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GUEST EDITORIAL

The enigma of objective and subjective measurement of response to cognitive behavioral therapy for insomnia: Call to action

Insomnia is associated with significant medical and psychiatric comorbidity, as well as increases in healthcare utilization, lost workplace productivity, and both personal and societal economic burden [1–10]. Several meta-analyses and systematic reviews of *subjective self-report* sleep improvement (primary outcome in most early studies) determined CBT-I is an effective and efficacious treatment resulting in rapid, long-lasting improvements in patient with insomnia with and without comorbidities [11–20]. Accordingly, CBT-I is considered the first line treatment for insomnia [21–24]. Fewer studies have investigated the effect of CBT-I on *objective* measures of sleep obtained from actigraphy and polysomnography (PSG). One recent meta-analysis [25] aggregated data across actigraphy and PSG and found mixed results. Given actigraphy and PSG measure different constructs (i.e., physical motion vs. brain activity) and many additional studies have utilized actigraphy-assessed sleep since 2011, it was important to have a contemporary investigation of the synthesized PSG and actigraphy outcome results for CBT-I.

In this issue, Mitchell and colleagues [26] conducted a methodologically rigorous, focused meta-analysis and systematic review examining the effects of CBT-I on objective sleep parameters as assessed via PSG and actigraphy. This review summarized the findings of 15 randomized controlled trials (RCTs) which primarily focused on CBT-I (i.e., studies were excluded if their main focus was on a comorbid psychiatric, medical, or sleep disorder in addition to insomnia). The authors examined between group-differences at post-treatment in order to focus only on the effect of CBT-I on objective sleep parameters including sleep onset latency (SOL), wake after sleep onset (WASO), sleep efficiency (SE) and total sleep time (TST).

Polysomnography results

The main findings of the PSG review indicated CBT-I did not result in any significant differences between treatment and control groups on any sleep parameters. As mentioned above, few studies ($n = 5$) examined PSG-derived sleep parameters and the CBT-I treatment arm sample sizes were generally small. There was significant heterogeneity among effect sizes for SE and a trend ($p = 0.068$) for TST. Given the high heterogeneity, more conservative random effects models were used. Unfortunately, random effects models tend to be biased by studies with very small sample sizes, which in the current study also had the most disparate effects. For instance, the study with the smallest sample [27] showed CBT-I had worse SE at post-treatment than control, and this study also found the largest negative differences for TST. It would have been interesting to see the outcomes had underpowered studies (e.g., $n < 25$) been excluded from analyses to prevent any spurious results from obscuring significant differences (e.g., Type II error). However, there were not enough studies to warrant the elimination of even a single one due to sample size.

Actigraphy results

Considerably more studies examined actigraphy-derived sleep parameters ($n = 13$). The main findings of the actigraphy review indicated CBT-I produced significantly shorter SOL and lower TST (approximately 30 m) for treatment compared to control, with no differences observed for WASO or SE. For these studies, there was again significant heterogeneity among effect sizes for SOL and TST, and a trend for SE. Given the much larger number of studies to pull from, the bias of the few studies with small sample sizes was minimized for these analyses, so one can have more confidence in these results.

The SOL results are expected but the TST results at first may seem surprising. The goal of CBT-I is to improve sleep, with which a decrease in TST is not typically associated. However, there are several compelling potential reasons for this finding. First, as the authors noted, CBT-I may actually result in decreases in objective TST as the goal of sleep restriction initially is to consolidate sleep with the potential side effect of temporarily reducing TST [26]. However, this is somewhat refuted by the authors' observation that follow-up data support a sustained effect of reduced actigraphy-derived TST. Another potential explanation is related to a primary goal of CBT-I: to reduce excessive time in bed (TIB). Previous work has indicated actigraphy typically overestimates total sleep time compared to PSG by an average of 0.8 h [28]. It then follows that if individuals in the control condition are spending excessive TIB, actigraphy may overassign quiescent non-sleep periods as sleep whereas their CBT-I-treated counterparts now have a shorter TIB (i.e., less opportunity for actigraphy to overestimate TST). Unfortunately no data on TIB was reported in the current review, so this remains a question.

Subjective results

Although it was not a primary objective of this review, the authors also reported on subjective (i.e., sleep diary-derived) sleep parameters ($n = 17$; some studies contributed multiple arms to the analyses). There was again significant heterogeneity among effect sizes for SOL, WASO, and SE, which raises concerns about the

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equity of types of CBT-I used across studies as well as other factors such as the quality of therapists and supervision, treatment fidelity, and patient adherence. Regardless, the findings indicated CBT-I produced significantly lower SOL and WASO and significantly higher SE, with no significant difference for TST. These results closely replicated those of other previous meta-analyses, and served as a strong validity marker that the results of the included studies are generally representative of the CBT-I literature which did not measure objective sleep.

Limitations and future directions

The authors observed several limitations in this review. The primary limitation was an insufficient sample of studies that used PSG. For historical perspective, Morin et al. [29] performed the first study to utilize PSG-assessed sleep as an objective outcome in 1993; only four additional studies reported on PSG outcomes in the three decades following Morin's study. Conversely, Friedman et al. [27] published the first study examining actigraphy outcome data in 2000, followed by 12 additional studies in the next two decades. These discrepancies in available data may be in part due to a clause in a panel of insomnia expert *Recommendations for a Standard Research Assessment of Insomnia* [30] in 2006 deeming objective measure of sleep is "Essential" in efficacy trials, with actigraphy *or* PSG as a dependent measure. Given the cost differential in assessment type, with PSG costing thousands of dollars per night to complete, score and interpret, whereas a single reusable activity monitor typically costs less than $1000, it unsurprising more studies use actigraphy than PSG.

However, if the field is ever going to fully understand the discrepancy between objective and subjective sleep responses to CBT-I, it is important that investigators and funders continue to utilize PSG/EEG as an outcome measure in future RCTs of CBT-I. Future studies should also include additional objective sleep constructs such as sleep architecture (e.g., delta and rapid eye movement sleep), fragmentation, and circadian biomarkers (e.g., amplitude and timing of temperature, hormonal and inflammatory markers) that might be more sensitive to the changes experienced by patients with insomnia and which are the latent constructs being assessed by self-report instruments such as diaries and questionnaires.

It is also important to include ambulatory PSG/EEG in future studies of CBT-I. In the current study, only two of the studies utilized ambulatory PSG for the assessment of outcomes. CBT-I specifically focuses on making changes to the bedroom environment, patient behaviors, and household conditions during the night. Ambulatory PSG is likely better at detecting major differences between CBT-I and control group behaviors and environments. These differences would be obscured by bringing both groups into the sterile sleep laboratory, where the control group's sleep cannot be disturbed by the poor stimulus control common in their homes. Conversely the CBT-I group might be less likely to follow stimulus control instructions, such as leaving the bed/bedroom if awake for more than 15 min.

Another important area of objective and subjective sleep that should become a standard outcome variable in future RCTs is examination of intraindividual or night-to-night variability in sleep. Most CBT-I trials focus primarily on mean differences (averaged over 1–14 nights) in sleep parameters between groups [31,32]. Limiting examination to mean sleep levels ignores the dynamic and complex night-to-night variability seen in the sleep of people with insomnia that is presumably improved by CBT-I. For instance, two insomnia patients (A and B) might have sleep that varies from 4 to 9 h, for an average of 6.5 h, per night at baseline. After CBT-I, patient A might end up with a consistent 6 h per night, while patient B continues to have variable sleep for 6.5 h per night. Patient A would probably consider the treatment a success, even though they are averaging a half hour less per night, because they do not have any nights where they are sleeping 2.5 h less (e.g., the 4 h night). This is especially important when one considers intraindividual severity is closely related to many of the daytime functioning and health complaints common in people with insomnia, such as depression, stress, diabetes, and heart conditions [32].

The authors observed other limitations in this review including heterogeneity in the literature with regard to methodology and reporting of objective sleep methods, insufficient information to perform subgroup analyses (e.g., participants taking hypnotic medications), and narrow inclusion criteria for the review which may limit generalizability. In particular, the authors noted inconsistency in reporting of PSG and actigraphy scoring, which makes comparison across studies exceedingly difficult. The authors also suggested few studies reported adequate blinding procedures, especially in the scoring of objective sleep data, which raises concern for biased outcomes.

Another particular limitation was the heterogeneity of "CBT-I" across studies, which could substantially impact the results. There may be substantial differences between, for example, two sessions of just sleep restriction and sleep hygiene, four sessions of brief behavioral therapy for insomnia, and six sessions of full, multicomponent CBT-I, all of which could have been included by the current review's inclusion criteria. Combining results from each of these treatments is likely to water down overall effect sizes artificially. It is also of note that the two studies that did not mention use of a manualized treatment or assessment of therapists' fidelity to the treatment were two of the studies with the most discrepant results [27,33].

Conclusions

Despite the substantial impact of insomnia, it remains undertreated. When identified, it is most often treated in primary care with pharmacotherapy, rather than the first-line recommendation, CBT-I [21]. Pharmacotherapy is associated with risks of dependence, tolerance, and poorer quality sleep, whereas CBT-I results in better long-term outcomes, no drug dependence or polypharmacy risk, virtually no medical risk, and potential cost savings.

It is important to remember patients presenting to therapy ultimately do not care what their "*objective*" results show, but are more concerned about their "*subjective*" experience of sleep and perhaps more importantly the effect their insomnia is having on their subjective daytime functioning. Although there has been an important push within mental health interventions for "*objective*" outcomes, it is important to remember just because a measure is considered "*objective*" does not mean it is an appropriate assessment of the construct (e.g., insomnia) it proposes to measure. Indeed, some have called for "responsiveness" or "sensitivity to change" to be considered as a psychometric characteristic separate from reliability and validity [34,35]. It is entirely plausible that the current objective measures (e.g., TST, SE) being used are not sensitive enough or appropriately focused to detect the experience of insomnia patients are self-reporting. Therefore, it is very important that the results of the current study *not* be interpreted as indicating lack of objective improvement after CBT-I. Instead, it is a call to action for CBT-I researchers to design future RCTs that include the following:

- Objectively assess sleep with both actigraphy *and* ambulatory PSG

- Expand our definition of objective sleep to measures that may be more sensitive to response (e.g., EEG power, architecture, fragmentation, circadian rhythms, and variability)
- Adequately power to find smaller effects and minimize spurious results
- Assess and report rigorous clinical trial details (e.g., fidelity and adherence assessment)
- Focus less on minimal intervention necessary for change and focus more on testing the most powerful interventions with the highest quality therapists

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.smrv.2019.08.003.

References

[1] Zee PC, Badr MS, Kushida C, Mullington JM, Pack AI, Parthasarathy S, et al. Strategic opportunities in sleep and circadian research: report of the joint task force of the sleep research society and American academy of sleep medicine. Sleep 2014;37(2):219.
[2] Taylor DJ, Pruiksma KE, Hale WJ, Kelly K, Maurer D, Peterson AL, et al. Prevalence, correlates, and predictors of insomnia in the US Army prior to deployment. Sleep 2016;39(10):1795–806.
[3] Rosekind MR, Gregory KB. Insomnia risks and costs: health, safety, and quality of life. Am J Manag Care 2010;16(8):617–26.
[4] Leger D, Scheuermaier K, Philip P, Paillard M, Guilleminault C. SF-36: evaluation of quality of life in severe and mild insomniacs compared with good sleepers. Psychosom Med 2001;63(1):49–55.
[5] Simon GE, VonKorff M. Prevalence, burden, and treatment of insomnia in primary care. Am J Psychiatry 1997;154(10):1417–23.
[6] Taylor DJ, Kelly K, Kohut ML, Song K-S. Is insomnia a risk factor for decreased influenza vaccine response? Behav Sleep Med 2016:1–18.
[7] Vgontzas AN, Liao D, Pejovic S, Calhoun S, Karataraki M, Basta M, et al. Insomnia with short sleep duration and mortality: the Penn State cohort. Sleep 2010;33(9):1159–64.
[8] Walsh JK, Engelhardt CL. The direct economic costs of insomnia in the United States for 1995. Sleep 1999;22(Suppl. 2):S386–93.
[9] Ozminkowski RJ, Wang S, Walsh JK. The direct and indirect costs of untreated insomnia in adults in the United States. Sleep 2007;30(3):263–73.
[10] Godet-Cayre V, Pelletier-Fleury N, Le Vaillant M, Dinet J, Massuel MA, Leger D. Insomnia and absenteeism at work. Who pays the cost? Sleep 2006;29(2):179–84.
[11] van Straten A, van der Zweerde T, Kleiboer A, Cuijpers P, Morin CM, Lancee J. Cognitive and behavioral therapies in the treatment of insomnia: a meta-analysis. Sleep Med Rev 2018;38:3–16.
[12] Trauer JM, Qian MY, Doyle JS, Rajaratnam SM, Cunnington D. Cognitive behavioral therapy for chronic insomnia: a systematic review and meta-analysis. Ann Intern Med 2015;163(3):191–204.
[13] Montgomery P, Dennis JA. Cognitive behavioural interventions for sleep problems in adults aged 60+. Cochrane Database Syst Rev 2003;(1).
[14] Taylor DJ, Pruiksma KE. Cognitive behavioral therapy for insomnia in psychiatric populations: a systematic review. Int Rev Pyschiatr 2014;26:205–13.
[15] Geiger-Brown JM, Rogers VE, Liu W, Ludeman EM, Downton KD, Diaz-Abad M. Cognitive behavioral therapy in persons with comorbid insomnia: a meta-analysis. Sleep Med Rev 2015;23:54–67.
[16] Koffel EA, Koffel JB, Gehrman PR. A meta-analysis of group cognitive behavioral therapy for insomnia. Sleep Med Rev 2015;19:6–16.
[17] Murtagh DRR, Greenwood KM. Identifying effective psychological treatments for insomnia: a meta-analysis. J Consult Clin Psychol 1995;63(1):79–89.
[18] Smith MT, Perlis ML, Park A, Smith MS, Pennington J, Giles DE, et al. Comparative meta-analysis of pharmacotherapy and behavior therapy for persistent insomnia. Am J Psychiat 2002;159(1):5–11.
[19] Morin CM, Culbert JP, Schwartz SM. Nonpharmacological interventions for insomnia: a meta-analysis of treatment efficacy. Am J Psychiat 1994;151(8):1172–80.
[20] Irwin M, Cole JC, Nicassio PM. Comparative meta-analysis of behavioral interventions for insomnia and their efficacy in middle-aged adults and in older adults 55+ years of age. Health Psychol 2006;25(1):3–14.
[21] Qaseem A, Kansagara D, Forciea MA, Cooke M, Denberg TD. Management of chronic insomnia disorder in adults: a clinical practice guideline from the American College of Physicians. Ann Intern Med 2016;165(2):125–33.
[22] Wilson SJ, Nutt DJ, Alford C, Argyropoulos SV, Baldwin DS, Bateson AN, et al. British Association for Psychopharmacology consensus statement on evidence-based treatment of insomnia, parasomnias and circadian rhythm disorders. J Psychopharmacol 2010;24(11):1577–601.
[23] Riemann D, Baglioni C, Bassetti C, Bjorvatn B, Dolenc Groselj L, Ellis JG, et al. European guideline for the diagnosis and treatment of insomnia. J Sleep Res 2017;26(6):675–700.
[24] National Institutes of Health. National institutes of health state of the science conference statement on manifestations and management of chronic insomnia in adults, June 13–15, 2005. Sleep 2005;28(9):1049–57.
[25] Okajima I, Komada Y, Inoue Y. A meta-analysis on the treatment effectiveness of cognitive behavioral therapy for primary insomnia. Sleep Biol Rhythm 2011;9(1):24–34.
[26] Mitchell L, Bisdounis L, Ballesio A, Omlin X, Kyle S. The impact of cognitive behavioural therapy for insomnia on objective sleep parameters: a meta-analysis and systematic review. Sleep Med Rev 2019;47:90–102.
[27] Friedman L, Benson K, Noda A, Zarcone V, Wicks DA, O'Connell K, et al. An actigraphic comparison of sleep restriction and sleep hygiene treatments for insomnia in older adults. J Geriatr Psychiatry Neurol 2000;13(1):17–27.
[28] Lauderdale DS, Knutson KL, Yan LL, Liu K, Rathouz PJ. Self-reported and measured sleep duration: how similar are they? Epidemiology (Cambridge, Mass) 2008;19(6):838–45.
[29] Morin CM, Kowatch RA, Barry T, Walton E. Cognitive-behavior therapy for late-life insomnia. J Consult Clin Psychol 1993;61(1):137–46.
[30] Buysse DJ, Ancoli-Israel S, Edinger JD, Lichstein KL, Morin CM. Recommendations for a standard research assessment of insomnia. Sleep 2006;29(9):1155–73.
[31] Van Dongen H, Vitellaro KM, Dinges DF. Individual differences in adult human sleep and wakefulness: leitmotif for a research agenda. Sleep 2005;28(4):479–96.
[32] Bei B, Wiley JF, Trinder J, Manber R. Beyond the mean: a systematic review on the correlates of daily intraindividual variability of sleep/wake patterns. Sleep Med Rev 2016;28:108–24.
[33] Lovato N, Lack L, Kennaway DJ. Comparing and contrasting therapeutic effects of cognitive-behavior therapy for older adults suffering from insomnia with short and long objective sleep duration. Sleep Med 2016;22:4–12.
[34] Guyatt G, Walter S, Norman G. Measuring change over time: assessing the usefulness of evaluative instruments. J Chronic Dis 1987;40(2):171–8.
[35] Guyatt GH, Deyo RA, Charlson M, Levine MN, Mitchell A. Responsiveness and validity in health status measurement: a clarification. J Clin Epidemiol 1989;42(5):403–8.

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