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Are Short Bad Sleep Nights a Hindrance to a Healthy Heart?

Commentary on Hoevenaar-Blom et al. Sleep duration and sleep quality in relation to 12-year cardiovascular disease incidence: the MORGEN Study. SLEEP 2011;34:1487-1492.

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The prospective study by Hoevenaar-Blom and colleagues[1] in this issue of SLEEP was carried out in a large representative sample of Dutch population. It confirms the previously reported associations between short (but not long) sleep duration and both cardiovascular (CVD) and coronary heart (CHD) disease incidence of fatal and non-fatal events. In addition, the strongest association was found in short sleepers with poor sleep quality, highlighting the importance of sleep quality, not just quantity, as a potential risk marker, or factor, for cardiovascular disease.

They studied over 20,000 adults who were followed up for an average of 12 yr. Self-reported duration of sleep was assessed at baseline and related to the incidence of fatal and non-fatal CV events during follow-up. In a third of the sample sleep quality (assessed by responses to the question "Do you usually rise rested?") was also measured in the first 2 yr. After adjustment for several potential confounders, participants who reported sleeping 6h or less per day had a 15% greater risk of CVD and a 23% greater risk of CHD than those sleeping 7h per night. Poor sleep quality was not significantly associated with adverse outcomes in fully adjusted models. However, the greatest risk was detected amongst short sleepers with poor quality sleep (63% for CVD and 79% for CHD). The study is large, with long follow-up, sufficient statistical power and excellent outcome assessment.[1]

The epidemiology of sleep deprivation and long-term outcomes has gained momentum in the last 15 yr with the publication of consistent associations between short sleep and adverse health outcomes, including total mortality[2], coronary heart disease and stroke,[3] type-2 diabetes,[4] obesity in children and adults,[5] hypertension,[6] and poor-self rated health.[7] Sleep disturbances may therefore be a cause, a consequence or a symptom of ill-health.[8] The concept of causality is reinforced by the strength of the associations,[1,3] their consistency across different populations,[3,9] the temporal sequence between exposure and outcomes,[1,3] its specificity,[10] and the biological plausibility.[11-12] Lack of sleep exerts deleterious effects on a variety of systems with detectable changes in metabolic, vascular, endocrine and immune pathways[11-12] (Figure 1). In turn, these mechanisms may directly contribute to the worsening of established cardiovascular risk factors, strong predictors of fatal and non-fatal outcomes.

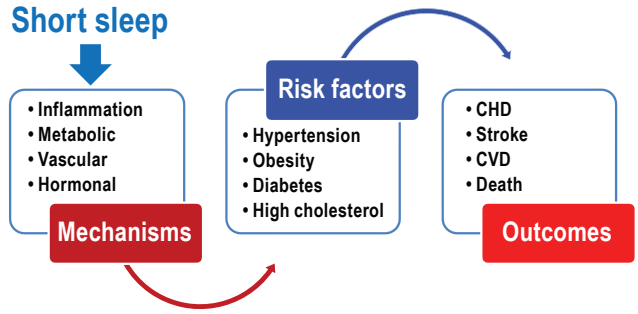

Figure 1

While the Hoevenaar-Blom et al. study[1] adds to the body of evidence in support of the causality link, it nevertheless does not rule out the view that short and poor sleep could be a consequence or a symptom of ill-health. Insomnia and poor sleep quality are very common in depression and often are the early signs. Likewise, people with CHD and other cardiovascular diseases suffer from depression. Lack of adjustment for this common neuropsychiatric condition, and for the frequent use of hypnotics, may, in part, confound the association reported, in particular that between poor sleep amongst short sleepers and CVD as well as CHD.

Other important limitations of large observational studies are inevitable. Examples of these limitations include the following: (1) representativeness (only 33% of the original cohort provided information on sleep quality); (2) accuracy of estimation of exposure (sleep quantity is almost invariably self-reported and information on sleep quality is often not validated and standardized, making its external validity difficult to assess): and (3) extrapolation on long-term effects (single point assessment of sleep taken as sustained exposures over time). In one study[10] the stability of the assessment of exposure was addressed by recording self-reported duration of sleep twice—5 yr apart—and by constructing a variable that would identify individuals who had reduced sleep duration over time. This exposure variable predicted CVD mortality (but not all-cause mortality) 10 yr later. Finally, sleep quality may be affected in different domains, like difficulty in initiating sleep, reduced slow wave sleep, sleep disordered breathing, microarousals and sleep fragmentation. It is not clear which of these components may be predicting ill-health.

How can we move forward this important area of research, given that sleep is a function common to all human beings and cardiovascular disease the most common cause of death, morbidity and disability in the world? Reversibility of effect is another strong clue for causality in epidemiological research. The

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ultimate proof of principle, therefore, should come from a randomized controlled clinical trial of sustained moderate sleep 'prolongation' on long term outcomes, or on some surrogate markers of morbidity, risk factors or mechanisms. Laboratory routines with acute short-term extreme changes in sleep duration have been carried out supporting the effects of sleep deprivation on some mechanisms.[11] However, these acute results cannot be taken as proof that such effects will be sustained in the longer term when homeostatic compensatory mechanisms may come into play. Currently we do not have effective and reproducible intervention tools and protocols to moderately increase sleep time (e.g., by 1 h per day) and to sustain such an intervention for months or years. Nor do we have any way of experimentally improving sleep quality without resort to medications.

In view of the likely multilevel determinants of quantity and quality of sleep (genetic, biological, behavioral, environmental, societal, secular, etc.), it appears that a major effort in future research should be to explore and validate new behavioral (non drug based) methods to prolong sleeping time and to improve quality that could be sustained for a long period of time. An early attempt to fill this gap is currently ongoing in relation to the effects of sleep prolongation and weight change.[13] In the meantime, epidemiological research should continue its contribution to etiology by improving measurements of exposure (sleep quantity and quality), by minimizing residual confounding, and by exploring additional outcomes and mechanisms that could be affected by sleep reduction and disturbances. Notwithstanding these needed developments, it is clear that population research has unveiled new concepts with important implications for public health and policy.[8]

CITATION

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DISCLOSURE STATEMENT

The authors have indicated no financial conflicts of interest.

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