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RESEARCH ARTICLE

Sleep–wake state discrepancy does not impair the efficacy of cognitive behavioural therapy for insomnia: Findings from a large clinic sample

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Summary

The current study determined the extent to which sleep–wake state discrepancy impairs the efficacy of cognitive behavioural therapy for insomnia in a real-world clinical sample. Sleep–wake state discrepancy occurs when there is an inconsistency between a person's subjective and objective sleep, and is a common phenomenon amongst patients with insomnia. Limited information is available on the effectiveness of cognitive behavioural therapy for insomnia in treating patients who experience significant sleep–wake state discrepancy in "real-world" samples. In the present study, all patients with insomnia received cognitive behavioural therapy for insomnia through an outpatient insomnia program ($N = 386$; mean age = 51.96 years, SD = 15.62; 65.97% [$N = 254$] female). Prior to treatment, participants completed a polysomnography sleep study and sleep diary, which was used to calculate sleep–wake state discrepancy. At pre-treatment, post-treatment and 3-month follow-up, participants completed the Insomnia Severity Index and other questionnaires, and 1 week of sleep diaries from which sleep-onset latency, wake after sleep onset and other sleep variables were calculated. There were no differences in self-reported sleep-onset latency, wake after sleep onset or Insomnia Severity Index scores at post-treatment or 3-month follow-up between quintiles of sleep–wake state discrepancy. These results indicate that sleep–wake state discrepancy at pre-treatment does not predict treatment response to cognitive behavioural therapy for insomnia. Future research could examine multi-night assessments of sleep–wake state discrepancy to determine whether variations in discrepancy may relate to pre-treatment insomnia severity and cognitive behavioural therapy for insomnia outcomes.

KEYWORDS
cognitive behavioural therapy for insomnia, insomnia, polysomnography, sleep diary, sleep initiation and maintenance, sleep–wake state discrepancy

1 | INTRODUCTION

One of the most common complaints in medical practice is insomnia (Benz et al., 2020). The chronic disorder affects about 6%–13% of the general population (Deloitte Access Economics, 2017; Harvey & Tang, 2012; van der Zweerde et al., 2019). The efficacy of treatment may alter due to a common feature of insomnia, where patients present with a substantial discrepancy between their self-reported sleep and their objectively measured sleep (Crönlein et al., 2019; Harvey & Tang, 2012). This phenomenon is known as sleep–wake state discrepancy, but has been referred to previously as sleep-state discrepancy, sleep misperception, subjective–objective sleep discrepancy, paradoxical insomnia, and similar terms (Bensen-Boakes, Osman, et al., 2022). Even for good sleepers, self-report estimates do not precisely match objective assessments of sleep (Cudney et al., 2022), but in the case of insomnia there can be substantial discrepancies between measures. This discrepancy usually presents when patients with insomnia underestimate total sleep time (TST) and sleep efficiency (Harvey & Tang, 2012), and overestimate the time it takes to fall asleep (sleep-onset latency; SOL) and/or the time spent awake during the night compared with polysomnography (PSG; wake after sleep onset [WASO]; Mercer et al., 2002). This study will test whether this common feature of insomnia is related to disorder severity and predicts worse treatment response.

Notably, sleep–wake state discrepancy could relate to the severity of insomnia, as recent evidence suggests that it contributes to the development and maintenance of insomnia (Bensen-Boakes, Lovato, et al., 2022). Thinking that sleeping difficulties are worse than they are could exacerbate anxiety about sleep and perpetuate sleeping difficulties (Harvey & Tang, 2012). In such cases, higher reliance on cognitive-focused treatments or components may be needed if sleep–wake state discrepancy is high. In addition, sleep–wake state discrepancy may indicate a different form of insomnia that objective sleep measures are unable to detect. In other words, patients are indeed hardly sleeping or sleeping poorly, which our traditional objective measures are too insensitive to detect (Andrillon et al., 2020). If this is the case, then this would suggest a neurophysiological cause to the insomnia symptoms (e.g. more fragmented sleep/altered sleep depth), which may exacerbate or perpetuate the insomnia and be more resistant to interventions that do not challenge these underpinnings. Therefore, high levels of sleep–wake state discrepancy may potentially make insomnia worse and more challenging to treat.

Given that sleep–wake state discrepancy may contribute to the perpetuation of the disorder, it may also have an impact on treatment efficacy. The best, first-line treatment for insomnia may resolve sleep–wake state discrepancy. Cognitive behavioural therapy for insomnia (CBTi) is the therapy of choice for chronic forms of insomnia (Crönlein et al., 2019). There are several individual components of CBTi, including sleep restriction, cognitive therapy, stimulus control therapy, sleep hygiene and psychoeducation, and relaxation (Benz et al., 2020). Due to sleep efficiency improvements and a better understanding of sleep–wake state discrepancy, CBTi may improve sleep–wake state discrepancy, which may explain the improvements in insomnia symptoms (Crönlein et al., 2019). However, although patients' sleep–wake state discrepancy improves following CBTi, it does not cease altogether (Crönlein et al., 2019). Given this, sleep–wake state discrepancy may thus increase the risk of relapse, impairing the longer-term efficacy of CBTi.

Past studies investigating sleep–wake state discrepancy and insomnia symptoms have been mainly conducted in research settings with relatively small samples. Because severe levels of sleep–wake state discrepancy occur in only a portion of patients with insomnia, prior studies are likely underpowered to detect effects (101 patients, 51.49% with insomnia [Means et al., 2003]; 63 patients with insomnia, 50% with sleep state discrepancy [Liao et al., 2018]; 20 patients with chronic insomnia [Crönlein et al., 2020]; 14 patients with chronic insomnia [Janků et al., 2020]), which may be why some studies have not found significant effects. Likewise, the research study context may influence results as participants may feel the need to exaggerate their sleeping difficulties at baseline to be eligible for the study (which usually involves receiving free treatment).

Because previous studies have mainly been conducted in relatively small samples, the current study will use a much larger clinical sample of patients with insomnia. This study will determine whether sleep–wake state discrepancy: (1) is associated with worse insomnia symptoms prior to treatment; and (2) impairs the efficacy of CBTi, in a "real-world" sample of patients with insomnia undergoing CBTi in an outpatient clinic.

2 | METHODS

2.1 | Participants

Participants were patients attending the Insomnia Treatment Program at the Repatriation General Hospital (Adelaide, South Australia) between February 2004 and November 2015. Five-hundred and thirty-eight patients with insomnia entered the program. Inclusion criteria were: informed consent for their clinical data to be used for research purposes; aged ≥ 18 years; and diagnosis of chronic insomnia according to DSM-IV criteria between 2004 and 2012, and DSM-V criteria between 2013 and 2015. Of the 455 patients who were eligible, 386 patients undertook PSG sleep recordings at pre-treatment and were therefore included in this study. The Southern Adelaide Clinical Human Research Ethics Committee reviewed and approved this study.

2.2 | Study design

This study employed a within-subjects, quasi-experimental design. All participants received CBTi. The independent variables were pre-treatment sleep–wake state discrepancy, which was the difference between self-reported and PSG-derived sleep variables (SOL, WASO and TST, continuous and split into quintiles), and time (pre-treatment, post-treatment, 3-month follow-up). The dependent variables were

changes in sleep diary-derived SOL and WASO, and changes in Insomnia Severity Index (ISI) scores from pre-treatment to post-treatment and follow-up.

3 | STUDY MATERIALS

3.1 | Insomnia treatment

Registered and clinical psychologists who were highly experienced in the administration of CBTi delivered treatment over approximately six, weekly, face-to-face sessions. This consisted of sleep restriction therapy (Spielman et al., 1987), stimulus control therapy (Bootzin, 1972), cognitive therapy (Harvey et al., 2007), relapse prevention and sleep education (Hauri, 1991). The emphasis of each individual therapy differed between patients, depending on factors such as the presentation of insomnia, the clinician's judgement, and the patient's goals for therapy. Findings from this clinical service were published previously, showing that the treatment is efficacious for treating insomnia (Sweetman et al., 2020).

3.2 | Sleep diary

Sleep diaries are regarded as the gold-standard of self-reported sleep measurement (Carney et al., 2012; Mallinson et al., 2019). Items for the sleep diary included questions such as "what time did you get into bed?" and "how many times did you wake up during the night?". Responses to these items were used to calculate time to fall asleep (SOL), time awake during the night (WASO) and TST. These variables were averaged across 1 week at pre-treatment, post-treatment and 3-month follow-up to create weekly outcome measures for sleep symptoms. Overnight sleep diaries that were completed on the night of the PSG at pre-treatment, along with same-night PSG data, were used to calculate sleep–wake state discrepancy variables.

3.3 | Home-based sleep study

Laboratory and home-based sleep studies were conducted at pre-treatment. Qualified technicians set up patients for overnight PSG using the 10–20 electrode placement system (Jasper, 1958). Once collected, qualified technicians manually scored the data into sleep stages across 30-s epochs (intervals) using standard scoring criteria (Berry et al., 2015). From these sleep staging data, standard sleep variables were calculated that align with the sleep variables of interest in this study, namely SOL, WASO and TST.

3.4 | Questionnaires

Several questionnaires were administered at pre-treatment, post-treatment and 3-month follow-up. These include the ISI to assess the severity of insomnia symptoms (Bastien et al., 2001), the Flinders Fatigue Scale (FFS) to assess fatigue (Gradisar et al., 2007), the Epworth Sleepiness Scale (ESS) to assess daytime sleepiness (Johns, 1992), the Daytime Feelings and Functioning Scale (DFFS) to assess mood, social and cognitive difficulties (Gradisar et al., 2006), the Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS) to assess sleep-related cognitions (Morin et al., 2007), and the Depression, Anxiety and Stress Scale-21 (DASS-21) to assess depression, anxiety and stress symptoms (Lovibond & Lovibond, 1996). Age, sex and body mass index (BMI) were obtained from patient health records.

3.4.1 | Procedures

Most participants were referred to the Insomnia Treatment Program by their general practitioner. At pre-treatment, participants completed a single-night sleep study and an overnight sleep diary on the following morning, which were used to calculate sleep–wake state discrepancy. Participants also completed questionnaires, as well as a week-long sleep diary (separate from the PSG night) in the week before treatment commenced (pre-treatment time point). Participants then completed approximately six, weekly sessions of CBTi. Immediately after treatment (post-treatment) and 3 months after treatment ended (follow-up), participants completed another week of sleep diaries and questionnaires.

3.4.2 | Statistical analyses

To assess sleep–wake state discrepancy, the differences between the single-night sleep diary and PSG sleep estimates were calculated for SOL, WASO and TST. These sleep–wake state discrepancy variables were calculated by subtracting the subjective measure (sleep diary from the sleep study night) from the objective measure (PSG). A negative value indicated participants underestimated the sleep variable, and a positive value indicated participants overestimated the variable. Each variable was split into quintiles for analysis.

One-way analyses of variance (ANOVAs) were used across quintiles for each sleep–wake state discrepancy variable to assess group differences on factors at pre-treatment, including participant demographics and insomnia symptoms.

Linear mixed models were conducted to test for differences in response to CBTi between each sleep–wake state discrepancy quintile at each time point following treatment. Maximum likelihood method was used to handle missing data. The covariance structure used was first-order autoregressive. Fixed effects were the main effects and interactions, participant ID was entered as the intercept, and time point was entered as repeated measures. These models tested for interactions between sleep–wake state discrepancy variables at pre-treatment (discrepancy between sleep diary and PSG-derived SOL, WASO and TST) by time point on changes in sleep diary-derived SOL and WASO, and ISI scores. They were conducted unadjusted in the

TABLE 1 Overall participant demographics.

Number of participants	386
Gender, no. female (%)	254 (65.97)
Age, M (SD)	51.96 (15.62)
BMI, M (SD)	26.27 (4.93)
FFS, M (SD)	18.41 (6.44)
ESS, M (SD)	5.83 (4.52)
DFFS, M (SD)	17.59 (7.25)
DBAS, M (SD)	39.46 (9.38)
DASS-depression, M (SD)	12.18 (9.86)
DASS-anxiety, M (SD)	7.41 (7.37)
DASS-stress, M (SD)	15.97 (9.33)

Abbreviations: BMI, body mass index; DASS, Depression, Anxiety and Stress; DBAS, Dysfunctional Beliefs and Attitudes about Sleep Scale; DFFS, Daytime Feelings and Functioning Scale; ESS, Epworth Sleepiness Scale; FFS, Flinders Fatigue Scale.

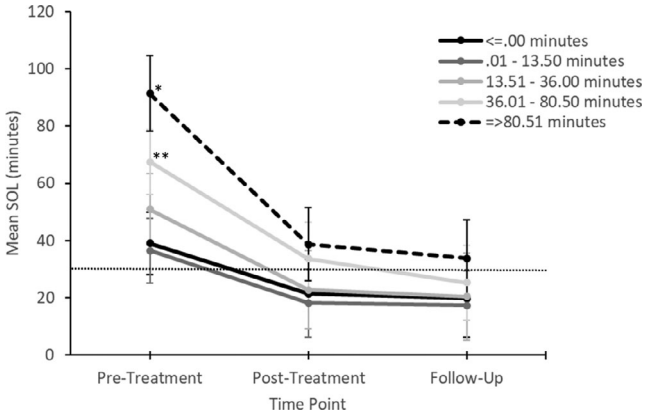

FIGURE 1 Interaction between sleep-state discrepancy for sleep-onset latency (SOL) by time point on self-reported SOL, with 95% confidence intervals. The dotted line indicates the clinically accepted threshold for SOL of 30 min. *$p < 0.05$ compared with quintiles 1, 2 and 3; **$p < 0.05$ compared with quintiles 1 and 2.

first instance, and then adjusted for age and sex, as insomnia symptoms typically vary by age and sex (Krishnan et al., 2006; Landolt & Borbély, 2001; Mourtazaev et al., 1995). Bonferroni adjusted post hoc tests were used to determine specific group differences where significant interactions were found. All analyses were conducted in SPSS (IBM, version 25).

4 | RESULTS

4.1 | Participant demographics

Participant demographics are shown in Table 1. Participants had a mean age of 51.96 years (SD = 15.62), with 65.97% of them being female ($N = 254$). The sample reported moderate levels of fatigue (FFS), low levels of excessive sleepiness (ESS), moderate levels of daytime impairment (DFFS), high levels of unhelpful beliefs about sleep (DBAS), and mild levels of depression, anxiety and stress (DASS-21).

No demographic factors were significantly different between quintiles of TST and SOL sleep–wake state discrepancy. For WASO sleep–wake state discrepancy, sex was the only demographic that was significantly different between groups ($F_{4,142} = 3.56$, $p = 0.008$, $\eta^2 = 0.091$). Games–Howell post hoc tests showed that quintile 1 (the group that underestimated WASO the most) had a significantly higher proportion of female versus male participants than quintile 2 ($p = 0.003$, $d = 0.95$). These findings are summarised in tables in Data S1.

4.2 | Effects of sleep–wake state discrepancy on change in self-reported SOL

There was no significant interaction between pre-treatment TST or WASO sleep–wake state discrepancy and time on self-reported SOL ($p > 0.551$). These interactions remained insignificant after adjusting for gender and age ($p > 0.106$).

There was a significant interaction between pre-treatment SOL sleep–wake state discrepancy and time on self-reported SOL ($F_{8,187.77} = 2.56$, $p = 0.011$; Figure 1). Bonferroni-adjusted post hoc tests showed that quintile 5 (≥ 80.51 min difference between measures) reported significantly higher self-reported SOL at pre-treatment than quintile 1 (≤ 0.00 min; M diff. = 52.27 min; SEM = 8.68; $p < 0.001$, $d = 8.51$), quintile 2 (0.01–13.50 min; M diff. = 54.87 min; SEM = 8.81; $p < 0.001$, $d = 8.81$) and quintile 3 (13.51–36.00 min; M diff. = 40.47 min; SEM = 9.17; $p < 0.001$, $d = 3.84$). Quintile 4 (36.01–80.50 min) had significantly higher self-reported SOL at pre-treatment than quintile 1 (≤ 0.00 min; M diff. = 28.38 min; SEM = 7.99; $p = 0.004$, $d = 5.10$) and quintile 2 (0.01–13.50 min; M diff. = 3 min; SEM = 8.13; $p = 0.002$, $d = 5.47$). All other group differences on self-reported SOL were not significant ($p > 0.05$). These effects did not differ after adjusting for gender ($F_{8,222.6} = 0.88$, $p = 0.532$) or age ($F_{8,227.86} = 1.49$, $p = 0.160$).

5 | EFFECTS OF SLEEP–WAKE STATE DISCREPANCY ON CHANGE IN SELF-REPORTED WASO

There was no significant interaction between pre-treatment TST sleep–wake state discrepancy and time on self-reported WASO ($F_{8,166.89} = 1.95$, $p = 0.056$).

There was a significant interaction between pre-treatment SOL sleep–wake state discrepancy and time on self-reported WASO ($F_{8,163.77} = 2.62$, $p = 0.010$; Figure 2). Bonferroni-adjusted post hoc tests showed that quintile 5 (≥ 80.51 min difference between measures) had significantly higher self-reported WASO at pre-treatment than quintile 1 (≤ 0.00 min; M diff. = 61.00; SEM = 14.95; $p < 0.001$, $d = 5.77$) and quintile 2 (0.01–13.50 min; M diff. = 54.04;

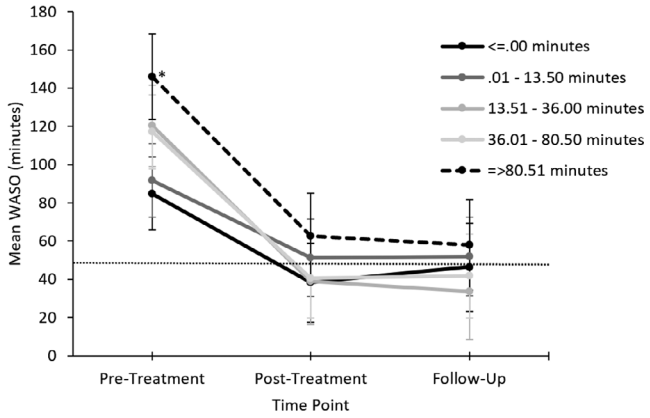

FIGURE 2 Interaction between sleep-state discrepancy for sleep-onset latency (SOL) by time point on self-reported wake after sleep onset (WASO), with 95% confidence intervals. The dotted line indicates the clinically accepted threshold for WASO (60 min [includes WASO and early-morning awakenings]). *$p < 0.05$ compared with quintiles 1 and 2.

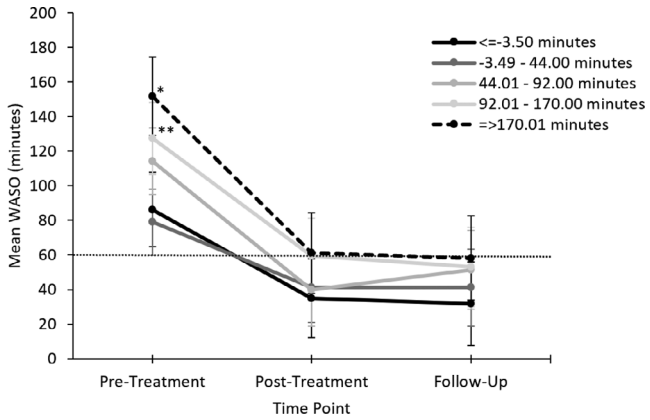

FIGURE 3 Interaction between sleep-state discrepancy for wake after sleep onset (WASO) by time point on self-reported WASO, with 95% confidence intervals. The dotted line indicates the clinically accepted threshold for WASO (60 min [includes WASO and early-morning awakenings]). *$p < 0.05$ compared with quintiles 1 and 2; **$p < 0.05$ compared with quintile 2.

SEM = 14.98; $p = 0.004$, $d = 5.10$). All other group differences on self-reported WASO were not significant ($p > 0.05$). The effect did not differ after adjusting for gender ($F_{8,198.10} = 0.69$, $p = 0.704$) or age ($F_{8,207.59} = 1.42$, $p = 0.189$).

There was a significant interaction between pre-treatment WASO sleep–wake state discrepancy and time on self-reported WASO ($F_{8,157.13} = 2.16$, $p = 0.033$; Figure 3). Bonferroni-adjusted post hoc tests found that quintile 4 (92.01–170.00 min difference between measures) had significantly higher self-reported WASO at pre-treatment than quintile 2 (−3.49 to 44.00 min; M diff. = 48.40; SEM = 14.26; $p = 0.008$, $d = 4.80$). Quintile 5 (≥ 170.01 min) had significantly higher self-reported WASO at pre-treatment than quintile 1 (≤ − 3.50 min; M diff. = 65.61; SEM = 15.83; $p < 0.001$, $d = 5.86$) and quintile 2 (−3.49 to 44.00 min; M diff. = 72.90; SEM = 14.92; $p < 0.001$, $d = 6.91$). All other group differences on self-reported WASO were not significant ($p > 0.05$). The effect did not differ after adjusting for gender ($F_{8,189.96} = 1.60$, $p = 0.128$) or age ($F_{8,193.86} = 0.87$, $p = 0.542$).

5.1 | Effects of sleep–wake state discrepancy over time on ISI scores

There was no significant interaction between pre-treatment TST sleep–wake state discrepancy and time on ISI ($p > 0.151$). A significant interaction effect of pre-treatment WASO discrepancy on ISI was observed after adjusting for age ($F_{8,134.02} = 2.07$, $p = 0.043$); however, no significant differences were found between sleep–wake state discrepancy quintiles after running post hoc tests ($p > 0.08$).

6 | DISCUSSION

This study examined the extent to which sleep–wake state discrepancy prior to CBTi predicts treatment response in a real-world clinical sample of patients with insomnia. There was no change in self-reported SOL, self-reported WASO or ISI scores for any sleep–wake state discrepancy variable at post-treatment or 3-month follow-up. The results indicate that severity of sleep–wake state discrepancy at pre-treatment does not predict treatment response as it did not impair the efficacy of CBTi.

The first-line treatment for insomnia, CBTi, is an efficacious treatment option for patients with varying levels of sleep–wake state discrepancy at pre-treatment as all participants improved with treatment. Crönlein et al. (2019) and Rezaie et al. (2018) also recently concluded that CBTi improved sleep–wake state discrepancy in their participants. The present study further extends these findings in a larger, real-world clinical sample. Despite the effectiveness of CBTi for sleep–wake state discrepancy, other comorbidities and varying features of insomnia need to be addressed.

Given the heterogenous patient population for insomnia, many psychiatric and medical disorders can co-occur (Harvey & Tang, 2012), and have the potential to impede the effectiveness of treatment. Some recent research has explored the effectiveness of CBTi when depression, anxiety and stress occur with insomnia (Sweetman et al., 2020). It is likely that some participants in this study would have presented with comorbidities that may have affected their response to CBTi; however, this information was unavailable in this dataset. We need to continue to explore the efficacy of CBTi for other comorbidities and varying features in insomnia in these large-scale studies to determine how well this treatment works across the insomnia patient population.

Despite the positive effects of CBTi, there may still be room for improvement. There were significant decreases in sleep difficulties; however, some participants continued exhibiting significant difficulty sleeping after treatment. Another study found that sleep–wake state discrepancy did not completely cease after treatment

(Crönlein et al., 2019). This is an important issue for future research, as it may be the case that lingering sleep–wake state discrepancy affects relapse risk.

A strength of this study was the sample, which was much larger than prior studies investigating sleep–wake state discrepancy in patients with insomnia, and was highly representative of a typical insomnia population based on age and sex (Appleton et al., 2022). Limitations of this study included that only a single-night sleep study was used to calculate sleep–wake state discrepancy. Having multiple nights would presumably be a better indicator of average and potential nightly variations in sleep–wake state discrepancy. Typically, there is high variability in the levels of sleeping difficulties people with sleep disorders report from night to night (Bredeli et al., 2022). Therefore, multi-night studies are warranted. Additionally, PSG was scored by traditional scoring methods, potentially ignoring important information in sleep microstructure associated with sleep–wake state discrepancy that could be more strongly related to clinical outcomes than the traditional metrics. As this was a clinical dataset, some information was missing about key demographic factors and treatment-related variables that have the potential to alter the results of the study (Sweetman et al., 2020), such as race, employment, income, education and comorbid medical conditions.

7 | CONCLUSIONS

The prediction that sleep–wake state discrepancy would impair CBTi efficacy was not supported. The most obvious finding to emerge from this study is that CBTi was still efficacious in the face of high levels of sleep–wake state discrepancy. Given the limitations of single-night PSG studies, future research could still examine multi-night assessments of sleep–wake state discrepancy to determine whether variations in discrepancy may relate to pre-treatment insomnia severity and treatment outcomes. Nonetheless, CBTi remains the treatment of choice for individuals with and without sleep–wake state discrepancy.

AUTHOR CONTRIBUTIONS

Zoe Moulder: Writing – original draft; investigation; writing – review and editing; formal analysis. **Alexander Sweetman:** Writing – review and editing; data curation. **Nicole Lovato:** Data curation; writing – review and editing. **Gorica Micic:** Writing – review and editing; data curation. **Leon Lack:** Data curation; writing – review and editing. **Hannah Scott:** Writing – review and editing; supervision.

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CONFLICT OF INTEREST STATEMENT

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DATA AVAILABILITY STATEMENT

Research data are not shared.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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