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Short sleep duration and insomnia associated with hypertension incidence

Michael A Grandner and **Michael L Perlis**
Behavioral Sleep Medicine Program, Department of Psychiatry, Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA, USA; and are at the Center for Sleep and Circadian Neurobiology, University of Pennsylvania, Philadelphia, PA, USA

Michael A Grandner: grandner@upenn.edu

Insomnia and short sleep duration are potentially separate but overlapping constructs, both of which may confer independent risk for adverse health outcomes. Accordingly, it is important to consider both in the context of epidemiologic research. The paper by Meng *et al*.[1] adopts precisely this approach when exploring if and how insomnia and/or short sleep duration may confer risk for hypertension.

Insomnia is a common condition. It is estimated that approximately 30% of the population suffers from some symptom of insomnia, and approximately 5–15% of the population likely meets criteria for an insomnia disorder.[2] Although this statistic has been widely cited and replicated for over several decades, it has not caused much alarm. Within the lay, medical and psychiatric communities, insomnia was frequently conceived of as a symptom rather than a disorder, or secondary to another condition that was the root cause and therefore should be the focus of treatment (for example, depression, chronic pain). However, the scientific literature has, over the last several years, demonstrated that insomnia is better characterized as an independent disorder. (This change is being reflected in DSM-5.) Further, although substantial research has established insomnia as an important risk factor for mental illness[3] and functional limitations,[4] other health risks of insomnia remained largely unexplored. Before 2006, there were only a very small number of papers that address cardiometabolic implications of insomnia. Since that time, there have been a number of studies that have demonstrated that insomnia is a disorder with medical consequences beyond mental health and quality of well-being.

The meta-analysis by Meng *et al*.[1] provides a valuable contribution to this literature. The authors examined risk of hypertension incidence associated with the cardinal symptoms of insomnia: difficulty initiating sleep (initial insomnia), difficulty maintaining sleep (middle insomnia) and difficulty with early morning awakenings (late insomnia). The authors identified seven unique studies that assessed insomnia symptoms and hypertension incidence. Overall, initial insomnia was not specifically related to hypertension incidence. However, both middle and late insomnia were associated with incident hypertension. Specifically, the relative risk ratios (RRR) for middle and late insomnia were RRR=1.20 (95% confidence interval (CI) 1.06–1.46) and RRR=1.14 (95% CI 1.07–1.20), respectively. Although these effects are generally modest, they are not surprisingly so, as there are many factors that predict hypertension incidence that may (or may not) be more directly related to

CONFLICT OF INTEREST
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causal factors (for example, obesity, genetics, age, sex). Despite this, insomnia was a robust risk factor.

In addition to examining insomnia, the authors examined short sleep duration. Although many individuals with short sleep duration report symptoms of insomnia,[5] these are separate constructs. Short sleep duration itself does not necessarily constitute a complaint—it frequently represents a portion of the normal variation of sleep duration experienced in the population. However, there is much discussion about how and why many people sleep less than 7–8 h, and to what degree this is detrimental.[6] Epidemiologic studies, which aggregate all 'short sleepers' (typically defined as <6h per night), have consistently found that this group is at increased risk of a number of adverse outcomes, including obesity, cardiovascular disease, diabetes and functional impairments.[6,7] As awareness of the importance obtaining adequate sleep increases in the public health domain, studies are needed to extend beyond the traditional cross-sectional design and explore longitudinal associations to begin to investigate causality.

Meng *et al*.,[1] in this issue, identified seven longitudinal studies that evaluated hypertension incidence associated with sleep duration. Overall, the pooled risk for hypertension incidence associated with short sleep duration was RRR=1.21 (95% CI 1.05–1.20). As the studies included in this analysis were heterogeneous, the authors explored whether certain factors were associated with markedly increased risk ratios. They found, for example, that when analyses were restricted to longer follow-up periods, this was increased to RRR=1.29 (95% CI 1.09–1.52). Also, when hypertension was determined objectively, the risk was nominally higher (RRR=1.24, 95% CI 1.04–1.49), as was the risk when sleep was measured objectively (RRR=1.29, 95% CI 1.03–1.62). This suggests that the relationships are more clearly evident when hypertension and/or sleep are measured objectively.

This study had a number of strengths. The authors took great care to perform a careful and thorough review of the literature. The guidelines set forth by the MOOSE study group (Meta-analyses Of Observational Studies in Epidemiology) outline 35 guidelines for meta-analyses.[8] The authors were able to apply nearly all of these guidelines to their study. In particular, the reporting of the search strategy was detailed; the reporting of selection, documentation, assessments of study quality and other statistical methods were comprehensive; the reporting of results was clear and consistent with the methods chosen; and the discussion and conclusions included thoughtful quantitative and qualitative discussion of the studies and findings.

In addition, the choice of the authors to focus on incidence of hypertension was a strength of the study. Although many reports may have been included if cross-sectional studies could be included, potentially improving statistical power and representativeness, the focus on longitudinal studies brings front and center the question of causality. This is particularly important, as most of the assessments of sleep focus on short-term experiences, whereas any assessment of hypertension (objective or subjective) likely represents a longer time period. For example, subjective diagnosis of hypertension almost always reflects an event that happened prior to the window in which sleep is assessed and even objective assessment of hypertension reflects cardiovascular health over an extended period (as resting blood pressure does not change very quickly). Therefore, this study provides evidence for the claim that the sleep issues are (at least in many cases) predating hypertension. Not only does this elevate the debate on this issue, but it also gives added credibility to the cross-sectional studies that cannot evaluate incidence.

Despite these and other strengths, this meta-analysis had a number of limitations. First, as in all other meta-analyses of epidemiologic sleep data, there is a large amount of variability in

types of sleep assessments (for example, survey item, questionnaire, actigraphy) and there is variability in defining the exposure of interest (for example, variable definitions of 'short' sleep, various definitions of sleep 'difficulties' with varying methods for classifying severity). These are major issues in epidemiologic sleep research.[6] This degree of variability is partially addressed in the study by Meng *et al.*[1] through analyses of subgroups of studies. However, these subgroups were so small that these findings may not be reliable. As sleep is increasingly identified as an important health risk factor, some consistency in estimation of relevant sleep variables in epidemiologic studies is needed. Currently, most cohorts that assess sleep do not use standardized instruments. Part of the reason is that brief standardized measures (for example, 1–3 item measures of sleep duration or insomnia), which would be useful for epidemiologic research, are not available.

Another issue with the current study is that sleep duration and insomnia re-evaluated separately. These are separate constructs, as many people with difficulty falling asleep still manage to obtain >6 h per night, and many who sleep <6 h per night do not have any trouble falling or staying asleep. However, there is an emerging literature that suggests that the interface of these two phenomena—individuals with insomnia that also obtain <6 h of sleep—are at particularly high risk for cardiometabolic disease and other adverse outcomes, including mortality.[9] Future epidemiologic studies should investigate this phenomenon to determine the relative roles of insomnia and short sleep duration, and the potential interaction of these.

In conclusion, the report by Meng *et al.*[1] represents an important contribution to the literature, documenting a role of short sleep and insomnia in hypertension incidence. Not only does this suggest that sleep issues may predate development of hypertension (at least in some cases), but it also highlights that the adverse outcomes of insomnia, as well as short sleep duration, go beyond the behavioral, psychological or functional domains and include important medical consequences as well.

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