976) is a 19-item self-report measure of chronotype and is thought to reflect the individual’s circadian rhythms. Items on the MEQ are rated on a 4 and 5 point Likert scale with answer options varying by item content. For ease of interpretation, scores on the MEQ were reversed, such that lower scores indicate increased morningness, and higher scores indicate increased eveningness. With the scores reversed, a score of 59 or above indicates an evening type, 42–58 indicates an intermediate type, and 41 or below indicates a morning type. The MEQ demonstrated adequate internal consistency (α = 0.88) in the Study 1 sample.

The Obsessive-Compulsive Inventory-Revised (OCIR; Foa et al., 2002) is an 18-item self-report measure of OC symptoms in the past month. The OCIR consists of 6 subscales measuring specific categories of OC symptoms (washing, checking, ordering, neutralizing, hoarding, obsessing). Items on the OCIR are rated on a Likert scale from 0 (not at all) to 4 (extremely), and higher scores indicated increased OC symptom severity. A score of 21 or higher suggests clinically significant OC symptoms. The OCIR demonstrated good internal consistency (α = 0.92) in the Study 1 sample.

1.3. Procedure

Participants were recruited through ResearchMatch, a national health volunteer registry that was 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 for which

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1 Note that there is variability in the literature regarding the term chronotype (Roenneberg et al., 2007). Here, chronotype refers to the morningness-eveningness continuum, consistent with recent genetic studies (e.g., Koskenvuo et al., 2007).

2 Due to an error in survey development for Study 1, data was not obtained for one item of the DASS depression subscale (item 2, “I felt that I had nothing to look forward to”). The depression subscale was retained given that the remaining items were depression-relevant and evidenced adequate internal consistency. This error was corrected prior to Study 2.
they may be eligible. Healthy (no reported conditions) ResearchMatch volunteers who lived within 15 miles of Vanderbilt University, or in 11 randomly selected states (Arkansas, Connecticut, Idaho, Illinois, Kansas, New Hampshire, New Jersey, North Carolina, North Dakota, Oklahoma, or South Dakota) were recruited. Vanderbilt University undergraduates in psychology courses were also recruited and received course credit for participating. Participants received a link to complete an online battery of questionnaires and were informed that they would have the opportunity to enter their name into a drawing for a $100 gift card. 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 individual participants included in the study.

1.4. Data analytic approach

Data analysis was employed with SPSS 23. A hierarchical multiple regression analysis was conducted to examine the relationship between eveningness and OC symptoms, controlling for depression symptoms. Depression symptoms were entered in step 1, and eveningness was entered in step 2 of the model:

\[ Y_1 = \beta_0 + \beta_1X_{ij} + \epsilon_i \]  
\[ Y_2 = \beta_0 + \beta_1X_{ij} + \beta_2X_{ij} + \epsilon_i \]  

Listwise deletion was utilized for missing data.

2. Study 1: results

2.1. Associations between study variables

Means and standard deviations for study variables are shown in Table 1. There were small, significant associations between eveningness and OC symptoms and eveningness and depression symptoms and a moderate, significant association between depression symptoms and OC symptoms.

2.2. Multiple regression

All regression coefficients are reported as unstandardized values. As shown in Table 2, depression symptoms significantly contributed to the model, \( F(1,340) = 68.78, p < .001 \) and accounted for 17% of the variance in OCD symptoms. Introducing eveningness to the model explained an additional 0.2% of the variance in OCD symptoms, and the \( R^2 \) change was not significant (\( p = .35 \)). When both depression symptoms and eveningness were included in the model, depression symptoms were significantly associated with OC symptoms (\( B = 1.25, \alpha = .16, \beta = .41, p < .001 \)). Similarly, there was a significant difference in baseline eveningness between those who completed the study \( M = 45.93 \) and those who did not \( M = 39.85 \), \( t(1576) = -8.28, p < .001 \). There was also a significant difference in baseline OC symptoms between those who completed the study \( M = 47.94 \) and those who did not \( M = 51.07 \), \( t(1264) = -4.70, p < .01 \). Finally, there was a significant difference in baseline OC symptoms between those who completed the study \( M = 10.40 \) and those who did not \( M = 13.07 \), \( r(1435) = 4.59, p < .01 \).

2.3. Discussion

Results of Study 1 indicated eveningness was correlated with increased OC symptoms. This finding is consistent with extant research linking eveningness to symptoms of psychopathology, including OCD, in cross-sectional samples (Alvaro et al., 2014). Contrary to prediction, depression was associated with OC symptoms over and above the effect of eveningness. This finding suggests that depression symptoms may be a better indicator of OC symptoms in cross-sectional samples and highlights the importance of accounting for depression when examining circadian correlates of OCD. Importantly, cross-sectional models of hypothesized causal relationships can be misleading (Cole and Maxwell, 2003). Accordingly, Study 2 examined the relationship between eveningness and change in OC symptoms over time to more thoroughly assess the role of circadian abnormalities in OC symptoms.

3. Study 2: methods

3.1. Participants

A total of 1613 participants enrolled in the study. 1,262 participants had complete data at Time 1, 671 participants had complete data at Time 2, and 578 participants had complete data at Time 3. 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 excluded from analysis. One participant was excluded for an out of range response to the Pittsburgh Sleep Quality Index total sleep time item (see Measures). A total of 382 participants (86.1% female) who met validity criteria and had complete data at all 3 time points were included for final analysis.

The mean age of the participants was 46.07 years (\( SD = 13.46 \)), ranging from 18 to 66 years. The ethnicity composition was as follows: Caucasian \( (n = 345; 90.3\%) \), African American \( (n = 14; 3.7\%) \), Asian \( (n = 4; 1\%) \), Hispanic/Latino \( (n = 9; 2.4\%) \), Other \( (n = 7; 1.8\%) \). Participants who did not complete the study did not differ from those who did complete the study on gender or race. However, there was a significant difference in age between those who completed the study \( M = 45.93 \) and those who did not \( M = 39.85 \), \( t(1576) = -8.28, p < .01 \). Similarly, there was a significant difference in baseline eveningness between those who completed the study \( M = 47.94 \) and those who did not \( M = 51.07 \), \( t(1264) = -4.70, p < .01 \). Finally, there was a significant difference in baseline OC symptoms between those who completed the study \( M = 10.40 \) and those who did not \( M = 13.07 \), \( r(1435) = 4.59, p < .01 \).

3.2. Measures

The DASS depression subscale (Lovibond and Lovibond, 1995) as described in Study 1 was utilized in Study 2 and demonstrated good internal consistency at time 1 (\( \alpha = .93 \)) in the Study 2 sample.

### Table 1

<table>
<thead>
<tr>
<th>Measure</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. MEQ</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>2. DASS</td>
<td>.28*</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>3. OCIR</td>
<td>.16*</td>
<td>.41*</td>
<td>–</td>
</tr>
<tr>
<td>M</td>
<td>50.31</td>
<td>3.19</td>
<td>11.28</td>
</tr>
<tr>
<td>SD</td>
<td>11.22</td>
<td>3.66</td>
<td>11.15</td>
</tr>
<tr>
<td>Range</td>
<td>23-78</td>
<td>0-17</td>
<td>0-54</td>
</tr>
</tbody>
</table>

Note. MEQ = Morningness-Eveningness Questionnaire; DASS = Depression, Anxiety, and Stress Scale, Depression Subscale; OCIR = Obsessive-Compulsive Inventory-Revised.

*p < .01
Table 3
Descriptive statistics and correlations for study measures (n = 382).

<table>
<thead>
<tr>
<th>Measure</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
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<tbody>
<tr>
<td>MEQ.T1</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>ISI.T1</td>
<td>.08</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>TST.T1</td>
<td>.05</td>
<td>–0.48**</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>DASS.T1</td>
<td>.17**</td>
<td>.44**</td>
<td>–0.13*</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>OCIR.T1</td>
<td>.12*</td>
<td>.31**</td>
<td>–0.10</td>
<td>.40**</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>ISI.T2</td>
<td>.17**</td>
<td>.78**</td>
<td>–0.38**</td>
<td>.42**</td>
<td>.31**</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>TST.T2</td>
<td>.004</td>
<td>–0.42**</td>
<td>.68**</td>
<td>–0.20**</td>
<td>–0.12*</td>
<td>–0.48**</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>OCIR.T3</td>
<td>.19**</td>
<td>.24**</td>
<td>–0.09</td>
<td>.28**</td>
<td>.74**</td>
<td>.36**</td>
<td>–0.12*</td>
<td>–</td>
</tr>
<tr>
<td>M</td>
<td>47.65</td>
<td>11.14</td>
<td>6.50</td>
<td>4.71</td>
<td>10.43</td>
<td>9.80</td>
<td>6.60</td>
<td>8.56</td>
</tr>
<tr>
<td>SD</td>
<td>11.29</td>
<td>6.11</td>
<td>1.31</td>
<td>5.06</td>
<td>8.87</td>
<td>6.20</td>
<td>1.23</td>
<td>8.67</td>
</tr>
<tr>
<td>Range</td>
<td>18-78</td>
<td>0-28</td>
<td>3-12</td>
<td>0-21</td>
<td>0-51</td>
<td>0-28</td>
<td>3-10</td>
<td>0-59</td>
</tr>
</tbody>
</table>

Note. MEQ.T1 = Morningness-Eveningness Questionnaire, Time 1; ISI.T1 = Insomnia Severity Index, Time 1; TST.T1 = Pittsburgh Sleep Quality Index, Total sleep time, Time 1; DASS.T1 = Depression, Anxiety, and Stress Scale, Depression Subscale, Time 1; OCIR.T1 = Obsessive-Compulsive Inventory-Revised, Time 1; ISI.T2 = Insomnia Severity Index, Time 2; TST.T2 = Pittsburgh Sleep Quality Index, Total sleep time, Time 2; OCIR.T3 = Obsessive-Compulsive Inventory-Revised, Time 3.

*p < .05
**p < .01

The MEQ (Horne and Ostberg, 1976) as described in Study 1 was utilized in Study 2 and demonstrated good internal consistency at time 1 (α = 0.88) in the Study 2 sample.

The OCIR (Foa et al., 2001) as described in Study 1 was utilized in Study 2 and demonstrated adequate and good internal consistency at times 1 and 3 (α = 0.88 and α = 0.90, respectively) in the Study 2 sample.

The Lie Scale demonstrated marginal internal consistency at time 1 (α = 0.67) in the present sample.

The Insomnia Severity Index (ISI; Bastien et al., 2001) is a 7-item self-report measure of insomnia symptoms over the past two weeks and is used to detect cases of insomnia and assess treatment response. Items on the ISI are rated on a Likert scale from 0 (none) to 4 (very severe), and higher scores indicate a higher severity of insomnia. A score of 15 or higher suggests clinically significant insomnia. The ISI demonstrated good internal consistency at time 2 (α = 0.90) in the Study 2 sample.

The Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989) is a 19-item self-report measure of sleep quality and sleep disturbance over the past month. Items include reports of sleep variables such as typical number of hours slept each night and typical bedtime, as well as rating sleep problems on a Likert scale from 0 (not during the past month) to 3 (three or more times a week). Only the total sleep time item was used in the present study. Total sleep time was reported in hours.

3.3. Procedure

Participants were recruited through ResearchMatch as described in Study 1. Participants received a link to complete an online battery of questionnaires and were informed that they would have the opportunity to enter their name into a drawing for a $50 gift card after the completion of each survey. Participants received a total of three identical survey batteries over a four-month period. Participants who completed the first survey (time 1) received the second and third surveys two (time 2) and four months (time 3), respectively, after completion of the first survey. Study data were collected and managed using REDCap (Harris et al., 2009) as described in Study 1. 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 individual participants included in the study.

3.4. Data analytic strategy

Data analysis was employed with SPSS 23. A multivariate mediation analysis was conducted to assess sleep disturbance and total sleep time at time 2 (at 2 months) as parallel mediators of the relationship between eveningness at time 1 and change in OC symptoms at time 3 (at 4 months). Depression and OC symptoms and the sleep mediators at time 1 were included in the model as covariates:

\[ M_1 = \beta_0 + \beta_1 X + \beta_2 C_1 + \beta_3 C_2 + \beta_4 C_3 + \beta_5 C_4 + \epsilon_M \]

\[ M_2 = \beta_0 + \beta_1 X + \beta_2 C_1 + \beta_3 C_2 + \beta_4 C_3 + \beta_5 C_4 + \epsilon_M \]

\[ Y = \beta_1 + \beta_2 X + \beta_3 M_1 + \beta_4 M_2 + \beta_5 C_1 + \beta_6 C_2 + \beta_7 C_3 + \beta_8 C_4 + \epsilon_Y \]

Mediation analyses were conducted with the PROCESS macro, and bias-corrected bootstrap confidence intervals were calculated to test the significance of the indirect effects (Hayes, 2013). Consistent with current approaches to mediation, significance of the indirect effects was examined regardless of the significance of the total effect (Hayes and Rookwood, 2017). This approach is supported by considerable research arguing that the requirement of a significant total effect can be misleading, as tests of the total effect are often underpowered relative to tests of indirect effects and are vulnerable to suppression effects when indirect effects are of opposite sign (Mackinnon et al., 2000; Rucker et al., 2011). Further, only the indirect effect (a*b) tests the mediating effect, or the degree to which a change in X results in a change in Y through the effect of X on M; thus, requiring a significant total effect utilizes more inferential tests than are necessary to support a claim (Hayes and Rookwood, 2017). 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. If more than one item was missing on a given measure, that measure was considered missing. During data analysis, consistent with the PROCESS macro (Hayes, 2013), listwise deletion was utilized for missing measures.

4. Study 2: results

4.1. Associations between study variables

Means and standard deviations for study variables are shown in Table 3. The majority of study variables were significantly correlated, \( p < .01 \), with the exceptions of total sleep time at time 1 and eveningness at time 1 and OC symptoms at time 1 and 2, and total sleep time at time 2 and eveningness at time 1.
Table 4
Unstandardized model coefficients for the hypothesized model predicting OCD symptoms controlling for depression symptoms (n = 382).

<table>
<thead>
<tr>
<th>Predictor</th>
<th>M1 (ISI.T2)</th>
<th>M2 (TST.T2)</th>
<th>Y (OCIR.T3)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Coef</td>
<td>SE</td>
<td>P</td>
</tr>
<tr>
<td>X (MEQ.T1)</td>
<td>a</td>
<td>0.057</td>
<td>0.018</td>
</tr>
<tr>
<td>M1 (ISI.T2)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M2 (TST.T2)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cov (OCIR.T1)</td>
<td></td>
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<tr>
<td>Cov (DASS.T1)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cov (ISI.T1)</td>
<td></td>
<td></td>
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<tr>
<td>Cov (TST.T1)</td>
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</tr>
<tr>
<td>Constant</td>
<td></td>
<td>-0.546</td>
<td>1.38</td>
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<tr>
<td></td>
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</tr>
<tr>
<td></td>
<td>R² = 0.623</td>
<td></td>
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</tr>
<tr>
<td></td>
<td>F(5,376) = 124.182</td>
<td></td>
<td></td>
</tr>
<tr>
<td>p</td>
<td>&lt; 0.001</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. MEQ.T1 = Morningness-Eveningness Questionnaire, Time 1; ISI.T1 = Insomnia Severity Index, Time 1; TST.T2 = Pittsburgh Sleep Quality Index, Total sleep time, Time 1; DASS.T1 = Depression, Anxiety, and Stress Scale, Depression Subscale, Time 1; OCIR.T1 = Obsessive-Compulsive Inventory-Revised, Time 1; ISI.T2 = Insomnia Severity Index, Time 2; TST.T2 = Pittsburgh Sleep Quality Index, Total sleep time Component Score, Time 2; OCIR.T3 = Obsessive-Compulsive Inventory-Revised, Time 3.

4.2. Mediation

All regression coefficients are reported as unstandardized values. As shown in Table 4 and Fig. 1, results of a mediation analysis using ordinary least squares regression revealed that eveningness at time 1 predicted change in OC symptoms at time 3 through its effect on preceeding change in sleep disturbance at time 2, but not through change in total sleep time at time 2. Eveningness at time 1 significantly predicted increased sleep disturbance (a = 0.057) at time 2, and increased sleep disturbance at time 2 significantly predicted increased OC symptoms at time 3 (0.473). In contrast, eveningness at time 1 did not predict total sleep time at time 2, and total sleep time at time 2 did not predict OC symptoms at time 3.

A 95% bias-corrected bootstrap confidence interval for the total indirect effect of sleep disturbance and total sleep time at time 2 (ab = 0.027) based on 10,000 bootstrap samples did not include zero (0.010 to 0.056), indicating a significant indirect effect of eveningness at time 1 on change in OC symptoms at time 3 through change in sleep disturbance at time 2. Effect size calculations estimating the ratio of the indirect effect to the total effect (Alwin and Hauser, 1975) indicate that 32% of the effect of eveningness at time 1 on change in OC symptoms at time 3 occurs through the indirect effect of change in sleep disturbance at time 2. In contrast, the 95% bias-corrected bootstrap confidence interval for the specific indirect effect total sleep time at time 2 (ab2 = -0.0002) based on 10,000 bootstrap samples did include zero (-0.006 to 0.003), indicating no significant indirect effect of eveningness at time 1 on change in OC symptoms at time 3 through total sleep time at time 2.

With sleep disturbance and total sleep time at time 2 included in the model, there remained a significant direct effect of eveningness at time 1 on change in OC symptoms at time 3 (c' = -0.057, p < .05). Notably, in the total effect model including depression and OC symptoms and sleep mediators at time 1 as covariates, eveningness at time 1
significantly predicted increased OC symptoms at time 3 (c = 0.084, p < .01), but depression symptoms at time 1 did not (p > .05).

4.3. Discussion

Results of Study 2 found that eveningness predicted increased OC symptoms over 4 months, even when controlling for depression symptoms. This finding is consistent with previous evidence for a role of circadian dysregulation in OCD (Lange et al., 2012; Nota et al., 2015) and extends these findings by indicating a prospective relationship between circadian variability and OC symptoms over and above the effect of depression symptoms. Furthermore, the relationship between eveningness and change in OC symptoms was mediated by preceding change in sleep disturbance, but not total sleep time. Consistent with previous research highlighting the role of sleep disturbance in OCD (Lange and Olatunji, 2016a; Cox and Olatunji, 2016b), this finding suggests that underlying variation in the circadian system has downstream effects on sleep, which in turn leads to increased OC symptoms.

4.4. General discussion

The present study examined the relationship between eveningness and OC symptoms and potential mediating processes. Results indicated that depression better accounts for the cross-sectional association between eveningness and OC symptoms. However, eveningness was found to be a more robust prospective predictor of change in OC symptoms in Study 2. Results of Study 2 also indicated that eveningness predicted increased sleep disturbance, but not total sleep time over 2 months. These findings replicate previous research indicating a divergence in the relationships between eveningness, sleep quality, and total sleep time, such that eveningness is linked to decreased in sleep quality (Vollmer et al., 2017), but is largely independent of habitual sleep duration (Merikanto et al., 2012; Roenneberg et al., 2007). The present findings suggest that eveningness does not prospectively predict habitual total sleep time, likely as a result of the increased variability of sleep duration on week nights and weekends commonly observed in evening types (Roenneberg et al., 2007). In contrast, the evidence for a prospective link between eveningness and sleep disturbance suggests that overlap between these processes may reflect an underlying genotype (Barclay et al., 2010), and sleep disturbance is further propagated by consistent mismatch between sleep timing and internal sleep drive (Duffy et al., 2001). This relationship suggests that underlying abnormalities in the circadian system may have a cascade effect, such that processes under circadian regulation, such as sleep, are disrupted by circadian dysregulation. Further, this finding suggests that eveningness may be a useful measure of variation in the circadian system that has downstream effects on subsequent circadian-regulated variables. Alternatively, the link between eveningness and sleep disturbance may reflect a mismatch between the biological clock and environmental demands (Minges and Redeker, 2016).

The finding that sleep disturbance predicts increased OC symptoms over 2 months is consistent with previous research linking subjective sleep quality to OC symptoms (Cox and Olatunji, 2016a; Timpano et al., 2014) and extends these findings by showing a prospective relationship between sleep disturbance and OC symptoms. In contrast, total sleep time did not significantly predict OC symptoms over 2 months. Of note is that previous research has found decreased objective total sleep time in those with OCD compared to healthy controls (Alfano and Kim, 2011; Voderholzer et al., 2007). Although few studies have examined specific subjective sleep parameters in OCD, one study found no differences in subjective total sleep time between those with OCD and healthy controls (Bobdey et al., 2002). These discrepancies between objective and subjective total sleep time suggest that subjective estimates of sleep duration may not capture OCD-related outcomes. Alternatively, objective sleep measures, such as actigraphy and polysomnography, sample nightly sleep parameters, while commonly used subjective sleep measures, such as the PSQI, capture retrospective habitual sleep variables (Ancoli-Israel et al., 2003; Buysse et al., 1989). Thus, discrepancies in the relationship of objective and subjective total sleep time to OC symptoms may reflect differences in the sampling period.

Results of Study 2 also showed that eveningness predicted increased OC symptoms over 4 months when controlling for baseline OC and depression symptoms. This finding is consistent with previous research suggesting that circadian dysregulation may contribute to OCD (Lange et al., 2012). This finding does contradict a recent study indicating no prospective association between chronotype and OC symptoms in a sample of adolescents (Alvaro et al., 2017). Notably, phase delay and eveningness are common among adolescents (Carskadon et al., 1999), which may mask the signal for a relationship between eveningness and OCD among this population. Given that chronotype is normally distributed in the adult population with morningness increasing with age (Roenneberg et al., 2007), one possibility is that adolescents who do not experience a phase advance to a more intermediate chronotype as they transition to adulthood may be at increased risk for developing OCD. Additional research is necessary to further examine the relationship between eveningness and OCD in the transition from adolescence to adulthood.

Study 2 also indicated that the relationship between eveningness and change in OC symptoms over 4 months was partially mediated by preceding change in sleep disturbance, but not total sleep time. This finding is consistent with recent research showing that sleep disturbance partially accounts for the relationship between eveningness and mood symptoms (Bakotic et al., 2017; Simor et al., 2015). To our knowledge, this is the first study to indicate a prospective relationship between eveningness and change in OC symptoms that is partially accounted for by changes in sleep disturbance. These findings suggest that the downstream effects of circadian dysregulation, such as sleep disturbance, lead to increased symptoms of OCD. However, additional research is necessary to replicate these findings over various time lags and in clinical samples.

A notable finding from both studies concerns the role of depression symptoms. While Study 1 found that depression symptoms accounted for significantly more variance in OC symptoms than eveningness in a cross-sectional sample, eveningness predicted change in OC symptoms over 4 months in Study 2, even with baseline depression included in the model. This result suggests that eveningness may be a more robust predictor of increases in OC symptoms over time than depression symptoms. Furthermore, mood symptoms may be a strong epiphenomenon of concurrent OC symptoms that masks the role of other processes. Given that eveningness outperformed depression symptoms as a prospective, but not cross-sectional, predictor of change in OC symptoms, the remaining question is one of mechanism. The results of Study 2 suggest that part of the reliability of the prospective signal of eveningness is due to interim sleep disturbance. However, consistent with previous research (Simor et al., 2015), a large portion of the effect of eveningness on change in OC symptoms remains unaccounted for, suggesting the presence of unmeasured intervening variables. Other dysregulated circadian processes, such as abnormal endocrine function, may work synergistically with sleep disturbance to more fully explain why eveningness contributes to the development of OC symptoms. Future research sampling multiple circadian processes is necessary to fully characterize the role of circadian abnormalities in OC symptoms.

The present findings add to a small, but growing body of research highlighting the role of circadian dysregulation in OCD (Lange et al., 2012; Wulff et al., 2011). In conjunction with extant research, the results of the present studies suggest that OCD prevention and intervention efforts may benefit from the consideration of circadian rhythms. Indeed, previous research suggests that circadian-related treatments are efficacious for multiple disorders, such as interpersonal and social rhythm therapy for bipolar disorder (Frank et al., 1994), cognitive behavior therapy for insomnia for depression (Manber et al., 2011), and light therapy for attention-deficit/hyperactivity disorder...
Together these findings suggest that treatments targeting circadian irregularities may have transdiagnostic utility. These studies offer the first evidence of evenness as a potential marker for circadian abnormalities in OC symptoms. However, these findings must be considered in the context of the study limitations. First, both studies utilized unselected samples, which limits generalizability to clinical samples. However, 21% and 15% of the samples for Study 1 and 2, respectively, were above threshold for clinically significant OC symptoms (Foa et al., 2002), and 9% and 17% of the samples for Study 1 and 2, respectively, were above threshold for clinically significant depression. Second, participants who did not complete the study reported higher baseline evenness and OC symptoms compared to those who did complete the study; however, this may suggest that the present model is able to detect even small increases in OC symptoms among those with less extreme levels of evenness. Third, both studies relied on self-report data. It is possible that subjective estimates of circadian variables may not perfectly reflect the underlying physiology. Fourth, given the single-method approach, the effects of shared method variance cannot be ruled out (Cole and Maxwell, 2003). Future research utilizing multimethod approaches is necessary to replicate these results. Fifth, research utilizing cross-lagged analyses is necessary to identify potential bidirectional relationships between these processes. Sixth, the relationships between study variables may be different over longer periods of time, particularly given findings suggesting that chronotype may change over time (Vedaa et al., 2013). Seventh, the TST item on the PSQI precludes the ability to distinguish between weekend and weekday TST, which are often disparate (Lauderdale, 2014), and the present model may vary as a function of day of the week. Finally, the absence of experimental manipulation of circadian processes precludes causal interpretation. Experimental paradigms intended to isolate circadian rhythms, such as forced desynchrony, may hold promise for this purpose. Despite these limitations, the present studies offer the first evidence for a role of evenness in explaining increases in OC symptoms through its effect on sleep disturbance using self-report measures. Future experimental research along these lines may offer important insights into deficits in arousal and regulatory systems that may contribute to the development of OCD.

Conflict of interest

The authors declare that they have no conflict of interest.

Compliance with ethical standards

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by any of the authors. This research did not receive any specific grant from funding agencies in the public, commercial, or non-profit sectors.

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