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Editorial

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Untangling the mechanisms of sleep restriction therapy

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Sleep restriction therapy (SRT), potentially the most active component of cognitive behavioral therapy for insomnia [1], has emerged as a clinically and cost-effective standalone treatment [2–4]. While efficacious, the mechanisms through which SRT improves insomnia are not fully understood. Maurer et al. [5] proposed the "Triple-R" model to explain the therapeutic effects of SRT, suggesting it principally operates by restricting time in bed, regularizing sleep–wake timing, and reconditioning the bedroom–sleep association. While these three "R's" represent proximal intervention targets, a cascade effect on underpinning and interacting cognitive-behavioral and physiological processes is suggested to address several different features of insomnia disorder. The model formulates a number of testable hypotheses but empirical support comes from a relatively small number of recent studies (e.g. [6–9]), chiefly because most research on SRT focuses on outcome rather than process and mechanism.

In this issue of *SLEEP*, Looman et al. [10] shed new light on change during SRT. They report a secondary analysis of a randomized controlled trial comparing 6 weeks of telephone-guided SRT ($n = 76$) with a sleep-diary control arm ($n = 71$). The main trial showed that SRT demonstrated superior clinical effects at post-treatment relative to the control group [3]. Using network intervention analysis (NIA), the authors modeled individual insomnia symptoms and related process variables across the intervention period. NIA estimates the direct (and potential indirect) effects of treatment allocation on specific variables within a network, while adjusting for the inter-relations between all other variables [11]. By using NIA for each of the 6 treatment weeks, the analysis illustrates how symptoms and process variables evolve over time. The authors assessed two different networks: one where the nodes represented the seven items of the insomnia severity index, and the other where the nodes represented proxies of process variables, including markers of SRT adherence (e.g. time-in-bed, TIB; bed/rise-time variability), insomnia maintenance factors (e.g. arousal, TIB, safety behaviors), and clinically relevant outcomes (e.g. sleep onset latency, SOL; Epworth sleepiness scale, ESS).

With respect to symptoms, the authors found that SRT led to direct, rapid, and sustained reduction in nocturnal symptoms (difficulty initiating and maintaining sleep), while also increasing interference with daily function in the first half of treatment. In the process network, TIB and bed/rise-time variability were reduced in the SRT group throughout treatment, but direct effects on diary-reported SOL and pre-sleep arousal (PSAS) tended to emerge later in treatment. The NIA methodology cannot resolve causal ordering between the two networks, but it seems likely that adhering to SRT recommendations—that is, reducing sleep opportunity and following prescribed bed and rise-times—drives rapid improvement in nocturnal symptom severity. The cost of initial sleep loss, however, might be a transient worsening of daytime function.

These findings broadly align with prior work comparing SRT to regularization of TIB [6], and to sleep compression [12], where SRT showed more rapid change in insomnia severity and sleep continuity but also greater side-effect burden. The somewhat delayed (direct) effect on sleep latency and arousal was surprising given that other work has shown improvement within the first few weeks. It should be noted that the PSAS total score was used to index self-reported arousal, which includes both somatic and cognitive components; SRT typically has a more pronounced effect on cognitive arousal [2, 6]. There was evidence of a potential indirect effect of SRT on SOL via change in TIB, which is a plausible sequence even if directionality cannot be inferred from process-to-process connections. A more general reflection on the NIA approach is that variables exhibiting large between-group differences in conventional univariate analyses (see fig. 1; Looman et al. [10]) were more likely to show direct treatment effects when interrogated within network models, presumably in part because their adjustment overwhelms detection of potentially smaller direct effects for other variables.

This interesting study showcases how network intervention analysis might be applied to understanding SRT-related change, but it also brings into sharp focus the inherent complexity of

elucidating behavioral treatment mechanisms. Looman et al. confirm that reduction in TIB and bed/rise-time variability are likely "activating mechanisms" [13] but the subsequent downstream cascade, which is potentially bi-directional, multi-variate, and non-linear [14], requires careful mapping across multiple levels of explanation. For example, what is the acute and long-term impact of SRT on light exposure, circadian phase/phase angle, sleep regularity, continuity, structure, and microstructure? These intermediate variables are presumed to lie on the path between implementation and insomnia improvement yet have received limited attention [5].

It is also possible that what drives short-term improvement in symptoms may differ from change processes necessary for the long-term maintenance of treatment effects. For example, acute reduction of TIB leads to initial sleep loss, which via increased sleep pressure enhances sleep consolidation, sleep quality, and improves severity of symptoms [6]. But if a straightforward sleep debt account were to apply, then subsequent TIB extension (and recovery of "objective" TST) would be expected to lead to the reappearance of insomnia symptoms. This does not appear to be the case, at least for average treatment effects. Does the potentiation and discharge of sleep pressure during SRT fundamentally alter the (im)balance between sleep-promoting and wake-promoting neural circuitry, leading to enduring effects on sleep structure and insomnia symptoms beyond the restriction phase; do patients, through SRT, (re)discover their true sleep need and optimize the timing of the sleep–wake cycle, alongside other zeitgebers; or might acute implementation of SRT instead lead to powerful cognitive change (perhaps through exposure-based mechanisms) to drive a re-framing of insomnia, irrespective of long-term change in sleep–wake regulatory processes? Perhaps all scenarios could apply, contingent on the baseline drivers of different insomnia presentations.

It is abundantly clear that we need better multi-level descriptions of change during SRT and the study by Looman et al. contributes to this endeavor. Such information might help prioritize further variables for exploratory network analysis as well as mediation hypothesis testing. To be confident about the specificity of pathways, SRT could be tested against comparator conditions that isolate specific intervention features and control for non-specific effects (e.g. time spent with therapist, expectation of benefit, and side-effects). Formal evaluation of SRT against mechanistic understanding of sleep and circadian regulation would also be of value. Ultimately, "back-translation" of SRT has the potential to reveal key mechanisms that might lead to better personalization of treatment as well as the development of new targeted therapies.

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