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

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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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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

symptoms, as well as additional questions about the impact of hallucinations rated on a 5-point Likert scale (1 = Very untrue; 5 = Very true).

Brief Patient Health Questionnaire (PHQ-4; Kroenke et al., 2009): Assesses for psychological distress using four items rated on a 4-point Likert scale (0 = not at all; 4 = nearly every day), and is also used to derive subscale scores for anxiety and depression.

2.5. Data analysis

All analyses were performed with Statistical Package for the Social Sciences version 22.0 (IBM Corp, Released 2013). The psychometric validity of measures was tested with factor analysis and reliability analysis. Latent class analysis (Latent GOLD, 4.0) was performed to explore if there were distinct sleep profiles in the participant sample. Baseline differences between clusters in demographic and clinical characteristics were subsequently assessed with independent *t*-test and chi-square procedures. Results of the cluster analysis were then used to determine whether differences in sleep profiles had any implications for treatment success in participants who had received CBT-I. Differences in response to CBT-I between different sleep profiles were examined using ANOVA of gain scores on outcome measures (significance level adjusted for multiple group comparisons using Holm–Bonferroni correction), and followed up with Tukey–Kramer post-hoc comparisons for unequal group sizes (or Games-Howell post hoc where unequal group variances existed) using an alpha level of 0.05. Overall effect sizes were derived using group mean scores and variances which were entered into the WebPower online calculator for One-Way ANOVA (Zhang and Yuan, 2015).

3. Results

Demographic and clinical characteristics of participants are presented in Table 1. All participants took daily antipsychotic medication and/or a mood stabiliser (sodium valproate, lithium). Psychiatric comorbidities (anxiety, borderline personality disorder, posttraumatic stress disorder, or eating disorder) were present in 41.9% (n = 31) of the whole sample.

3.1. Exploratory analysis of sleep profiles in schizophrenia and related psychoses (Aim 1)

Clustering analysis was performed in 74 participants. Latent Class Analysis cluster procedure was used to determine the underlying structure of sleep dysfunction in psychosis and categorize individuals on the basis of various sleep characteristics. Ultimately three sleep variables were most appropriate for generating the final cluster model. These were sleep duration (TST), sleep efficiency (SE), and sleep onset latency (SOL).

The Akaike Information Criterion (AIC3) was used to evaluate model fit. According to this criterion, the model with the smallest information criterion value should be preferred. The number of parameters used to estimate the model (Npar), classification error, and degrees of freedom (*df*) were also considered (Table 2). The level of contribution of each indicator to the final latent class structure was assessed by the information content statistic (R^2), which can be interpreted similarly to the commonalities in traditional factor analysis, and was as follows: Sleep duration R^2 = 0.65, Sleep efficiency R^2 = 0.74, and Sleep onset latency R^2 = 0.10.

The best-fitting model was a three-class solution: Cluster 1 (n = 33; 44.6%), Cluster 2 (n = 28; 37.8%) and Cluster 3 (n = 13; 17.6%), and this model also had the most meaningful clinical interpretability. Fig. 1 presents partial conditional probabilities ranging 0 to 1 for individual sleep variables within each of the clusters.

The identified clusters could be described as follows:

Table 1
Baseline demographic and clinical characteristics of samples for Aim 1 (N = 74) and Aim 2 (n = 40).

Measure	LCA sample (N = 74)	CBT-I sample (n = 40)
Age (years) ± SD	42.31 ± 10.8	41.68 ± 11.0
Gender (Male N, %)	39 (52.7%)	20 (50%)
Education		
Primary	2 (2.7%)	1 (2.5%)
Secondary	45 (60.8%)	25 (62.5%)
Post-school qualifications	27 (36.5%)	14 (35%)
Unemployed	57 (77.0%)	33 (82.5%)
Primary diagnosis		
Schizophrenia or schizoaffective disorder	43 (58.1%)	20 (50%)
Psychosis (NOS, organic, first episode)	7 (9.4%)	4 (10%)
Bipolar disorder	15 (20.3%)	8 (20%)
Major depression with psychotic features	5 (6.8%)	5 (12.5%)
Borderline personality disorder with psychotic features	4 (5.4%)	3 (7.5%)
Medications[1]		
Antipsychotics	64 (86.5%)	35 (87.5%)
Antidepressants	41 (55.4%)	15 (37.5%)
Mood stabilizers	4 (5.4%)	2 (5%)
Anticonvulsants	28 (37.8%)	15 (37.5%)
Benzodiazepines	21 (28.4%)	12 (30%)
Sedative medication	12 (16.2%)	9 (22.5%)
Chlorpromazine equivalents[2]	510.18 ± 460.83	529.81 ± 90.41
BNF percentages (%)[3]	65.75 ± 60.48	67.96 ± 11.20

[1] Missing medication type information: n = 2; [2]Missing medication dose information: n = 11; [3] BNF = Percentages of the British National Formulary's maximum recommended daily dose for antipsychotic use, Unable to calculate BNF information: n = 10.

Table 2
Testing five subsequent latent class analysis models.

Model	L^2	*df*	Npar	Classification error	AIC3
Once cluster	86.3	49	7	–	502.3
Two clusters	54.8	45	11	0.05	482.8
Three clusters	**37.9**	**41**	**15**	**0.09**	**477.9**
Four cluster	32.6	37	19	0.11	486.7
Five clusters	26.6	33	23	0.12	490.6

Notes: L^2 = Model fit likelihood ratio chi-squared statistic; *df* = Degrees of freedom; Npar = Number of parameters used to estimate model; AIC3 = Akaike Information Criterion.

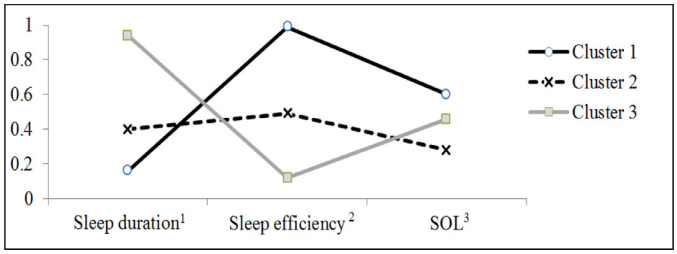

Fig. 1. Partial conditional probabilities for the three-class model
Notes: [1] For sleeper duration, higher probabilities represent longer total sleep time (TST); [2] For sleep efficiency, higher probabilities represent worse sleep efficiency (SE); [3] For SOL, higher probabilities represent longer sleep onset latency (SOL).

- *Cluster 1 ("Classic severe insomnia")* was the largest cluster (n = 33; 44.6%). Participants in this cluster were likely to report short sleep duration (average TST = 5.05 h), very poor sleep efficiency (average SE = 50.64%), and significantly prolonged SOL (average SOL = 94.9 min).

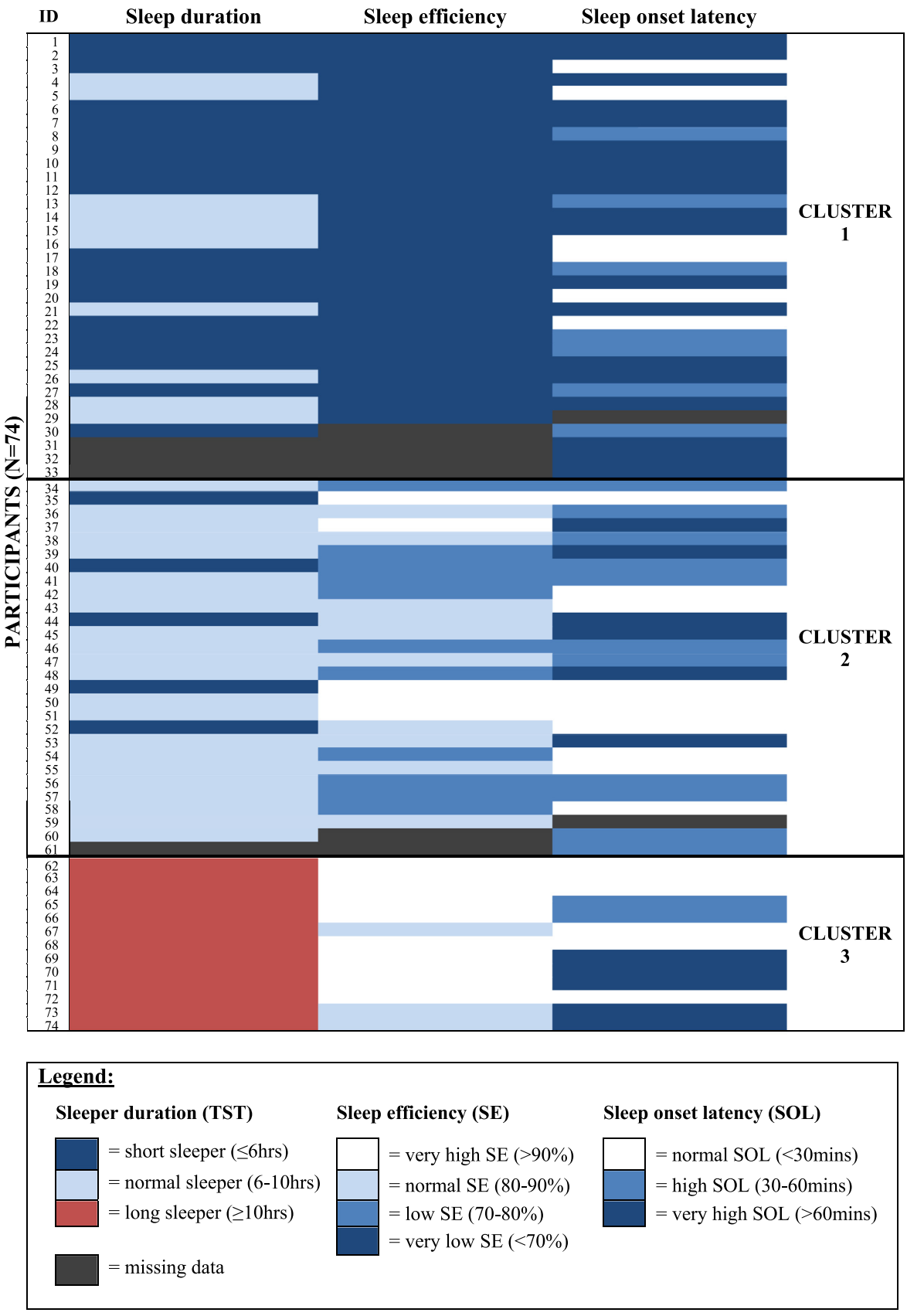

Fig. 2. Cluster profile matrix.

- *Cluster 2 ("Insomnia with normal sleep duration")* (n = 28; 37.8%) was characterized by individuals with prolonged SOL (average SOL = 57.2 mins) and slightly inefficient sleep (average SE = 81.22%), but normal sleep duration (average TST = 6.8 h).
- *Cluster 3 ("Insomnia with hypersomnia")* (n = 13; 17.6%) comprised participants who had insomnia comorbid with hypersomnia symptoms. They demonstrated excessively long sleep (average TST = 11.3 h) with a high sleep efficiency (average SE = 96.6%) although they complained of sleeping poorly, and over a third reported significantly prolonged SOL (average SOL = 86.15 mins).

A visual representation of the sleep profile of participants in each cluster may be seen in Fig 2, with each line representing a participant's sleep profile on each of the sleep variables: sleep duration, sleep efficiency, and sleep onset latency. Shades of blue are indicative of a gradient of deficits, with darker colours indicating greater sleep dysfunction. Fig 2 shows Cluster 1 to be the most consistently impaired (darker colors) across all three sleep variables, followed by Cluster 2. Red cells point to individuals with excessive sleep duration (Cluster 3).

3.1.1. Baseline demographic, clinical and sleep characteristics of clusters 1, 2 and 3

Table 3 shows the profile of the three clusters on demographic variables, PSQI scores and clinical symptoms. There were no significant group differences on demographic or clinical symptom variables. As expected given their profile above, Cluster 1 had significantly worse sleep on PSQI measures of sleep duration (i.e. Sleep Duration domain score and raw TST), sleep efficiency (i.e. Sleep Efficiency domain and raw SE), and global scores, compared to Clusters 2 and 3. The groups performed similarly on other sleep domains.

3.2. Differences in CBT-I treatment efficacy between sleep clusters (Aim 2)

Differences in treatment response to CBT-I between the three clusters were examined with a between groups ANOVA of gain scores on the dependent variables from baseline to post-treatment (Table 4).

3.2.1. Changes in sleep post CBT-I

There were significant overall effects showing the benefits of CBT-I on multiple sleep domains, namely PSQI Global ($F_{(1,\ 25)}$ = 22.23, p < .001), Sleep Disturbance ($F_{(1,\ 26)}$ = 10.95, p = .003), Sleep Latency ($F_{(1,\ 35)}$ = 12.92, p = .001), Sleep Quality ($F_{(1,\ 37)}$ = 29.36, p < .001), and raw SOL scores ($F_{(1,\ 35)}$ = 14.10, p = .001).

There were also significant differences between clusters in levels of improvement (i.e. gains) on the PSQI Sleep Duration ($F_{(2,\ 36)}$ = 7.63, p = .002), and raw TST ($F_{(2,\ 36)}$ = 15.51, p < .001) and SE ($F_{(2,\ 35)}$ = 10.265, p < .001) variables (See Table 4). Post hoc analyses revealed superior improvements in Cluster 1 compared to Cluster 2 and 3 on Sleep Duration, raw TST and SE (all p < .05). Average raw data for SE increased by 45% in Cluster 1, whilst there was no change in the other two clusters.

Cluster 2 showed a trend towards greater improvement in Daytime Dysfunction (difference of 0.6 points, with a large effect size: d = 0.79) compared to Cluster 1 (medium effect size: d = 0.57) and 3 (no change: d = 0.00), however this difference did not reach statistical significance.

Analyses showed significant difference in TST between the 3 clusters. When evaluating pre-post changes in TST, the direction of improvements differed between clusters. TST *increased* in Cluster 1 (by 1.46 h) and Cluster 2 (by 20 min), but *shortened* in Cluster 3 (by 1.37 h). The findings represented significant improvements for both Clusters 1 and 3 compared to Cluster 2 (both p < .05) as they moved closer towards a more normal, healthy range of sleep duration (See Fig. 3).

Finally, there were significant pre-post group differences in sleep hygiene (Table 4). Cluster 3 showed the most improved scores on the

Table 3
Baseline characteristics of different sleep clusters (N = 74).

Measure	Cluster 1 (n = 33)	Cluster 2 (n = 28)	Cluster 3 (n = 13)	Significance	Effect size (f)[6]	Post-hoc comparison
Age (years)	45.4 ± 11.8	40.5 ± 9.0	38.5 ± 10.38	$F_{(2,\ 71)}$ = 2.66, p = .08	0.28	1 = 2 = 3
Gender: Males (%)	16 (48.5%)	17 (60.7%)	6 (46.2%)	$\chi^2_{(2,\ 74)}$ = 1.18, p = .55		1 = 2 = 3
Highest education (%)				$\chi^2_{(4,\ 74)}$ = 9.17, p = .06		1 = 2 = 3
Primary school	1 (3.0%)	1 (3.6%)	0 (0.0%)			
Secondary school	24 (72.7%)	11 (39.3%)	10 (76.9%)			
Diploma/Graduate	8 (24.3%)	16 (57.1%)	3 (23.1%)			
Unemployed (%)	27 (81.8%)	19 (67.9%)	11 (84.6%)	$\chi^2_{(2,\ 74)}$ = 2.18, p = .34		1 = 2 = 3
Chlorpromazine equivalents[1]	437.8 ± 430.5	489.7 ± 490.7	659.3 ± 486.1	$F_{(2,\ 62)}$ = 0.92, p = .40	0.17	1 = 2 = 3
MINI-p total score	14.8 ± 7.6	13.8 ± 7.3	15.8 ± 7.4	$F_{(2,\ 65)}$ = 0.33, p = .72	0.10	1 = 2 = 3
PHQ-4 total score	5.9 ± 3.2	6.0 ± 3.5	6.2 ± 3.9	$F_{(2,\ 70)}$ = 0.03, p = .97	0.03	1 = 2 = 3
***PSQI* measure**						
Global score	14.8 ± 3.2	11.0 ± 2.7	9.6 ± 2.3	$F_{(2,\ 56)}$ = 15.25, p = .001*	0.76	**1 > 2 = 3**
Domain scores						
Sleep duration	1.9 ± 1.0	0.7 ± 1.0	0.0 ± 0.0	$F_{(2,\ 67)}$ = 23.03, p = .001*	0.90	**1 > 2 > 3**
Sleep disturbance	1.9 ± 0.7	1.6 ± 0.6	2.0 ± 0.8	$F_{(2,\ 60)}$ = 1.55, p = .22	0.22	1 = 2 = 3
Sleep latency	2.6 ± 0.8	2.3 ± 0.8	2.0 ± 1.2	$F_{(2,\ 69)}$ = 2.56, p = .08	0.25	1 = 2 = 3
Sleep efficiency	2.9 ± 0.4	1.0 ± 0.9	0.2 ± 0.4	$F_{(2,\ 67)}$ = 119.74, p < .001*	1.94	**1 > 2 > 3**
Daytime dysfunction	1.8 ± 1.0	1.7 ± 0.7	1.8 ± 1.0	$F_{(2,\ 60)}$ = 0.12, p = .89	0.06	1 = 2 = 3
Sleep quality	2.0 ± 0.9	2.0 ± 0.9	1.8 ± 0.8	$F_{(2,\ 71)}$ = 0.34, p = .71	0.10	1 = 2 = 3
Need for sleep medications	1.6 ± 1.4	2.1 ± 1.2	1.5 ± 1.5	$F_{(2,\ 71)}$ = 1.07, p = .35	0.17	1 = 2 = 3
Raw scores						
SOL (Mins)[2]	94.9 ± 65.0	57.2 ± 36.5	86.2 ± 92.5	$F_{(2,\ 69)}$ = 2.78, p = .07	0.25	1 > 2 = 3
TST (Hrs)[3]	5.1 ± 1.5	6.8 ± 1.5	11.3 ± 1.5	$F_{(2,\ 67)}$ = 79.51, p < .001*	1.50	**1 < 2 < 3**
TIB (Hrs)[4]	10.2 ± 1.7	8.5 ± 2.1	11.7 ± 1.1	$F_{(2,\ 71)}$ = 15.94, p < .001*	0.70	**1 > 3 > 2**
SE (%)[5]	50.7 ± 14.0	81.2 ± 10.6	96.6 ± 10.0	$F_{(2,\ 67)}$ = 81.10, p < .001*	1.58	**1 < 2 < 3**
SHiK-B total score	6.4 ± 3.7	5.2 ± 2.6	7.7 ± 3.4	$F_{(2,\ 70)}$ = 2.73, p = .07	0.27	1 = 2 = 3

Note: PSQI = Pittsburgh Sleep Quality Index; SOL = Sleep onset latency; TST = Total sleep time; TIB = Time in bed; SE = Sleep efficiency; SHiK-B = Sleep Hygiene Behaviours Scale; MINI-p = adapted Mini International Neuropsychiatric Interview – Psychosis section; PHQ-4 = Brief Patient Health Questionnaire; [1]Chlorpromazine equivalents calculated for: Cluster 1 n = 30; Cluster 2 n = 24; Cluster 3 n = 11; [2] Item 2 of PSQI. Significant differences found using Games-Howell statistic due to unequal group variances; [3]Item 4 of PSQI; [4]Calculated from Item 1 and Item 4 of PSQI; [5]Calculated by TST divided by TIB; [6]Overall effect size between the groups derived using WebPower's effect size calculator for One-way ANOVA using mean baseline scores and variances (Zhang and Yuan, 2015). *Significant p-value after applying Holm–Bonferroni correction.

Table 4
Scores (mean and standard deviation) on measures for sleep clusters 1–3 pre- and post-CBT-I intervention, and significance of gain.

Measure	Cluster 1 (n = 17)		Cluster 2 (n = 14)		Cluster 3 (n = 9)		Significance of gains scores	Effect size (f)[1]	Post-hoc comparisons
	Baseline	Post-treat	Baseline	Post-treat	Baseline	Post-treat			
PSQI measure									
Global score	15.7 ± 3.1	11.6 ± 3.1	12.1 ± 2.3	9.8 ± 4.3	9.5 ± 1.9	7.5 ± 1.9	$F_{(2, 25)} = 1.63$, $p = .22$	0.33	1 = 2 = 3
Domain scores									
Duration	2.1 ± 1.1	1.1 ± 1.2	0.8 ± 1.1	0. 7 ± 1.1	0.0 ± 0.0	0.0 ± 0.0	$F_{(2, 36)} = 7.63$, $p < .01$*	0.69	**1 > 2 = 3**
Disturbance	2.2 ± 0.8	1.5 ± 0.7	1.9 ± 0.3	1.6 ± 0.5	2.0 ± 0.8	1.8 ± 0.5	$F_{(2, 26)} = 1.39$, $p = .27$	0.33	1 = 2 = 3
Latency	2.4 ± 1.0	1.6 ± 1.0	2.5 ± 0.7	2.0 ± 0.9	1.8 ± 1.3	1.4 ± 0.7	$F_{(2, 35)} = 0.988$, $p = .38$	0.23	1 = 2 = 3
Efficiency	2.8 ± 0.4	1.8 ± 1.2	1.0 ± 0.6	1.2 ± 1.2	0.1 ± 0.3	0.1 ± 0.3	$F_{(2, 35)} = 4.92$, $p = .01$	0.54	1 > 2 = 3
Daytime dysfunction	2.2 ± 0.9	1.8 ± 0.9	2.0 ± 0.5	1.4 ± 0.7	2.0 ± 0.8	2.0 ± 0.8	$F_{(2, 26)} = 0.78$, $p = .47$	0.20	1 = 2 = 3
Quality	2.1 ± 0.8	1.5 ± 0.9	2.3 ± 0.7	1.3 ± 0.7	1.8 ± 0.8	1.0 ± 0.5	$F_{(2, 37)} = 1.14$, $p = .33$	0.24	1 = 2 = 3
Need for medications	1.7 ± 1.5	1.9 ± 1.5	2.2 ± 1.1	1.9 ± 1.4	1.4 ± 1.5	1.0 ± 1.3	$F_{(2, 37)} = 1.42$, $p = .25$	0.27	1 = 2 = 3
Raw scores									
SOL (Mins)	84.8 ± 65.4	39.1 ± 40.0	58.9 ± 26.2	44.5 ± 24.8	78.9 ± 93.4	27.1 ± 17.5	$F_{(2, 35)} = 1.39$, $p = .26$	0.25	1 = 2 = 3
TST (Hrs)	5.1 ± 1.3	6.5 ± 1.8	6.8 ± 1.8	7.1 ± 1.7	11.7 ± 1.6	10.3 ± 1.1	$F_{(2, 36)} = 15.51$, $p < .01$*	0.87	**1 > 2 > 3**
TIB (Hrs)	10.4 ± 1.9	9.3 ± 1.8	8.7 ± 2.5	9.1 ± 1.9	11.8 ± 1.2	10.8 ± 1.5	$F_{(2, 36)} = 3.01$, $p = .06$	0.41	1 = 2 = 3
SE (%)	49.8 ± 12.0	72.4 ± 20.2	79.0 ± 6.1	79.1 ± 11.9	99.3 ± 10.2	96.6 ± 11.3	$F_{(2, 35)} = 10.25$, $p < .01$*	0.78	**1 > 2 = 3**
SHiK-B total	5.4 ± 3.7	4.9 ± 3.4	4.5 ± 2.8	4.0 ± 2.4	8.0 ± 3.3	4.8 ± 3.8	$F_{(2, 36)} = 6.42$, $p < .01$*	0.56	**1 = 2 < 3**
MINI-p total	14.3 ± 7.4	13.4 ± 7.3	13.8 ± 6.8	12.2 ± 5.8	19.1 ± 6.7	16.1 ± 6.4	$F_{(2, 33)} = 0.90$, $p = .42$	0.22	1 = 2 = 3
PHQ-4 total	6.5 ± 3.2	5.1 ± 3.2	6.4 ± 3.1	5.5 ± 4.0	6.2 ± 4.0	3.6 ± 2.7	$F_{(2, 36)} = 1.30$, $p = .29$	0.24	1 = 2 = 3

Notes: PSQI = Pittsburgh Sleep Quality Index; SOL = Sleep onset latency; TST = Total sleep time; TIB = Time in bed; SE = Sleep efficiency; SHiK-B = Sleep Hygiene Behaviours Scale; MINI-p = adapted Mini International Neuropsychiatric Interview – Psychosis section; PHQ-4 = Brief Patient Health Questionnaire. [1]Overall effect size between the groups derived using WebPower's effect size calculator for One-way ANOVA using mean gain scores and variances (Zhang and Yuan, 2015). *Significant p-value after applying Holm-Bonferroni correction.

SHiK-B, relative to Clusters 1 and 2 (both $p < .05$).

3.2.2. Changes in clinical symptoms

All three clusters showed improvements in severity of psychotic symptoms ($F(1,33) = 9.16$, $p = .005$) and psychological distress ($F(1, 36) = 13.91$, $p = .001$) following treatment. On average, overall psychological distress, anxiety and depression subscale scores were no longer at clinical levels by post-treatment (i.e. PHQ-4 total score < 6, anxiety and depression scores < 3). The magnitude of improvement in clinical symptoms was similar across the three clusters, as shown by a lack of significant group differences in the gain scores analysis.

4. Discussion

This study sought to identify sleep subtypes in a sample of individuals with schizophrenia (Aim 1), and investigate any differences in the clusters' responses to CBT-I treatment (Aim 2). Results revealed three distinct sleep profiles. These were linked to different types of sleep improvement following CBT-I, although levels of improvement in the severity of psychopathology were similar across the groups.

4.1. Sleep profiling in schizophrenia and related psychoses

By conducting exploratory latent class analysis on PSQI data, we were able to identify three important sleep parameters (sleep duration, sleep efficiency, and sleep onset latency) which were used to classify

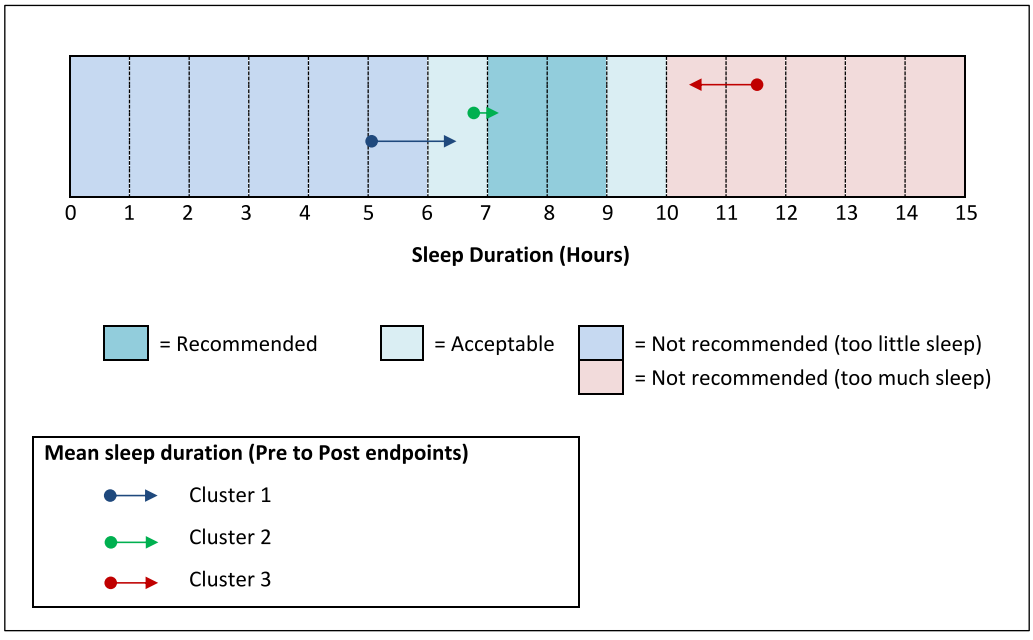

Fig. 3. Mean improvements in subjective total sleep time (TST) between clusters following adapted CBT-I treatment.

three sleep clusters. In line with previous studies (Gruber et al., 2009; Steinan et al., 2016; van Mill et al., 2010), we found differences in TST such that short sleeper (≤6 h), normal sleeper (6–10 h) and long sleeper (≥ 10 hours) groups were clearly identified in the current sample. The overall model was enhanced by the inclusion of SE and SOL as dependent variables, suggesting that variability in sleep profiles of people with schizophrenia and related psychoses extends beyond sleep duration.

The three sleep clusters were termed as follows: Cluster 1 - classic severe insomnia, Cluster 2 - insomnia with normal sleep duration, and Cluster 3 - insomnia with hypersomnia. Sleep duration and sleep efficiency mostly differentiated Cluster 1 (excessively short sleep; very poor SE) from Cluster 3 (excessively long sleep; high SE). Cluster 2 differed from the other groups on the basis of inefficient sleep and normal sleep duration. All three profiles featured prolonged sleep onset latency as a characteristic, although Cluster 1 was most severe with an average of 94.9 min.

Cluster 3 exhibited a feature of sleep dysfunction known as hypersomnia, which is rarely addressed despite being a prominent problem in this clinical group (Chiu et al., 2016; Gruber et al., 2009; Lieberman et al., 2005; Steinan et al., 2016). Traditional sleep measures do not assess hypersomnia (e.g. PSQI, Insomnia Severity Index), so the current identification of long sleepers underscores the value of sleep profiling. On the current measures, this cluster demonstrated very long sleep duration, high sleep efficiency and prolonged SOL. In contrast to hypersomnia in the general population (which shows short SOL), hypersomnia is often associated with long SOL in the psychiatric literature (Plante, 2017; Soehner et al., 2014; Vgontzas et al., 2000), in line with suggestions that psychiatric hypersomnia may be a disorder of hyperarousal (Vgontzas et al. 2000).

4.2. Responses to CBT-I treatment

All three sleep profiles demonstrated significant improvements on sleep and clinical symptoms after CBT-I treatment. As a group, participants were able to fall asleep quicker, sleep better, and showed improvements in depression, anxiety, and overall psychological distress.

As predicted, there were important differences in the magnitude and direction of improvements between profiles. Sleep parameters in participants in Cluster 1 (classic severe insomnia) showed the largest changes in treatment response, as evidenced by significantly longer total sleep time and improved sleep efficiency. Notwithstanding such improvements, their average sleep efficiency following CBT-I was 72.4%, which is still far below the normal range for adults (80%−95%; Ohayon et al., 2004). As suggested above, insomniacs who also have short sleep are more likely to have biological and medical problems (e.g. hypertension, diabetes, sleep apnoea) (Bathgate et al., 2017; Chin et al., 2010; Vgontzas et al., 2009). Consequently, further investigations around the addition of biological treatments (e.g. surgery, pharmacotherapy) may assist in maximising treatment gains for this group.

Cluster 2 (insomnia with normal sleep duration) showed a mixed response to CBT-I compared to the other clusters. At post-treatment, this group reported improvements in daytime functioning and TST, but no changes in sleep efficiency. Of note, Cluster 2 had the least severe sleep profile, and it was closer to the normal range at baseline than the other groups. Additionally, Cluster 2 increased their TIB post-treatment (from 8.7 to 9.1 hours) which would leave their SE relatively stagnant given the increase in TST. This suggests that an additional emphasis on time in bed restriction may be needed for this group. Nevertheless, sleep duration in Cluster 2 had improved such that their average TST now reached the recommended range (7 to 9 h) (Fig. 3), and sleep efficiency was on the cusp of the recommended range. Furthermore, improvement in psychopathology was similar to that of the other groups. In the general population it is estimated that only 50% of people with insomnia have short sleep duration (Vgontgas et al., 2009), and this underscores the importance of a holistic assessment of insomnia that is not focussed only on sleep parameters to evaluate treatment response.

At baseline, Cluster 3 (insomnia with hypersomnia) showed long sleep duration, high SE, and prolonged SOL. Following CBT-I, this group manifested significant reductions in raw TST and marked improvements in SOL (more than halved the time taken to fall asleep). This is the first study, to our knowledge, demonstrating the benefits of CBT-I in individuals with a profile of hypersomnia and psychosis. These individuals also showed significantly greater improvement in sleep hygiene, and were able to reduce TST towards a healthier range. It is important to note that the PSQI global score and Sleep Duration domain were not sensitive in detecting oversleeping, and therefore that measures of SOL, TST, and Daytime Dysfunction may provide clearer indications of sleep improvement for individuals with hypersomnia. Long sleep duration confers the same negative health outcomes as for short sleepers, and therefore merits greater research and treatment (Gruber et al., 2009; Kripke et al., 2002; van Mill et al., 2010).

In the current study, attention to the direction of TST change after treatment (i.e. increase or decrease towards a normal range of sleep) proved important. Existing meta-analytic reviews of CBT-I in primary insomnia (Morin et al., 1994; Murtagh and Greenwood, 1995; Okajima et al., 2011; Trauer et al., 2015) and secondary insomnia in psychiatric and medical conditions (Geiger-Brown et al., 2015; Koffel et al., 2015; Wu et al., 2015) have historically reported small improvements in TST (average increase of 7.6–43.1 min; all small effect sizes), however patient heterogeneity at baseline may have masked treatment effects. These studies generally conceptualised increases in TST as indicative of sleep improvement, rather than evaluating improvement as an increase or decrease towards a normal range of sleep. Such research assumes that no change in TST indicates a treatment non-response, however, large improvement in SE and daytime dysfunction can occur with no change in TST. Future studies should consider examining TST in short, normal, and long sleepers separately.

The capacity of CBT-I to address insomnia in short, normal, and long sleepers with psychosis (as shown in the current findings) holds much clinical relevance, as sleep duration can fluctuate during different stages of illness and medication changes (Gruber et al., 2011; Kaplan and Harvey, 2009; Neylan et al., 1992). A successful outcome in all three sleep profiles argues for the versatility of CBT-I in severe mental illness.

4.3. Limitations and future directions for research

There were a number of limitations in this study. This study used subjective reports of sleep parameters which may be influenced by sleep state misperceptions (Fernandez-Mendoza et al., 2011). However, subjective reports are normally used in the diagnosis of sleep disorders and to determine patient response and treatment effectiveness (Moul et al., 2004). It would be fruitful for future studies to utilise objective sleep measures and biological factors such as heart rate and respiratory events in the investigations of sleep subtyping. A further limitation of the study was the small sample size used for Latent Class Analysis, which generally requires a minimum sample of 100 to 200 participants (Wurpts and Geiser, 2014). The clusters identified in this study are very much preliminary in nature, and replication studies using much larger samples are required. Future research with larger sampling is also required to confirm the different sleep profiles in this and other clinical conditions. Lastly, the role of psychiatric co-morbidity and psychiatric medications in sleep subtyping merits greater consideration. For example, somnolence is a common side-effect of antipsychotics (Gao et al., 2008, 2013), which may be contributing to prolonged total sleep time and time in bed. In addition, sleep profiles may change according to psychiatric states. In mood disorders, for example, sleep changes according to mood cycle, such that individuals with bipolar disorder may be more likely to experience a ‘classic severe insomnia’ profile during manic episodes, and an ‘insomnia with

hypersomnia' profile leading up to, and during, depressive episodes (Kaplan et al., 2011; Kaplan and Harvey, 2009).

4.4. Implications for treatment

A number of treatment implications may be drawn from this study. Firstly, as prolonged SOL was a common feature of all three sleep profiles, strategies such as time in bed restriction, winding down before bed, stimulus control, and relaxation, are likely to benefit the majority of clients with schizophrenia who present for CBT-I treatment. Together, these elements can assist clients to go to bed feeling sleepy at both a physiological and psychological level. Where applicable, managing negative mood, anxiety, and psychotic symptoms will be important to create a comfortable scenario for falling asleep, and helps reduce reliance on medications which may be causing undesirable side effects (e.g. excessive daytime tiredness).

For people with classic severe insomnia profiles (Cluster 1), the focus should be on increasing both TST and SE. Elements most likely to be successful for improving these domains include stimulus control, avoiding clockwatching, psychoeducation about nocturnal wakeups, and addressing unhelpful beliefs about sleep, as well as time in bed restriction. These help to reduce sleep monitoring behaviours and over-concern with lack of sleep that increase arousal during the night. If sleep problems are severe, individuals may also require additional biological treatments to assist in normalising sleep in the short term.

For clients with normal sleep duration but reduced SE (Cluster 2), the focus is not so much on increasing TST, but making sleep more efficient and reducing daytime dysfunction. Clients may benefit from stimulus control, psychoeducation, and addressing unhelpful beliefs about sleep, and time in bed restriction to enhance sleep efficiency. It may be that a greater focus on cognitive-based strategies would be more helpful for this profile, as it was found to be particularly effective for normal sleepers with insomnia (Bathgate et al., 2017). Behavioural experiments that address sleep state misperception, such as showing clients discrepancies in their objective versus subjectively measured sleep data (e.g. actigraphy vs. sleep diaries; Tang and Harvey, 2006), and teaching clients to differentiate between sleepiness and fatigue, may be of particular benefit.

For those with comorbid insomnia and hypersomnia (Cluster 3), sleep hygiene and behavioural activation may be most important. People with hypersomnia generally spend excessive time in bed and/or have excessive daytime sleepiness (Kaplan et al., 2015), and therefore, behavioural activation and activity scheduling should be used to manage daytime fatigue and reduce napping (Chiu et al., 2016; Waite et al., 2016). Establishing a regular wake-up time may also be particularly helpful for limiting time in bed. Finally, a review of medication timing and dosing is advised, as this may be implicated in hypersomnia symptoms for individuals with schizophrenia (Gao et al., 2013; Lieberman et al., 2005).

5. Conclusions

In summary, our data suggest that different sleep profiles exist in schizophrenia and related psychoses. By identifying these sleep profiles, we were able to show areas in which people are most likely to benefit from CBT-I. In terms of sleep parameters, adapted CBT-I appears most effective for individuals who were identified as having 'classic severe insomnia', and also for normalising sleep in those with insomnia co-morbid with hypersomnia. It is also important to consider that individuals reporting insomnia with normal sleep duration may have less room to improve on sleep parameters, and their response to CBT-I may be better monitored through daytime dysfunction.

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Disclosure

The authors declare no conflict of interest.

Supplementary materials

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References

Anderson, K.N., Pilsworth, S., Sharples, L.D., Smith, I.E., Shneerson, J.M., 2007. Idiopathic hypersomnia: a study of 77 cases. Sleep 30 (10), 1274–1281.

Bathgate, C.J., Edinger, J.D., Krystal, A.D., 2017. Insomnia patients with objective short sleep duration have a blunted response to cognitive behavioural therapy for insomnia. Sleep 40 (1), zsw012.

Benjamins, J.S., Migliorati, F., Dekker, K., Wassing, R., Moens, S., Blanken, T.F., te Lindert, B.H.W., Mook, J.S., Van Someren, E.J.W., 2016. Insomnia heterogeneity: characteristics to consider for data-driven multivariate subtyping. Sleep Med. Rev. 36, 71–81.

Buysse, D.J., Reynolds III, C.F., Monk, T.H., Berman, S.R., Kupfer, D.J., 1989. The Pittsburgh sleep quality index: a new instrument for psychiatric practice and research. Psychiatry Res. 28 (2), 193–213.

Chin, K., Oga, T., Takahashi, K.-i., Takegami, M., Nakayama-Ashida, Y., Wakamura, T., Sumi, K., Nakamura, T., Horita, S., Oka, Y., 2010. Associations between obstructive sleep apnea, metabolic syndrome, and sleep duration, as measured with an actigraph, in an urban male working population in Japan. Sleep 33 (1), 89–95.

Chiu, V.W., Harvey, R.H., Sloan, N.B., Ree, M., Lin, A., Janca, A., Waters, F., 2015. Cognitive and behavioural factors associated with insomnia in inpatients with schizophrenia and related psychoses. J. Nerv. Ment. Dis. 203 (10), 798–803.

Chiu, V.W., Ree, M., Janca, A., Iyyalol, R., Waters, F., 2018. Treatment effectiveness of cognitive behavioural therapy for insomnia (CBT-I) for schizophrenia in 'real world' clinical settings. In V. Chiu, Cognitive Behavioural Therapy for Insomnia (CBT-1) in schizophrenia and related psychoses, Doctor of Philosophy, The University of Western Australia. DOI:10.4225/23/5ad043ff9a033.

Chiu, V.W., Ree, M., Janca, A., Waters, F., 2016. Sleep in schizophrenia: exploring subjective experiences of sleep problems, and implications for treatment. Psychiatr. Q. 87 (4), 633–648.

Dashevsky, B.A., Kramer, M., 1998. Behavioural treatment of chronic insomnia in psychiatrically ill patients. J. Clin. Psychiatry. 59 (12), 693–701.

Dekker, K., Blanken, T., Van Someren, E., 2017. Insomnia and personality – a network approach. Brain Sci. 7 (3), 28.

Edinger, J.D., Wyatt, J.K., Stepanski, E.J., Olsen, M.K., Stechuchak, K.M., Carney, C.E., Chiang, A., Crisostomo, M.I., Lineberger, M.D., Means, M.K., Radtke, R.A., Wohlgemuth, W.K., Krystal, A.D., 2011. Testing the reliability and validity of DSM-IV-TR and ICSD-2 insomnia diagnoses: results of a multitrait-multimethod analysis. Arch. Gen. Psychiatry 68 (10), 992–1002.

Fernandez-Mendoza, J., Calhoun, S., Bixler, E.O., Pejovic, S., Karataraki, M., Liao, D., Vela-Bueno, A., Ramos-Platon, M.J., Sauder, K.A., Vgontzas, A.N., 2010. Insomnia with objective short sleep duration is associated with deficits in neuropsychological performance: a general population study. Sleep 33 (4), 459–465.

Fernandez-Mendoza, J., Calhoun, S.L., Bixler, E.O., Karataraki, M., Liao, D., Vela-Bueno, A., Ramos-Platon, M.J., Sauder, K.A., Basta, M., Vgontzas, A.N., 2011. Sleep misperception and chronic insomnia in the general population: the role of objective sleep duration and psychological profiles. Psychosom. Med. 73 (1), 88–97.

Fernandez-Mendoza, J., Vgontzas, A.N., Liao, D., Shaffer, M.L., Vela-Bueno, A., Basta, M., Bixler, E.O., 2012. Insomnia with objective short sleep duration and incident hypertension: the Penn State Cohort. Hypertension 60 (4), 929–935.

Freeman, D., Waite, F., Startup, H., Myers, E., Lister, R., McInerney, J., Harvey, A.G., Geddes, J., Zaiwalla, Z., Luengo-Fernandez, R., Foster, R., Clifton, L., Yu, L.-M., 2015. Efficacy of cognitive behavioural therapy for sleep improvement in patients with persistent delusions and hallucinations (BEST): a prospective, assessor-blind, randomised controlled pilot trial. Lancet Psychiatry 2 (11), 975–983.

Gao, K., Ganocy, S.J., Gajwani, P., Muzina, D.J., Kemp, D.E., Calabrese, J.R., 2008. A

review of sensitivity and tolerability of antipsychotics in patients with bipolar disorder or schizophrenia: focus on somnolence. J. Clin. Psychiatry 69 (2), 302–309.
Gao, K., Mackle, M., Cazorla, P., Zhao, J., Szegedi, A., 2013. Comparison of somnolence associated with asenapine, olanzapine, risperidone, and haloperidol relative to placebo in patients with schizophrenia or bipolar disorder. Neuropsychiatr. Dis. Treat. 9, 1145–1157.
Geiger-Brown, J.M., Rogers, V.E., Liu, W., Ludeman, E.M., Downton, K.D., Diaz-Abad, M., 2015. Cognitive behavioural therapy in persons with comorbid insomnia: a meta-analysis. Sleep. Med. Rev. 23, 54–67.
Gruber, J., Harvey, A.G., Wang, P.W., Brooks III, J.O., Thase, M.E., Sachs, G.S., Ketter, T.A., 2009. Sleep functioning in relation to mood, function, and quality of life at entry to the systematic treatment enhancement program for bipolar disorder (STEP-BD). J. Affect. Disord. 114 (1–3), 41–49.
Gruber, J., Miklowitz, D.J., Harvey, A.G., Frank, E., Kupfer, D., Thase, M.E., Sachs, G.S., Ketter, T.A., 2011. Sleep matters: sleep functioning and course of illness in bipolar disorder. J. Affect. Disord. 134 (1–3), 416–420.
Harvey, A.G., Talbot, L.S., Gershon, A., 2009. Sleep disturbance in bipolar disorder across the lifespan. Clin. Psychol. Sci. Pract. 16 (2), 256–277.
Haynes, P.L., Parthasarathy, S., Kersh, B., Bootzin, R.R., 2011. Examination of insomnia and insomnia treatment in psychiatric inpatients. Int. J. Ment. Health. Nurs. 20 (2), 130–136.
Hirshkowitz, M., Whiton, K., Albert, S.M., Alessi, C., Bruni, O., DonCarlos, L., Hazen, N., Herman, J., Katz, E.S., Kheirandish-Gozal, L., Neubauer, D.N., O'Donnell, A.E., Ohayon, M., Peever, J., Rawding, R., Sachdeva, R.C., Setters, B., Vitiello, M.V., Ware, J.C., Adams Hillard, P.J., 2015. National sleep foundation's sleep time duration recommendations: methodology and results summary. Sleep Health 1 (1), 40–43.
Horne, J.A., Ostberg, O., 1976. A self-assessment questionnaire to determine morningness-eveningness in human circadian rhythms. Int. J. Chronobiol. 4 (2), 97–110.
IBM Corp, Released, 2013. IBM SPSS Statistics For Windows, Version 22.0. IBM Corp, Armonk, New York.
Kaplan, K.A., Gruber, J., Eidelman, P., Talbot, L.S., Harvey, A.G., 2011. Hypersomnia in inter-episode bipolar disorder: does it have prognostic significance. J. Affect. Disord. 132 (3), 438–444.
Kaplan, K.A., Harvey, A.G., 2009. Hypersomnia across mood disorders: a review and synthesis. Sleep Med. Rev. 13 (4), 275–285.
Kaplan, K.A., McGlinchey, E.L., Soehner, A., Gershon, A., Talbot, L.S., Eidelman, P., Gruber, J., Harvey, A.G., 2015. Hypersomnia subtypes, sleep and relapse in bipolar disorder. Psychol. Med. 45 (8), 1751–1763.
Koffel, E.A., Koffel, J.B., Gehrman, P.R., 2015. A meta-analysis of group cognitive behavioural therapy for insomnia. Sleep Med. Rev. 19, 6–16.
Kripke, D.F., Garfinkel, L., Wingard, D.L., Klauber, M.R., Marler, M.R., 2002. Mortality associated with sleep duration and insomnia. Arch. Gen. Psychiatry. 59 (2), 131–136.
Kroenke, K., Spitzer, R.L., Williams, J.B.W., Löwe, B., 2009. An ultra-brief screening scale for anxiety and depression: the PHQ–4. Psychosomatics 50 (6), 613–621.
Kryger, M.H., Rosenberg, R., Martin, L., Kirsch, D.B., 2015. Kryger's Sleep Medicine Review: A Problem-Oriented Approach. Elsevier Health Sciences, Philadelphia.
Latent GOLD 4.0, Released, 2005. Statistical Innovations Inc., Belmont, Massachusetts.
Lieberman, J.A., Stroup, T.S., McEvoy, J.P., Swartz, M.S., Rosenheck, R.A., Perkins, D.O., Keefe, R.S.E., Davis, S.M., Davis, C.E., Lebowitz, B.D., Severe, J., Hsiao, J.K., 2005. Effectiveness of antipsychotic drugs in patients with chronic schizophrenia. New Engl. J. Med. 353 (12), 1209–1223.
Miller, C.B., Bartlett, D.J., Mullins, A.E., Dodds, K.L., Gordon, C.J., Kyle, S.D., Kim, J.W., D'Rozario, A.L., Lee, R.S.C., Comas, M., Marshall, N.S., Yee, B.J., Espie, C.A., Grunstein, R.R., 2016. Clusters of insomnia disorder: an exploratory cluster analysis of objective sleep parameters reveals differences in neurocognitive functioning, quantitative EEG, and heart rate variability. Sleep 39 (11), 1993–2004.
Morin, C.M., Culbert, J.P., Schwartz, S.M., 1994. Nonpharmacological interventions for insomnia. Am. J. Psychiatry. 151 (8), 1172.
Moul, D.E., Hall, M., Pilkonis, P.A., Buysse, D.J., 2004. Self-report measures of insomnia in adults: rationales, choices, and needs. Sleep Med. Rev. 8 (3), 177–198.
Murtagh, D.R.R., Greenwood, K.M., 1995. Identifying effective psychological treatments for insomnia: a meta-analysis. J. Consult. Clin. Psychol. 63 (1), 79–89.
Myers, E., Startup, H., Freeman, D., 2011. Cognitive behavioural treatment of insomnia in individuals with persistent persecutory delusions: a pilot trial. J. Behav. Ther. Exp Psychiatry. 42, 330–336.
Neylan, T.C., van Kammen, D.P., Kelley, M.E., Peters, J.L., 1992. Sleep in schizophrenic patients on and off haloperidol therapy: clinically stable vs relapsed patients. Arch. Gen. Psychiatry. 49 (8), 643–649.
Ohayon, M.M., Carskadon, M.A., Guilleminault, C., Vitiello, M.V., 2004. Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals. Sleep 27 (7), 1255–1273.
Okajima, I., Komada, Y., Inoue, Y., 2011. A meta-analysis on the treatment effectiveness of cognitive behavioural therapy for primary insomnia. Sleep Biol. Rhythms. 9, 24–34.
Perlis, M.L., Gehrman, P., 2013. Types of Insomnia. In: Kushida, C.A. (Ed.), Encyclopedia of Sleep. Academic Press, Oxford, UK, pp. 199–202.
Plante, D.T., 2017. Sleep propensity in psychiatric hypersomnolence: A systematic review and meta-analysis of multiple sleep latency test findings. Sleep Med. Rev. 31 (Supplement C), 48–57.
Sánchez-Ortuño, M.M., Edinger, J.D., 2010. A penny for your thoughts: patterns of sleep-related beliefs, insomnia symptoms and treatment outcome. Behav. Res. Ther. 48, 125–133.
Sheehan, D.V., Lecrubier, Y., Sheehan, K.H., Amorim, P., Janavs, J., Weiller, E., Hergueta, T., Baker, R., Dunbar, G.C., 1998. The mini-international neuropsychiatric interview (M.I.N.I): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. J. Clin. Psychiatry 59 (Suppl. 20), 22–33.
Soehner, A.M., Kaplan, K.A., Harvey, A.G., 2013. Insomnia comorbid to severe psychiatric illness. Sleep. Med. Clin. 8 (3), 361–371.
Soehner, A.M., Kaplan, K.A., Harvey, A.G., 2014. Prevalence and clinical correlates of co-occurring insomnia and hypersomnia symptoms in depression. J. Affect. Disord. 167 (Suppl. C), 93–97.
Steinan, M.K., Scott, J., Lagerberg, T.V., Melle, I., Andreassen, O.A., Vaaler, A.E., Morken, G., 2016. Sleep problems in bipolar disorders: more than just insomnia. Acta. Psychiatr. Scand. 133 (5), 368–377.
Tang, N.K., Harvey, A.G., 2006. Altering misperception of sleep in insomnia: behavioural experiment versus verbal feedback. J. Consult. Clin. Psychol. 74 (4), 767–776.
Trauer, J.M., Qian, M.Y., Doyle, J.S., Rajaratnam, S.M., W., Cunnington, D., 2015. Cognitive behavioural therapy for chronic insomnia: a systematic review and meta-analysis. Ann. Intern. Med. 163 (4), 191–204.
van Mill, J.G., Hoogendijk, W.J., Vogelzangs, N., van Dyck, R., Penninx, B.W., 2010. Insomnia and sleep duration in a large cohort of patients with major depressive disorder and anxiety disorders. J. Clin. Psychiatry 71 (3), 239–246.
Vgontzas, A.N., Bixler, E.O., Kales, A., Criley, C., Vela-Bueno, A., 2000. Differences in nocturnal and daytime sleep between primary and psychiatric hypersomnia: diagnostic and treatment implications. Psychosomatic Med. 62 (2), 220–226.
Vgontzas, A.N., Fernandez-Mendoza, J., Liao, D., Bixler, E.O., 2013. Insomnia with objective short sleep duration: the most biologically severe phenotype of the disorder. Sleep Med. Rev. 17 (4), 241–254.
Vgontzas, A.N., Liao, D., Pejovic, S., Calhoun, S., Karataraki, M., Bixler, E.O., 2009. Insomnia with objective short sleep duration is associated with type 2 diabetes: a population-based study. Diabetes Care 32 (11), 1980–1985.
Wagley, J.N., Rybarczyk, B., Nay, W.T., Danish, S., Lund, H.G., 2013. Effectiveness of abbreviated CBT for insomnia in psychiatric outpatients: sleep and depression outcomes. J. Clin. Psychol. 69 (10), 1043–1055.
Waite, F., Evans, N., Myers, E., Startup, H., Lister, R., Harvey, A.G., Freeman, D., 2016. The patient experience of sleep problems and their treatment in the context of current delusions and hallucinations. Psychol. Psychother. 89 (2), 181–193.
Waters, F., Ree, M.J., Chiu, V., 2017. Delivering CBT For Insomnia in Psychosis: A Clinical Guide. Routledge, New York.
Waters, F., Sinclair, C., Rock, D., Jablensky, A., Foster, R.G., Wulff, K., 2011. Daily variations in sleep–wake patterns and severity of psychopathology: a pilot study in community-dwelling individuals with chronic schizophrenia. Psychiatry Res. 187 (1), 304–306.
Wu, J.Q., Appleman, E.R., Salazar, R.D., Ong, J.C., 2015. Cognitive behavioural therapy for insomnia comorbid with psychiatric and medical conditions: a meta-analysis. JAMA. Intern. Med. 175 (9), 1461–1472.
Wurpts, I.C., Geiser, C., 2014. Is adding more indicators to a latent class analysis beneficial or detrimental? Results of a Monte-Carlo study. Front. Psychol. 5, 920.
Zhang, Z., Yuan, K.H., 2015. WebPower: Statistical power analysis online. https://webpower.psychstat.org/wiki/start accessed 8 January 2018.