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Research paper

Attention-deficit hyperactivity disorder symptoms add risk to circadian rhythm sleep problems in depression and anxiety

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ABSTRACT

Background: Comorbid ADHD symptoms may partly account for circadian rhythm disturbances in depression and anxiety disorders.
Methods: Self-reported sleep characteristics of 2090 participants in the Netherlands Study of Depression and Anxiety were assessed using the Munich Chronotype Questionnaire. We defined 3 groups: healthy controls (HC), persons with lifetime depression and/or anxiety disorders (LDA), and those with both LDA and high ADHD symptoms (LDA+ADHD), using the Conner's Adult ADHD Rating Scale.
Results: Sleep characteristics were least favorable in the LDA+ADHD group. Important group differences between LDA+ADHD, LDA and HC were found for extremely late chronotype (12% vs. 5% vs. 3%; $p < .001$), sleep duration < 6 h (15% vs. 5% vs. 4%; $p < .001$), and for an indication of the Delayed Sleep Phase Syndrome (DSPS; 16% vs. 8% vs. 5%; $p < .001$). After adjustment for covariates, including depression and anxiety, presence of ADHD symptoms increased the odds ratio for late chronotype (OR=2.6; $p=.003$), indication of DSPS (OR=2.4; $p=.002$), and sleep duration < 6 h (OR=2.7; $p=.007$).
Limitations: ADHD conceptually overlaps with symptom presentation of depression and anxiety. We used a cross-sectional study design, and used self reported sleep characteristics.
Conclusions: High ADHD symptoms were associated with an increased rate of circadian rhythm sleep disturbances in an already at-risk population of people with depression and/or anxiety disorders. Circadian rhythm sleep disorders, as often seen in ADHD are not entirely due to any comorbid depression and/or anxiety disorder. Adequate treatment of such sleep problems is needed and may prevent serious health conditions in the long term.

1. Introduction

Sleep problems are highly prevalent and impairing in depression (Tsuno et al., 2005) and anxiety disorders (Mellman, 2006; Papadimitriou and Linkowski, 2005; van Mill et al., 2010). These patients have reported both shorter and longer sleep durations, and trouble falling asleep (Alfano et al., 2013; Breslau et al., 1996; Mellman, 2006). Also, sleep problems constitute independent risk factors for developing a new episode of depression or anxiety in those who had previously recovered (Ohayon, 2002). Similarly, sleep problems frequently occur in attention-deficit hyperactivity disorder (ADHD) (Baird et al., 2012; Kooij and Bijlenga, 2013; Schredl et al., 2007; Van der Heijden et al., 2005a), and severity of sleep problems relates to ADHD severity (Bijlenga et al., 2013a; Gau et al., 2007; Mahajan et al., 2010). Moreover, depression, anxiety disorders, and ADHD are often comorbid (Biederman et al., 1993; Kessler, 2007; Lamers et al., 2011).

Circadian rhythm disturbances may contribute to the development or severity of mental disorders, such as depression, anxiety disorders, and ADHD (Baird et al., 2012; Dueck et al., 2012; van Veen et al., 2010). One of the most prominent circadian rhythm sleep disorders in ADHD is the delayed sleep phase syndrome (DSPS). DSPS is marked by a phase delay in melatonin secretion (Bijlenga et al., 2013b; van Veen et al., 2010), and is characterized by chronic late sleep, late rising, and the inability to fall asleep or to wake up at earlier times (APA, 2000). Those with DSPS are often referred to as having extremely late chronotypes, which is the term for one's biologically driven preference for timing of sleep and wake (Roenneberg et al., 2003).

The prevalence of DSPS in the general population is estimated

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at 0.2–3.1% (Hazama et al., 2008; Schrader et al., 1993; Yazaki et al., 1999), but it is much more common in psychiatric disorders (Dagan, 2002). Amongst DSPS patients, there are high rates of depression (16%) (Kamei et al., 1998), social phobia (18%), and panic disorder (8%) (Yamadera et al., 1998). The rate of ADHD in patients with DSPS is yet unknown. It has been suggested that psychological features of depression, such as social withdrawal, may induce a loss of social cues that normally help to synchronize the circadian rhythm (Shirayama et al., 2003). Also in other studies, late chronotype has been related to depressive mood (Chelminski et al., 1999; Hidalgo et al., 2009; Kitamura et al., 2010; Merikanto et al., 2013), higher levels of depressive symptoms (Selvi et al., 2007), and increased number of ADHD symptoms (Bijlenga et al., 2013a). Of adult ADHD patients, 26–78% has DSPS (Bijlenga et al., 2013a; van Veen et al., 2010). A delayed sleep pattern results in short sleep duration when there are early morning obligations such as work or school (Lack and Wright, 2007). Short sleep on a chronic basis leads to exhaustion, and has been identified as a risk factor for various serious health conditions, such as obesity, diabetes, cardiovascular disease, increased inflammation, and cancer (Knutson, 2010; Maury et al., 2010; Pinheiro et al., 2006).

We assessed whether ADHD symptoms add risk to developing circadian rhythm sleep disturbances, in those with or without depression and anxiety. We used data from a large Dutch cohort. If ADHD independently increases the risk for circadian rhythm sleep disturbances, it seems important that clinicians become more aware of both delayed sleep and its consequences, as well as possible ADHD, when encountering sleep disturbances in depression and anxiety disorders.

2. Methods

2.1. Participants

We analyzed data from the Netherlands Study of Depression and Anxiety (NESDA) in which sleep and circadian rhythm disturbances were assessed. The NESDA study is a longitudinal cohort study designed to investigate the long-term course and consequences of depressive and anxiety disorders in individuals aged 18–65 years old. NESDA included 2981 participants at baseline, and consisted of healthy controls, persons with a remitted or current depressive and/or anxiety disorder. A detailed description of the study can be found elsewhere (Penninx et al., 2008). The research protocol was approved by the Ethical Committee of participating universities and written informed consent was obtained from all participants. Circadian rhythm information was collected at the 2-year follow-up assessment, which was considered the baseline for the present analyses (n=2327). ADHD symptoms were measured in 2092 of these subjects at the 4-year assessment, of which we excluded two participants (1%) who had clinical ADHD symptoms without having lifetime depression and/or anxiety disorders, leading to the ultimate sample size of 2090 for the present analyses. The low prevalence rate of clinical ADHD symptoms among healthy controls (1–3%), compared to the international prevalence rate of 3–6% may be explained by the exclusion of psychiatric comorbidities during the initial sampling for the control group in NESDA, decreasing the chance of including people with ADHD in the control group. Several studies have indicated that 66–78% of adult ADHD patients has at least one comorbid psychiatric disorder, and in clinical practice the mean number of psychiatric comorbidities is three (Biederman et al., 1993; Kessler et al., 2006; Kooij et al., 2010). Since ADHD is a childhood-onset, chronic disorder (APA, 2000), ADHD was assumed to be present throughout the NESDA study, which allowed for the use of circadian rhythm information at the 2-year follow-up, and the ADHD information at the 4-year follow-up assessments.

2.2. Circadian rhythm sleep outcomes

Sleep outcomes were assessed using the 11-item Munich Chronotype Questionnaire (MCTQ) (Roenneberg et al., 2007). The MCTQ is a self-report measure consisting of questions on bedtime, wake-up time, midsleep, sleep-onset latency, and sleep duration on nights before work and free days, and chronotype in childhood and adulthood. Following Roenneberg et al., (2007), sleep outcomes on free days were considered the most accurate reflection of the intrinsic circadian rhythm, because subjects are then less influenced by alarm clocks or work schedules. Sleep outcomes on work days are regarded as the extrinsically imposed rhythm, since (very) late chronotypes often report having a long sleep-onset latency (Morin and Espie, 2003) and chronic short sleep duration (Roenneberg et al., 2007). In this case, work schedules do not match the intrinsic circadian rhythm. The main circadian rhythm sleep outcomes were late chronotype, and an indication of DSPS (which both are indicators of the underlying circadian disturbance), and short sleep duration on work days (which is one of the consequences of a delayed sleep). We used the time of midsleep on free days, corrected for sleep debt on work days (MSFsc) as a continuous parameter for chronotype (Roenneberg et al., 2007), and used the self-reported chronotype in adulthood as a categorical parameter of chronotype (Gibertini et al., 1999). MSFsc was calculated with the following formula: $MSFsc = MSF - 0.5^{*}(SDF-[5^{*}SDW+2^{*}SDF]/7)$, in which MSF is the midpoint of sleep on free days, SDF is the sleep duration on free days, and SDW is the sleep duration on work days. The indication for DSPS was defined as the inability to fall asleep at a preferred bed time before 00:30 h, and having a sleep-onset latency of 30 min or more on work days, or a self-rating of being an extremely late chronotype in childhood or adulthood, as reported on the 7-point chronotype classification of the MCTQ (Kooij and Bijlenga, 2013). Short sleep was defined as a sleep duration shorter than 6 h per night.

2.3. ADHD symptoms

ADHD symptoms were assessed at the 4-year follow-up of NESDA using the Conners' Adult ADHD Rating Scale – Screening Version (CAARS-S: SV) (Conners et al., 1999), a 30-item self-report questionnaire addressing the presence of DSM-IV criteria for ADHD symptoms. The CAARS contains two subscales: the 9-item 'Inattentive symptoms' (range 0–27) and the 9-item 'Hyperactivity/Impulsivity symptoms' (range 0–27), and also the 12-item 'ADHD index' (range 0–36). The ADHD index identifies those at risk for ADHD, by additionally scoring ADHD characteristic behaviors such as sensation-seeking, extraversion, distractibility, low self-esteem, and mood swings. In order to identify high scores per subscale, the raw scores on subscales were converted into standardized scores (*T*-scores) using American age- and gender normative data, following the CAARS manual (Conners et al., 1999). *T*-scores of 65 or above were above the clinical relevant cut-off, and were defined as 'high ADHD symptoms'. We used the dichotomous 'high ADHD score' to determine any relationship between ADHD and circadian rhythm sleep problems. Then, we used the continuous scores to examine if circadian rhythm sleep problems were related to specific symptom domains of ADHD. Lastly, the continuous ADHD index score was used in order to investigate any dose-response relationship between ADHD and circadian rhythm sleep problems. The CAARS has good test-retest reliability ($r=0.89–0.95$) and has high discriminant validity for the ADHD index (73% sensitivity). Although the Dutch version of the CAARS has not been validated yet, a high model-fit was found between German and American norm data (Christiansen et al., 2011), justifying the CAARS as a reliable and cross-culturally valid measure of ADHD symptoms in adults (Christiansen et al., 2012).