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Long sleep duration: A risk to health or a marker of risk?

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Although the function of sleep remains a biological enigma, there is an accumulating mass of empirical evidence demonstrating that habitually short sleep duration can have adverse behavioral and non-behavioral consequences. In recent years, there has evolved a parallel notion that habitually long sleep duration may also impact health. In fact, the repeated observance of a U-shaped relationship between sleep duration and mortality across several epidemiologic studies (1-12) has led to the question of whether there is an ‘optimal’ amount of sleep necessary to maintain good health and maximize longevity. In their review, Grandner and Drummond (13) address the health-related significance of long sleep duration (i.e., habitual sleep duration longer than the ‘optimal’ amount) and assess the state of the evidence regarding its role as a potential risk factor for mortality. Some of the methodological limitations pertinent to the interpretation of epidemiologic data on long sleep duration and mortality were highlighted in the review. These include assessment of sleep duration by reliance on self reports, and threats to generalizability stemming from variability in the definitions of long sleep duration, sample demographics, and covariate adjustment across studies. Furthermore, a principal concern inherent in all epidemiological investigations on the association between long sleep duration and mortality is the observational study design. Unlike experimental studies, epidemiologic studies need to rely on observations, or data, collected from subjects in their natural setting over a defined period of observation. Because assignment to a set of independent variables (e.g., habitual sleep duration) is not possible, the challenge of estimating causal effects can be formidable. Thus, understanding all possible shortcomings in these studies is necessary to fully decipher the implications of the data on long sleep duration and to gauge the potential impact of modifying sleep duration to some ‘optimal’ level for improved health outcomes.

Specific factors that could alter the association between long sleep duration and mortality can have distinct roles in the putative causal pathway either as mediators or confounders. Proposed mediators that could causally link long sleep duration to mortality are distinct from confounders, or factors which are related to both mortality and long sleep duration but are not themselves in the causal pathway. Grandner and Drummond list several proposed “mechanisms” that account for the observed relationship between long sleep duration and mortality, which could be grouped accordingly as confounders (e.g., depression, sleep apnea, fatigue, poor sleep quality), and true mediators of the effect (e.g., lack of challenge, shortened photoperiod). Such distinctions are critical in order to appraise the current state of the evidence and to identify new directions for further research efforts.

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In interpreting the results of statistical models on which inferences from the cited epidemiological studies rely, one of the most important considerations is the potential for residual confounding. Residual confounding occurs when a factor known to be associated both with the disease outcome (e.g., mortality) and with the exposure (e.g., long sleep duration) is not measured in the study or not adequately controlled for in the analysis. Omission of confounding covariates would lead to unadjusted or crude estimates, or, if the confounder is not adequately measured, to incomplete adjustment. The latter is perhaps a more insidious source of bias as it is often difficult to ascertain how well the factor is measured even after identifying it as a potential source of confounding. For example, obesity is a commonly identified confounding factor as well as a source of residual confounding in epidemiological studies, as it is usually assessed with the body mass index (BMI) even though many health-related outcomes are better predicted by more precise measures of adiposity such as percent body fat or the amount of visceral fat (14;15).

On the topic of long sleep duration and mortality, a key confounding factor is sleep apnea, which is often not assessed in large cohort studies, but has been associated with higher mortality (16) and with long sleep duration (17). As discussed by Grandner and Drummond, some of the available studies have adjusted for “reliable predictors of sleep apnea, including age, gender, BMI, and ethnicity”. However, as proxy measures of sleep apnea, adjustment for these factors would by definition introduce bias due to residual confounding. The potential for residual confounding bias should not be underestimated when strong confounders are measured with error (18). This can occur with the use of proxy measures for conditions such as sleep apnea, difficult-to-measure conditions such as depression, or for any variable that fluctuates over time. Consideration of residual confounding is particularly relevant in understanding the impact of poor health status on the relationship between low socioeconomic position with long sleep duration and mortality, as socioeconomically disadvantaged groups are more likely to carry a greater burden of physical and mental health conditions as well as having a lower likelihood of being diagnosed and treated.

In addition to the concerns of confounding, incomplete assessment of habitual sleep patterns can also impact our ability to infer the impact of sleep duration on mortality. Without question, sleep duration is a dynamic phenomenon with short- and long-term fluctuations that are, in part, determined by other aspects of our lives (e.g., work, family obligations). Therefore, assessment of health-related consequences requires consideration of such time-dependent patterns and perhaps even the possibility that there are periods of vulnerability during which deviance from the ‘optimal’ amount of sleep has the most deleterious effects. Understanding the potential roles of age, cohort, and period effects is common in modern day epidemiology (19) but relatively unexplored in the area of sleep disorders medicine. Observed differences in survival as a function of sleep duration may result from interactions between these effects necessitating a careful exposition in understanding any trends in mortality.

In their review, the authors draw an analogy between sleep duration and caloric intake to signify an ‘optimal’ level of habitual sleep duration. Thus, just as there is an optimal level of caloric intake, there is perhaps a ‘healthy set-point’ for sleep duration that all of us should achieve. While this is a useful analogy, an alternative comparison could also be made with other factors that have U-shaped associations with mortality, such as BMI (20-22). For example, the observations of higher mortality among those categorized as having a normal BMI, compared to overweight BMI, are best explained by reverse causation (*i.e.*, illness leading to weight loss) and residual confounding, and not because a lower BMI itself leads to increased risk of death. Thus, it is certainly plausible that long sleep duration is a surrogate marker of other factors that predispose to higher mortality rates.

From a population health standpoint, the potential burden of adverse consequences of long sleep duration must be placed in some perspective by examining the relative prevalence of habitual sleep duration (based on self-reports) at both short and long ends of the spectrum. In 15,000 respondents sampled from existing epidemiological cohort studies between 1995 and 1998 for the Sleep Heart Health Study, the prevalence of short sleep duration (< 7 hours) was 27.5%, while the prevalence of long sleep duration (≥ 9 hours) was only 9.2% (Punjabi NM, Personal Communication). These estimates fall within the prevalence ranges of short and long sleep duration reported in most other community-based samples. While not reported in the published articles, the proportion of long sleepers without any underlying physical or mental comorbidity is likely to be even smaller, and thus, its independent impact on mortality patterns likely diminished.

What is clear from the literature is that long sleep duration cannot be assessed as a risk factor, or independent causal factor, for mortality without also assessing the potential confounding impact of other health conditions such as depression, sleep apnea, and other physical comorbidities and behaviors (e.g., alcohol consumption) that, through various mechanisms, may manifest themselves through extended sleep duration. Furthermore, while there are many external pressures that can limit sleep duration to less than the optimal amount, it appears unlikely that habitual extension of sleep beyond optimal levels is a common behavior in otherwise healthy adults. The bulk of the literature supports that habitually long sleep duration is likely to be an indicator of poor physical and mental health status, but offers only weak evidence that extension of sleep, as an optional or directly modifiable behavior, is a driving causal force in patterns of mortality. Whatever the eventual truth may be regarding the role of sleep duration and mortality, it is perhaps best to follow the old proverb: "Everything in moderation".

Reference List

1. Amagai Y, Ishikawa S, Gotoh T, Doi Y, Kayaba K, Nakamura Y, et al. Sleep duration and mortality in Japan: the Jichi Medical School Cohort Study. *J Epidemiol* 2004;14(4):124–128. [PubMed: 15369129]
2. Ayas NT, White DP, Manson JE, Stampfer MJ, Speizer FE, Malhotra A, et al. A prospective study of sleep duration and coronary heart disease in women. *Arch Intern Med* 2003;163(2):205–9. [PubMed: 12546611]
3. Gottlieb DJ, Schulman DA, Nam BH, D'Agostino RA, Kannel WA. Sleep duration predicts mortality: the Framingham Study. *Sleep* 2002;25:A108.
4. Hammond EC. Some preliminary findings on physical complaints from a prospective study of 1,064,004 men and women. *Am J Public Health Nations Health* 1964;54:11–23. [PubMed: 14117648]
5. Heslop P, Smith GD, Metcalfe C, Macleod J, Hart C. Sleep duration and mortality: The effect of short or long sleep duration on cardiovascular and all-cause mortality in working men and women. *Sleep Med* 2002;3(4):305–14. [PubMed: 14592192]
6. Kojima M, Wakai K, Kawamura T, Tamakoshi A, Aoki R, Lin Y, et al. Sleep patterns and total mortality: a 12-year follow-up study in Japan. *Journal of Epidemiology* 2000;10(2):87–93. [PubMed: 10778032]
7. Kripke DF, Simons RN, Garfinkel L, Hammond EC. Short and long sleep and sleeping pills. Is increased mortality associated? *Arch Gen Psychiatry* 1979;36(1):103–16. [PubMed: 760693]
8. Kripke DF, Garfinkel L, Wingard DL, Klauber MR, Marler MR. Mortality associated with sleep duration and insomnia. *Arch Gen Psychiatry* 2002;59:131–6. [PubMed: 11825133]
9. Patel SR, Ayas NT, Malhotra MR, White DP, Schernhammer ES, Speizer FE, et al. A prospective study of sleep duration and mortality risk in women. *Sleep* 2004;27(3):440–444. [PubMed: 15164896]
10. Pollak CP, Perlick D, Linsner JP, Wenston J, Hsieh F. Sleep problems in the community elderly as predictors of death and nursing home placement. *J Community Health* 1990;15(2):123–135. [PubMed: 2355110]

11. Tamakoshi A, Ohno Y. Self-reported sleep duration as a predictor of all-cause mortality: results from the JACC study, Japan. *Sleep* 2004;27(1):51–54. [PubMed: 14998237]
12. Wingard DL, Berkman LF. Mortality risk associated with sleeping patterns among adults. *Sleep* 1983;6(2):102–7. [PubMed: 6878979]
13. Grandner MA, Drummond SPA. Who Are the Long Sleepers? Towards an Understanding of the Mortality Relationship. *Sleep Medicine Reviews*. 2007
14. Kuk JL, Katzmarzyk PT, Nichaman MZ, Church TS, Blair SN, Ross R. Visceral fat is an independent predictor of all-cause mortality in men. *Obesity (Silver Spring)* 2006;14(2):336–341. [PubMed: 16571861]
15. Rexrode KM, Carey VJ, Hennekens CH, Walters EE, Colditz GA, Stampfer MJ, et al. Abdominal adiposity and coronary heart disease in women. *JAMA* 1998;280(21):1843–1848. [PubMed: 9846779]
16. Marin JM, Carrizo SJ, Vicente E, Agusti AG. Long-term cardiovascular outcomes in men with obstructive sleep apnoea-hypopnoea with or without treatment with continuous positive airway pressure: an observational study. *Lancet* 2005;365(9464):1046–1053. [PubMed: 15781100]
17. Gottlieb DJ, Redline S, Nieto FJ, Baldwin CM, Newman AB, Resnick HE, et al. Association of usual sleep duration with hypertension: the Sleep Heart Health Study. *Sleep* 2006;29(8):1009–1014. [PubMed: 16944668]
18. Christenfeld NJ, Sloan RP, Carroll D, Greenland S. Risk factors, confounding, and the illusion of statistical control. *Psychosom Med* 2004;66(6):868–875. [PubMed: 15564351]
19. Szklo, M.; Nieto, FJ. *Epidemiology: beyond the basics*. Gaithersburg: Aspen Publishers, Inc.; 2000. p. 4-16.
20. Flegal KM, Graubard BI, Williamson DF, Gail MH. Excess deaths associated with underweight, overweight, and obesity. *JAMA* 2005;293(15):1861–1867. [PubMed: 15840860]
21. Gelber RP, Kurth T, Manson JE, Buring JE, Gaziano JM. Body mass index and mortality in men: evaluating the shape of the association. *Int J Obes (Lond)*. 2007
22. Manson JE, Bassuk SS, Hu FB, Stampfer MJ, Colditz GA, Willett WC. Estimating the number of deaths due to obesity: can the divergent findings be reconciled? *J Womens Health (Larchmt)* 2007;16(2):168–176. [PubMed: 17388733]