Towards an Improved Neuropsychology of Poor Sleep?


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PATIENTS WITH PERSISTENT INSOMNIA COMPLAIN NOT ONLY OF SLEEP PROBLEMS BUT ALSO OF DAY-TIME DEFICITS, WHICH THEY LINK TO THEIR POOR sleep. This 24-hour perspective on the impact of insomnia, of course, is enshrined in DSM and ICSD criteria, although the exact relationship between such sleep and wake experiences remains poorly understood, even at the phenomenological level. In the most common primary insomnia sub-type, Psychophysiological Insomnia, we expect these night-time and daytime patient experiences to be both objectively verifiable and demonstrably associated. Therein has lain a problem, because even this highly recognisable behavioural prototype has proven somewhat stubborn to reveal its neuropsychological and neurocognitive underpinnings. More particularly, some have doubted the existence of an objective deficit in insomnia. Perhaps insomnia is no more than some form of pervasive sleep hypochondriasis?

But in truth we have only just begun to scratch the surface, and there have been methodological problems, with research studies using differing methodologies yielding, unsurprisingly, differing results. A fair criticism is that previous studies have comprised small and poorly characterised samples, and applied relatively blunt instruments, largely incapable of the necessary dissection of cognitive performance in insomnia.

Results from the study by Edinger and colleagues in this issue of Sleep are likely to make an impact for several reasons. First, they have recruited the largest sample size to date; second, primary insomnia participants were very well screened; third, they used a gender and age-matched control population; and fourth, the study protocol is comprehensive (3 PSG nights, 4-trial MSLTs, battery of performance tasks, and appropriate subjective measurements). Importantly, the study incorporates some manipulation of the complexity of psychomotor tasks, making it more likely that there is sufficient measurement sensitivity to detect the subjective impairment characteristic of insomnia.

In our view, their essentially confirmatory analyses using PSG (people with insomnia had poorer objective sleep than controls, but their self-reports overestimated the symptoms) and MSLT (longer duration in insomnia despite higher levels of subjective sleepiness) lend considerable authority to the study’s main findings. The authors report that people with insomnia do exhibit performance deficits (longer response latencies, and greater response variability) on a range of reaction time tests, specifically those that were, in their words, more “challenging.” Here they are referring to switching of attention tests, which invoke parallel cognitive processes (intact attention, concentration, response inhibition, and rapid decision making). Edinger at al suggest that these may be useful for tapping into typical subjective report because they “…may closely approximate the deficits PI sufferers present clinically when they complain of inability to concentrate and a general lack of mental sharpness.”

Such findings appear to fit well with the idea of “compensatory effort.” Tasks that are increasingly challenging or complex may be more capable of unmasking performance decrements. That is, the extra effort recruited by people with insomnia to maintain normal functioning, in these circumstances, becomes exhausted. By comparison, studies that use relatively simple tasks (and/or non-constant routines) may not be capable of documenting deficits in baseline performance between normal and poor sleepers, nor indeed treatment gains post intervention. This is classic Type 2 error. We should be wary, therefore, because the daytime impact of insomnia on cognitive performance will not be tested if we design studies expecting that higher order deficits of the type identified in narcolepsy or obstructive sleep apnea are what we ought to find. We suggest rather that the field may benefit from explorations into the mediating role of effort in performance maintenance, with potential research strands including self-report mental effort during testing as well as compensatory recruitment of cerebral resources, as has been documented in OSA patients.

Edinger and colleagues also found that objective sleep measures were more strongly related to participants’ daytime performances than were the subjective measures. It is difficult to know what to make of this finding. It could simply be an artefact of method variance that objective outcomes are more likely to co-vary with one another, as are subjective ones, with objective-subjective correlations being inevitably lower. Nevertheless, it is important to show in a disorder that is usually diagnosed and treated solely on its self-reported characteristics, that it (a) does have measurable substrates, and (b) that these...
substrates interrelate. More work is needed here, to explore the full matrix, of sleep (objective, subjective) by performance (objective, subjective) using sensitive, gold-standard measurements in each domain.

This is a potentially influential paper that should be read, and it is likely to become highly cited. Apart from being the best available study in the field, it points a way forward for future hypotheses-testing studies about the exact nature of the performance deficits in insomnia. However, we need to develop a much more sophisticated neuropsychology of insomnia. Perhaps an attention-switching deficit can capture the clinical complaint. Can we now use information-processing theory to better explain the daytime aspects of the disorder?

REFERENCES