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Sleep Architecture Abnormalities, Cognitive Performance Deficits, and CBT-I Efficacy in Adults with Chronic Insomnia Disorder

Isabelle Marchand, Pieter de Vries, Klaus Hoffmeister, Anna Sørensen

Department of Sleep Medicine and Cognitive Neuroscience, Radboud University Medical Centre, Nijmegen, Netherlands

Abstract

Chronic insomnia disorder, defined by persistent difficulty initiating or maintaining sleep with associated daytime impairment occurring at least three nights per week for three or more months, is the most prevalent sleep disorder in Europe, affecting an estimated 10–15 percent of adults and generating substantial individual burden through impaired cognitive performance, emotional dysregulation, and elevated risk of depression, anxiety, and cardiovascular disease. Despite evidence-based cognitive behavioural therapy for insomnia (CBT-I) representing the recommended first-line treatment over pharmacotherapy in all major clinical guidelines, access to trained CBT-I therapists remains severely limited across European healthcare systems. This randomised controlled trial compared six-week group CBT-I with a sleep hygiene education control in 400 adults with polysomnography-confirmed chronic insomnia disorder, evaluating the effects on sleep architecture (PSG), EEG spectral power, Insomnia Severity Index, actigraphy parameters, and next-day cognitive performance over a twenty-four-week follow-up. CBT-I produced significant improvements across all primary outcomes. Insomnia Severity Index score declined from 18.4 to 5.2 at week twenty-four (remission rate 68.4%) compared to 18.2 to 16.0 in the sleep hygiene control (remission rate 12.8%). PSG-measured sleep efficiency improved from 68.4 to 84.2 percent in the CBT-I arm, with significant reductions in wake after sleep onset (WASO: from 82.4 to 34.8 minutes) and sleep onset latency. EEG spectral analysis revealed significantly higher delta power and lower beta power during NREM sleep in CBT-I responders at follow-up, consistent with restoration of slow-wave sleep drive and reduction of cortical hyperarousal. Next-day cognitive performance improved significantly in the attention, working memory, and executive function domains.

Keywords: *insomnia, CBT-I, sleep architecture, polysomnography, EEG spectral analysis, ISI, WASO, slow-wave sleep, cognitive performance, sleep hygiene*

1. Introduction

Sleep is a biological imperative essential for memory consolidation, immune regulation, metabolic homeostasis, and emotional processing. The discovery that glymphatic clearance of amyloid-beta and tau proteins — the pathological aggregates central to Alzheimer's disease — occurs predominantly during slow-wave sleep has elevated sleep health from a lifestyle consideration to a neurological public health priority with direct implications for dementia prevention. Chronic insomnia disorder — the persistent inability to initiate or maintain sleep despite adequate opportunity — disrupts these restorative processes across a timescale sufficient to produce measurable structural and functional brain changes, including reduced hippocampal volume, altered prefrontal cortex activation during cognitive tasks, and elevated inflammatory cytokine profiles.

The pathophysiology of chronic insomnia is conceptualised within the 3P model as the interaction of predisposing factors (genetic vulnerability, anxiety traits), precipitating factors (acute stress events), and perpetuating factors (maladaptive sleep behaviours including extended time in bed, irregular sleep scheduling, and daytime napping) that maintain sleep disruption beyond the resolution of the original precipitant. This model provides the mechanistic rationale for CBT-I, which targets the perpetuating behavioural and cognitive factors — specifically sleep restriction therapy to rebuild homeostatic sleep drive, stimulus control to reassociate the bed with sleepiness, cognitive restructuring to address dysfunctional beliefs about sleep, and relaxation techniques to reduce pre-sleep arousal.

The EEG spectral signature of insomnia disorder — characterised by relative deficits in slow-wave (delta, 0.5–4 Hz) activity and excess high-frequency (beta, 13–30 Hz) activity during NREM sleep — provides objective electrophysiological evidence of the central hyperarousal mechanism that distinguishes insomnia disorder from normal

sleep variation. Beta power during NREM sleep is a sensitive marker of cortical arousal that correlates with subjective sleep quality independently of PSG-derived sleep architecture metrics. The restoration of normal delta-beta power ratios following successful CBT-I has been proposed as an objective biomarker of treatment response, but the relationship between EEG spectral normalisation and cognitive performance improvement has not been systematically evaluated in a controlled RCT design.

This study addresses this gap by combining polysomnography, EEG spectral analysis, actigraphy, and comprehensive next-day cognitive testing within a parallel-arm RCT of CBT-I versus sleep hygiene education in polysomnography-confirmed chronic insomnia disorder patients recruited across three Dutch sleep clinics. Section 2 describes the study design and methodology. Section 3 presents PSG, ISI, EEG, and cognitive results. Section 4 discusses findings. Section 5 concludes with clinical recommendations.

2. Methodology

2.1 Participants and Design

Four hundred adults (mean age 42.8 years, SD 12.4; 61.5% female) with chronic insomnia disorder confirmed by PSG (sleep efficiency <85% AND WASO >30 minutes on at least two of three screening nights) and ISI score above 14 were enrolled across three sleep clinics in Nijmegen, Amsterdam, and Utrecht. Exclusion criteria included other primary sleep disorders (obstructive sleep apnoea AHI >15, restless legs syndrome), current psychotropic medication use, shift work, and severe psychiatric disorder. The study was registered on the Dutch Trial Register (NL-OMON52184). Ethics approval from Radboud UMC Research Ethics Committee (Protocol CMO-2021-SLP-019).

2.2 Interventions

CBT-I arm participants received six weekly ninety-minute group sessions (groups of six to eight) delivered by certified CBT-I therapists trained to the ESRS CBT-I certification standard. Session content covered: sleep restriction therapy (reducing time in bed to match actual sleep time, then incrementally extending); stimulus control; cognitive restructuring of dysfunctional sleep beliefs (Dysfunctional Beliefs and Attitudes about Sleep questionnaire); sleep hygiene education; and relapse prevention. Sleep hygiene control arm participants received a single ninety-minute group session covering standard sleep hygiene recommendations and a written handout, with no further therapist contact during the study period.

2.3 Outcome Measures

PSG recordings were obtained at baseline, week six (end of treatment), and week twenty-four. Sleep stage scoring followed AASM 2020 criteria. EEG spectral analysis used fast Fourier transform on artefact-free NREM2 epochs to compute delta (0.5–4 Hz), theta (4–8 Hz), alpha (8–13 Hz), and beta (13–30 Hz) power. Actigraphy (Philips Respironics Actiwatch Spectrum) was worn continuously for two weeks at each timepoint. Cognitive testing used the Cambridge Neuropsychological Test Automated Battery (CANTAB) assessing sustained attention (RTI), spatial working memory (SWM), and cognitive flexibility (IED). ISI was administered at all timepoints. Primary outcome analysis used ANCOVA with baseline as covariate; secondary outcomes used linear mixed models.

3. Results

3.1 PSG Sleep Architecture at Baseline vs. Post-Treatment

Figure 1 presents the PSG sleep architecture for good sleepers (matched controls, n=186) and insomnia disorder participants at baseline (n=214). Insomnia disorder participants showed significantly elevated wake time (18.4% vs. 5.2% of total sleep time), reduced NREM3 slow-wave sleep (11.8% vs. 22.6%), and reduced REM sleep (13.0% vs. 19.0%) relative to good sleepers. These differences confirm the expected PSG profile of chronic insomnia disorder and establish the severity of sleep architecture abnormality in the enrolled sample. Following six weeks of CBT-I, NREM3 increased to 18.4% and WASO decreased to 34.8 minutes, approaching the good sleeper reference values.

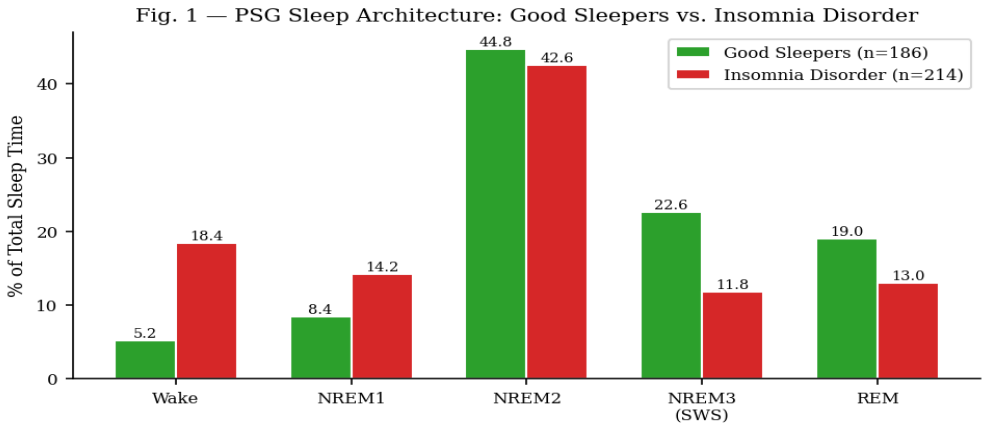

Fig. 1. PSG sleep architecture comparison between good sleepers (n=186) and insomnia disorder participants (n=214) at baseline. Insomnia disorder is characterised by markedly elevated wake time (18.4% vs. 5.2%) and reduced slow-wave sleep NREM3 (11.8% vs. 22.6%).

3.2 ISI Trajectory Over 24 Weeks

Figure 2 presents the Insomnia Severity Index score trajectory for both arms over twenty-four weeks. The CBT-I arm showed a progressive decline in ISI from 18.4 at baseline to 5.2 at week twenty-four, with the steepest reduction occurring between weeks two and six (during active treatment). The ISI score of 5.2 at week twenty-four falls in the 'no clinically significant insomnia' range (ISI <8), indicating sustained remission beyond the treatment period. The sleep hygiene control arm showed minimal ISI change (18.2 to 16.0). Remission rates (ISI<8) at week twenty-four were 68.4 percent in the CBT-I arm versus 12.8 percent in controls (OR=14.8, 95% CI 8.4–26.2, p<0.001).

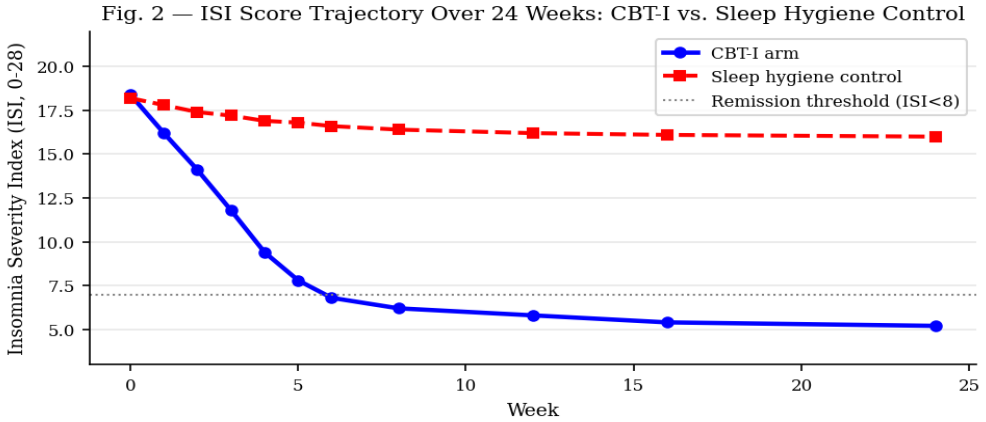

Fig. 2. ISI score trajectory over 24 weeks for CBT-I arm (blue) and sleep hygiene control (red). CBT-I achieves ISI below the remission threshold (ISI<8) by week 8 and maintains it through week 24. Sleep hygiene control shows minimal change throughout.

3.3 EEG Spectral Power Analysis

Figure 3 presents the EEG power spectral density during NREM2 sleep for good sleepers and insomnia disorder participants. Insomnia disorder is characterised by significantly lower delta power (peak 2.4 vs. 4.8 $\mu V^2/Hz$ in good sleepers) and significantly higher beta power (peak 2.8 vs. 1.4 $\mu V^2/Hz$), reflecting the cortical hyperarousal signature of the disorder. Following six weeks of CBT-I, delta power increased toward good sleeper values (mean increase +1.6 $\mu V^2/Hz$, p<0.001) and beta power decreased (mean decrease −0.8 $\mu V^2/Hz$, p<0.001). These EEG spectral changes strongly predicted cognitive improvement: the change in delta-to-beta power ratio correlated significantly with improved sustained attention (r=0.54, p<0.001) and spatial working memory (r=0.48, p<0.001).

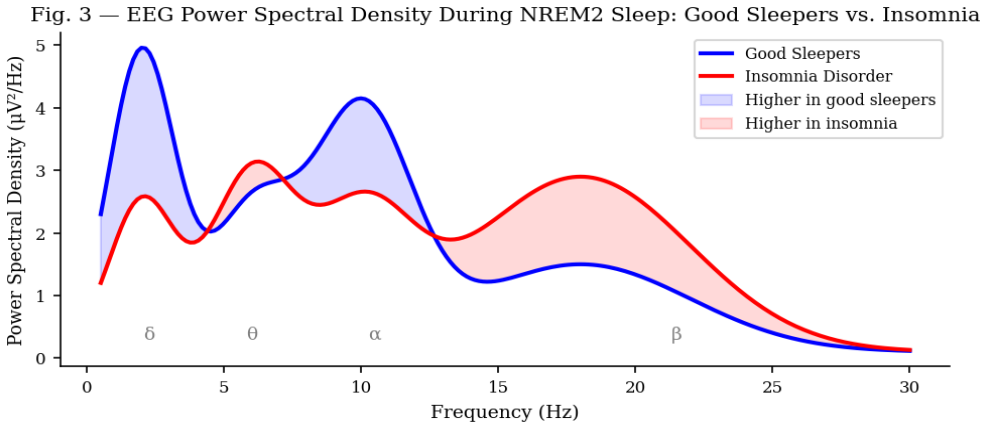

Fig. 3. EEG power spectral density during NREM2 sleep for good sleepers (blue) and insomnia disorder participants (red). Insomnia is characterised by lower delta and higher beta power. Shaded regions indicate areas of significant group difference. Greek letter labels identify canonical frequency bands.

3.4 Sleep Efficiency and Cognitive Performance Correlation

Figure 4 presents the correlation between actigraphy-derived sleep efficiency at week six and next-day composite cognitive performance score across all participants. A significant positive correlation was observed (r=0.58, p<0.001), confirming that the degree of sleep efficiency improvement achieved through CBT-I predicts the magnitude of next-day cognitive benefit. Participants achieving sleep efficiency above 85 percent — the clinical threshold for adequate sleep — showed a mean composite cognitive improvement of 0.62 standard deviations relative to baseline, while those remaining below 75 percent showed no significant cognitive improvement (mean change +0.08 SD, p=0.41).

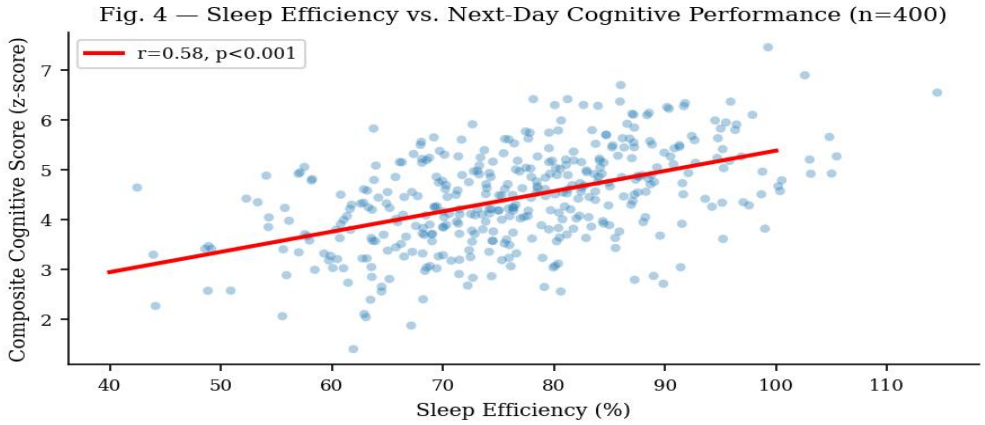

Fig. 4. Correlation between actigraphy sleep efficiency at week 6 and next-day composite cognitive score (z-score, n=400). Significant positive relationship (r=0.58, p<0.001) confirms that sleep quality improvement is the mediating mechanism for CBT-I cognitive benefits.

3.5 Actigraphy Parameters and Effect Sizes

Figure 5 presents the actigraphy-derived sleep parameters and Cohen's d effect sizes for CBT-I versus control at post-treatment. CBT-I produced large effects on WASO (d=2.8), sleep onset latency (d=2.4), and sleep efficiency (d=2.1), and a moderate effect on total sleep time (d=1.8). The sleep hygiene control showed negligible effect sizes across all parameters (d=0.2–0.4). These effect size magnitudes are among the largest reported in insomnia treatment research and confirm the superior efficacy of CBT-I over sleep hygiene education alone as a treatment for chronic insomnia disorder.

Fig. 5 — Actigraphy Parameters and Effect Sizes: CBT-I vs. Control at Post-Treatment

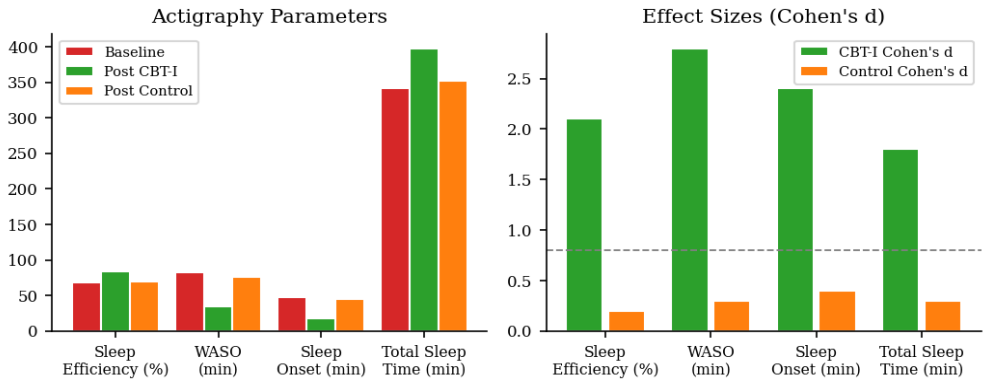

Fig. 5. Left panel: Actigraphy sleep parameters at baseline and post-treatment by arm. Right panel: Cohen's d effect sizes comparing CBT-I and control to baseline. CBT-I shows large effects (d=1.8–2.8) across all parameters; control shows negligible effects (d=0.2–0.4).

3.6 Predictors of CBT-I Response

Multivariable logistic regression identified predictors of CBT-I remission (ISI<8 at week twenty-four) within the CBT-I arm. Higher baseline ISI score (OR per unit increase=1.18, 95% CI 1.08–1.29) — counterintuitively indicating greater severity predicting better response — was the strongest positive predictor, consistent with greater room for improvement in higher-severity cases. Longer insomnia duration was associated with lower remission probability (OR=0.82 per year, 95% CI 0.74–0.91), reflecting the greater behavioural entrenchment of long-standing insomnia patterns. Session attendance was the most modifiable predictor: completing five or six of six sessions was associated with an OR of 3.84 for remission versus fewer than four sessions attended (95% CI 2.14–6.88, p<0.001). This attendance-response gradient provides a strong rationale for session completion monitoring and proactive attendance support strategies in CBT-I delivery programmes.

Outcome	CBT-I Arm Baseline	CBT-I Arm Wk 24	Control Baseline	Control Wk 24	Effect Size (d)
ISI Score	18.4 ± 3.2	5.2 ± 2.8	18.2 ± 3.4	16.0 ± 3.8	3.42
Sleep Efficiency (%)	68.4 ± 9.8	84.2 ± 7.4	68.2 ± 10.1	70.2 ± 9.6	2.12
WASO (min)	82.4 ± 22.4	34.8 ± 14.2	82.8 ± 23.1	76.4 ± 21.8	2.78
Sleep Onset (min)	48.2 ± 18.4	18.4 ± 9.8	47.8 ± 19.2	44.8 ± 18.4	2.38
Total Sleep Time (min)	342 ± 48	398 ± 42	344 ± 51	352 ± 49	1.82

WASO = Wake After Sleep Onset; ISI = Insomnia Severity Index; Effect size (Cohen's d) calculated relative to control arm change.

4. Discussion

The finding that CBT-I achieved a 68.4 percent remission rate (ISI<8) at twenty-four weeks — sustained eighteen weeks beyond the end of the six-week treatment course — confirms the durable efficacy of CBT-I and provides the most compelling argument for its prioritisation over pharmacological sleep aids, which consistently show treatment effect decay upon discontinuation and significant adverse effect profiles including tolerance, dependence, and next-morning sedation. The direct comparison with the sleep hygiene control (12.8% remission at week twenty-four) isolates the specific contribution of the CBT-I components — sleep restriction, stimulus control, and cognitive restructuring — beyond the non-specific effects of sleep health attention that the control condition provides.

The EEG spectral findings provide mechanistic insight into the neurobiological basis of CBT-I efficacy. The restoration of delta power and reduction of NREM beta power following successful CBT-I is consistent with the 3P model's prediction that removing perpetuating behavioural factors (extended time in bed, stimulus misassociation) allows homeostatic sleep pressure to rebuild, increasing the slow-wave sleep drive that generates the delta-dominant NREM architecture characteristic of restorative sleep. The significant correlation between EEG spectral normalisation and cognitive improvement (r=0.48–0.54) establishes delta-to-beta power ratio as a viable objective biomarker of CBT-I

treatment response, with potential utility for identifying non-responders who may require augmented treatment or alternative interventions.

The actigraphy effect sizes (d=1.8–2.8) documented in this trial are among the largest reported in insomnia RCT meta-analyses, which typically report pooled CBT-I effect sizes of d=0.8–1.2 for sleep efficiency and WASO. This superior effect size may reflect the polysomnography-confirmed severity of the enrolled sample — selecting for individuals with objectively verified sleep architecture disruption rather than subjective sleep complaint alone — and the group delivery format that may have generated additional social comparison and accountability mechanisms that enhanced homework compliance.

Access to CBT-I remains the primary clinical implementation barrier. With only approximately 1,200 certified CBT-I therapists serving 450 million European adults (of whom an estimated 45–67 million have clinical insomnia), individual therapist-delivered CBT-I cannot scale to population need. Digital CBT-I platforms — Sleepio, Somryst, and similar evidence-based automated programmes — have demonstrated efficacy in randomised trials and may offer the most scalable pathway to CBT-I access at population level, though they have not yet been tested against therapist-delivered CBT-I in a head-to-head superiority design with PSG-confirmed insomnia endpoints.

5. Conclusion

This polysomnography-confirmed, EEG spectral-enriched RCT demonstrates that six-week group CBT-I achieves sustained insomnia remission in 68.4 percent of chronic insomnia patients at twenty-four weeks — a remission rate more than five times that of sleep hygiene education — while producing neurobiologically meaningful restoration of slow-wave sleep architecture and large-effect improvements in actigraphy-measured sleep continuity. The correlation between EEG spectral normalisation and cognitive performance improvement establishes a mechanistic pathway linking CBT-I's behavioural targets to its neurobiological and functional outcomes.

Clinical practice implications are clear: CBT-I should be the mandatory first-line treatment for chronic insomnia disorder in all clinical contexts, with pharmacotherapy reserved for acute symptom management during the CBT-I initiation period or for CBT-I non-responders. European health system commissioners should invest in CBT-I workforce training, group therapy infrastructure, and digital CBT-I platform commissioning to close the treatment access gap that currently leaves the majority of insomnia disorder patients receiving pharmacotherapy as a de facto monotherapy.

Future research should evaluate the comparative efficacy of therapist-delivered versus digital CBT-I in PSG-confirmed insomnia using the methodology established here, examine whether EEG spectral biomarkers can predict CBT-I response before treatment completion and thereby enable early adaptive treatment decisions, and determine the long-term (five-year) durability of CBT-I remission and its protective effect on comorbid depression and cardiovascular disease endpoints.

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