Psychiatry Research 268 (2018) 279–287

Contents lists available at ScienceDirect

Psychiatry Research

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

Sleep profiles and CBT-I response in schizophrenia and related psychoses

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ARTICLE INFO

Keywords:
Sleep profile
Insomnia
Hypersomnia
Cognitive behavioural therapy
Schizophrenia
Psychosis
Sleep duration

ABSTRACT

This study investigated sleep subtypes in schizophrenia, and their response to Cognitive Behavioural Therapy for Insomnia (CBT-I) treatment. Sleep profiling was conducted using latent class analysis on baseline Pittsburgh Sleep Quality Index data ($N = 74$ outpatients with schizophrenia who were poor sleepers, 52% male, mean age = 41.4 years). Of these, 40 took part in CBT-I treatment. Analyses revealed three sleep subtypes based on total sleep time (TST), sleep efficiency (SE), and sleep onset latency (SOL) parameters: Cluster 1 ('classic severe insomnia', 44.6%), Cluster 2 ('insomnia with normal sleep duration', 37.8%), and Cluster 3 ('insomnia with hypersomnia', 17.6%). Gains analysis of pre- and post-treatment data from CBT-I participants revealed improvements in sleep and psychopathology in all three clusters, although there were some group differences in the areas and magnitude of improvement. Cluster 1 showed the greatest benefits with longer TST and improved SE. Cluster 2 showed a comparatively blunted treatment response although TST moved closer to recommended sleep guidelines. Cluster 3 showed significant reductions in TST. Altogether, this is the first demonstration of different sleep profiles in schizophrenia and their influence on treatment response to CBT-I. It also supports the notion that therapies should be tailored to the person and their insomnia presentation.

1. Introduction

Sleep disorders often occur in people diagnosed with schizophrenia-spectrum disorders. An estimated 80% display symptoms of insomnia (Soehner et al., 2013), such as complaints of insufficient sleep, difficulties getting to sleep, waking up during the sleep period, and/or early waking and being unable to go back to sleep.

Recent studies conducted in the general population have revealed that insomnia may have different subtypes. For example, it has been shown that individuals may be differentiated at the level of insomnia severity or sleep duration (Bathgate et al., 2017; Miller et al., 2016), and that the source of variation may be explained in terms of individual differences in biological mechanisms, cognitive biases, lifestyle factors and/or personality traits (Benjamins et al., 2016; Dekker et al., 2017; Perlis and Gehrman, 2013; Sánchez-Ortuño and Edinger, 2010).

Most of the literature has focused on sleep duration as a distinct sleep dimension. Studies show that insomniacs who are short sleepers (<6 h) are defined by a phenotype which includes both biological correlates and cognitive underpinnings (e.g. Bathgate et al., 2017; Miller et al., 2016; Vgontzas et al., 2013). Evidence presented in support includes an increased incidence of diabetes (Vgontzas et al., 2009), hypertension (Fernandez-Mendoza et al., 2012), hyperactivation of the hypothalamic-pituitary-adrenal (HPA) axis, reduced cognitive performance (Fernandez-Mendoza et al., 2010), and increased mortality (Kripke et al., 2002), compared to people who sleep within a normal range of 7 to 9 hours. By contrast, insomniacs who sleep poorly in the context of normal sleep duration are better characterised by a psychological profile involving sleep state misperception, depression, anxious-ruminative personality traits, and poor coping resources, on a background of normal HPA axis activity and lack of medical comorbidities (Fernandez-Mendoza et al., 2011).

The benefits of insomnia subtyping include the development of a more accurate taxonomy of sleep disorders and improved diagnosis (Edinger et al., 2011). Identifying subtypes may also help to refine treatment targets to better meet the patient's needs (Bathgate et al., 2017; Vgontgas et al., 2013). For example, insomnia which is underpinned by psychological factors may be most responsive to cognitive and/or behavioural treatment, while the involvement of biological

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https://doi.org/10.1016/j.psychres.2018.07.027
Received 29 August 2017; Received in revised form 16 July 2018; Accepted 16 July 2018
Available online 24 July 2018
0165-1781/

factors may require pharmacotherapy (Vgontzas et al., 2013). There is empirical support for this proposal, with Bathgate et al. (2017) showing that insomniacs with normal sleep duration (in whom psychological factors may dominate over biological factors) had a better response to Cognitive Behavioural Therapy for Insomnia (CBT-I) than insomniacs with short sleep duration (in whom biological contributors may be implicated).

In individuals with severe mental illness, insomnia can significantly impede recovery, worsen symptoms and reduce quality of life. They too benefit substantially from CBT-I (Dashevsky and Kramer, 1998; Freeman et al., 2015; Haynes et al., 2011; Myers et al., 2011; Wagley et al., 2013; Waters et al., 2017). Despite recent advances in clinical research, sleep subtyping remains largely unexplored in psychiatric disorders, with few exceptions (e.g. van Mill et al., 2010; Kaplan et al., 2015). Attention is now turning to the large individual differences in sleep profiles (Harvey et al., 2009; Kaplan et al., 2011; Waters et al., 2011), and to variations in treatment success rates in schizophrenia and related psychoses (ranging from 29% to 75%; Dashevsky and Kramer, 1998; Freeman et al., 2015; Haynes et al., 2011; Myers et al., 2011; Wagley et al., 2013). Given such heterogeneity, there is a pressing need to investigate whether sleep subtypes can be discerned, and whether these are differentially associated with treatment response to CBT-I and other clinical correlates.

The current study sought to identify subtypes of insomnia in 74 participants with schizophrenia and related psychoses using latent class analysis (Aim 1), and to examine whether these subtypes were differentially associated with CBT-I treatment response as measured using self-reported sleep and clinical symptom measures (Aim 2). It was hypothesised that differences in sleep profiles would be revealed, and that these profiles would respond differently in the magnitude of improvements found after CBT-I.

2. Methods

This study uses data collected as part of an open-label trial of adapted CBT-I (as an adjunct to their usual medication) with a control group ('Treatment As Usual', TAU) described elsewhere (Chiu et al., 2017).

2.1. Participants

Participants were recruited from mental health outpatient services, drop-in centres, and sub-acute care units in the community. Inclusion criteria for all participants were: (a) a diagnosis of schizophrenia-spectrum disorder (i.e. schizophrenia, schizo-affective disorder, other psychosis) or psychiatric disorder with psychotic features (current hallucinations and delusions), as endorsed by their treating psychiatrist or case worker; (b) stable clinical condition and capacity to provide informed consent as endorsed by their treating psychiatrist or case worker; (c) self-reported symptoms of insomnia (problems getting to sleep, staying asleep, and/or early morning awakening, resulting in impaired daytime functioning); (d) a total score of five or more on the Pittsburgh Sleep Quality Index (PSQI) which is indicative of clinically significant sleep problems (Buysse et al., 1989), and (e) age > 18 years.

2.2. Procedure

All participants underwent baseline assessment with a range of self-report measures (see below) (n = 74). Participants then took part in either CBT-I (n = 50) or a TAU comparison condition (n = 24). All participants were included in the analyses for Aim 1, but only CBT-I participants were included in the analysis for Aim 2. A total of 40 participants completed two or more CBT-I treatment sessions and were included in the final analysis for Aim 2. Treatment lasted four to six weeks, in which participants received four weekly (or sometimes fortnightly) sessions of adapted CBT-I. The content of sessions were as follows:

- Session 1: Goal formation, Psychoeducation, Sleep hygiene, Winding down, Stimulus control and Establishment of regular rising time.
- Session 2: Exploring reasons for tiredness, Developing energy-generating strategies, Education on use/misuse of bright light, Reforming unhelpful beliefs about sleep, and Strategies for nightmares.
- Session 3: Strategies to reduce impact of low mood, intrusive thoughts and hallucinations on sleep, Addressing clock watching, Cognitive restructuring, and Brief relaxation.
- Session 4: Addressing impact of medications and substance use on sleep, Reviewing personal cycles of insomnia, Stress management, and Relapse prevention planning.

More detailed description of the program and its contents are presented in Waters et al. (2017). Baseline measures were used to analyse sleep profiles, and post-treatment outcomes were used to assess intervention efficacy.

2.3. Measures used for the subtyping of sleep profiles (Aim 1)

Subtyping made use of information derived at baseline from the *Pittsburgh Sleep Quality Index* (PSQI; Buysse et al., 1989), specifically, the raw data on total sleep time (TST), sleep efficiency (SE), sleep onset latency (SOL), time in bed (TIB), bedtime, rising time, and frequency of bad dreams and trouble staying awake. Participants were coded on these variables according to parameters used to quantify poor sleep in the literature as follows:

- *Sleep duration (TST):* 1 = short sleepers (≤6 h), 2 = normal sleepers (6–10 h), or 3 = long sleepers (≥10 h) (Hirshkowitz et al., 2015);
- *Sleep efficiency (SE):* 0 = very high (≥90%), 1 = normal (80–90%), 2 = low (70–80%), 3 = very low (≤ 70%) (Anderson et al., 2007; Kryger et al., 2015);
- *Sleep onset latency (SOL):* 1 = normal (≤30 mins), 2 = poor (30–60 mins), 3 = very poor (≥60 mins) (Kryger et al., 2015);
- *Time in bed (TIB):* 1 = normal (≤9 h), 2 = high (9–12 h), 3 = very high (≥ 12 h) (Kaplan et al., 2015);
- *Bedtime:* 1 = early sleeping time (6–9pm), 2 = normal sleeping time (9pm–2am), 3 = late sleeping time (2am–6am) (Horne and Ostberg, 1976);
- *Rise time:* 1 = early rising time (5am or earlier), 2 = normal rising time (5–10am), 3 = late rising time (10am or later) (Horne and Ostberg, 1976);
- *Bad dreams*: 0 = none in the past month, 1 = less than once a week, 2 = one to two times a week, 3 = three or more times a week (Buysse et al., 1989);
- *Daytime tiredness* (trouble staying awake): 0 = none in the past month, 1 = less than once a week, 2 = one to two times a week, 3 = three or more times a week (Buysse et al., 1989).

2.4. Measures used for the evaluation of CBT-I treatment effectiveness (Aim 2)

Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989): Assesses seven different sleep domains with 19-items rated along a 4-point Likert scale (0 = Not during the past month; 3 = Three or more times a week). It is also used to derive an individual's estimate of their SOL, TST, TIB, and SE over the last month.

Sleep Hygiene Behaviours Scale (SHiK-B; Chiu et al., 2015): Assesses for poor sleep hygiene practices in the past month using a 4-point Likert scale (0 = not at all; 4 = nearly every day). Scores can range from 0 to 21, with higher scores reflecting poorer sleep hygiene.

Adapted Mini International Neuropsychiatric Interview – Psychosis section (MINI-p; Sheehan et al., 1998): Evaluates seven psychotic