CBT-I and the short sleep duration insomnia phenotype: a comment on Bathgate, Edinger and Krystal

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Comment on: Bathgate CJ, Edinger JD, Krystal AD. Insomnia Patients with Objective Short Sleep Duration have a Blunted Response to Cognitive Behavioral Therapy for Insomnia. Sleep 2016. [Epub ahead of print].

Although the DSM-5 and the ICSD-3 do not discriminate among insomnia types or subtypes anymore, it appears that some specific insomnia phenotypes remain important to study. One of them is the object of the present paper: insomnia with short sleep duration. Since Vgontzas and colleagues (1) put forward a heuristic model of two insomnia phenotypes based on objective sleep duration, they have suggested that insomnia with short sleep duration is the most severe biological phenotype of insomnia, and research in this area has been blooming. The Penn State group has studied the impact of this phenotype on adolescents and its association with depression risks and inflammation (2-4). A recent review by Fernandez-Mandoza (5) also suggested that besides increased physiological hyperarousal and cardiometabolic and neuropsychiatric risks, insomnia with short sleep duration may even respond differently to treatment compared to other insomnia phenotypes.

Cognitive Behavioural Therapy for Insomnia (CBT-I) has been used in numerous studies and its long-term efficacy has been recognized (6-9). CBT-I is the treatment of choice for insomnia (10). Despite this, since insomnia with short sleep duration is associated with physiological arousal, perhaps patients with this condition would benefit more from pharmacotherapy than CBT-I.

It is unknown whether individuals with insomnia presenting with short sleep duration respond differently to CBT-I than those presenting with more normative sleep duration. This is precisely what Bathgate, Edinger and Krystal (11) aimed to study. From data collected as part of another study, they systemically divided 60 individuals suffering from insomnia (DSM-IV criteria) into two groups according to their sleep duration: short (less than 6 hours, N=30) and normal (6 hours and more; N=30) based on actigraphy (PSG data being unavailable). Once groups were aggregated, they received CBT-I treatment (1, 2, 4 or 8 sessions). CBT-I mainly addressed dysfunctional beliefs and attitudes and behavioral strategies (stimulus control). Pre and post measures as well as 6 months follow-up data were obtained using questionnaires, sleep diaries, and actigraphy. The results were in line with previous hypotheses: individuals suffering of insomnia and sleeping 6 hours or less, according to actigraphy data, were less likely to be responsive to CBT-I than those sleeping more than 6 hours; additionally, this effect persisted through follow-up. Thus, insomnia coupled with short-sleep duration might be one of the phenotypes which presents more challenges when treated with CBT-I. Since the individuals of the study were all 'pure' insomnia sufferers, no other disorders could be linked to the limited responsiveness of individuals to the most common non-pharmacological treatment for insomnia: CBT-I.

The results from Bathgate *et al.* provide one more reason, besides lack of adherence or challenging disorders, why some individuals do not respond well to treatment.

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Whilst the seminal study by Ong, Kuo and Manber (12) does suggest a drop-out rate between 0% and 8% within the context of two RCTs, they also point to drop out rates ranging from 9.7% to 38.3% in clinical settings. Further, in their own analysis, 39.96% of subjects dropped out. These high levels are especially seen in clinical settings (12). Maybe these drop-out rates included short sleep duration insomnia patients. The question now is: What do we do with insomnia patients presenting with short sleep? Do we still consider them for CBT-I or would they benefit more from pharmacotherapy? Or would a modification to CBT-I be required? Of note, neither CBT-I nor insomnia pharmacotherapy have shown a consistent ability to increase sleep duration.

Future research should examine treatment approaches in this group of insomnia patients. For example, a study could verify whether these limitations are also seen with pharmacotherapy or with combined therapy (pharmacotherapy and CBT-I). Because individuals suffering from insomnia and presenting with a short-sleep duration show increased health risks compared to individuals suffering from insomnia and sleeping 6 hours and more, it should be a priority to find a beneficial treatment for these short-sleepers. In addition, if insomnia with short-sleep duration is biologically determined, its neurobiological underpinnings should be studied so to adapt treatment to this population. Meanwhile, in current CBT-I trials, it might be advisable to take into account baseline sleep duration when treatment efficacy is the main outcome.

Curiously, Bathgate et al. claimed that CBT-I is not efficacious for those suffering sleep-state misperception (i.e., paradoxical insomnia). To our knowledge, there are few studies examining treatment specifically among patients with this condition. One study by the Lichstein group (13), although limited by the number of participants, did show that individuals suffering from paradoxical insomnia, when confronted to their own PSG data, more readily accept the fact they are grossly misestimating their sleep. In addition, personally addressing PSG data decreases hyperarousal linked to misperception and then, insomnia patients may be more receptive to CBT-I. It is not clear if the allusion to the lack of effectiveness of CBT-I treatment in the Bathgate et al. (11) paper refers to clinical data (from practice) or published research; perhaps it simply reflects a lack of efficacy data. Misperception of sleep is quite a challenge and its underlying mechanisms remain poorly studied (14). Still, cognitive therapy, which by definition decreases emotional arousal and cognitive arousal (this latter being itself linked to cortical arousal) might plausibly be effective in diminishing sleep misperception. Bathgate et al. suggest that this is not the case, despite a sufficient justification for this claim. It is also still unknown the extent to which sleep misperception contributes to the limited effectiveness of CBT-I in general clinical trials. Paradoxical insomnia, to some extent, thus also appears as a phenotype unresponsive to CBT-I according to Bathgate et al. It would be interesting to develop a protocol taking into account both phenotypes. More research should be devoted to also understanding the misperception of sleep as it may be one of the limitation of the efficacy of current insomnia treatments.

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Footnote

Conflicts of Interest: Michael Grandner declares having received funds from FitBit and CurAegis Technologies. Jason G. Ellis is the author of The One Week Insomnia Cure by Vermillion and is the director of Sleep Research and Consulting Ltd. Celyne H. Bastien has no conflict of interest.

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