EDITORIAL

Can Habitual Sleep Duration Harbor Sleep Debt?

Comment on Klerman EB; Dijk DJ. Interindividual variation in sleep duration and its association with sleep debt in young adults. SLEEP;28(10):1253-1259

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IN THIS ISSUE, KLERMAN AND DIJK1 REPORT AN EXPERIMENT THAT FOCUSES ON HOW TO INTERPRET DIFFERENCES AMONG PEOPLE IN HABITUAL BEDREST duration (HBD) using daytime sleep propensity and a sleep extension paradigm to evaluate the relationship between HBD and purported sleep need. There is considerable scientific evidence that sleep debt can develop in a dose-response manner relative to reduced time in bed, and that the effects can be cumulative and include significant functional consequences, especially in objective measures of alertness.2,4 But we cannot say a priori whether naturally occurring shorter-duration HBDs are a sign of sleep debt. That is, we do not know to what extent variation among people in habitual sleep duration found in any given population of healthy individuals is primarily a reflection of inter-individual variability in biological sleep need, or to what extent it reflects variation in sleep debt resulting from a forced reduction in sleep duration that masks basal sleep need.7 The problem making this discrimination evident when attempting to explain what is perhaps one of the most ubiquitous features of human sleep-wake behavior—namely the extension of sleep duration on weekends or holidays or days off. Is the extra sleep (nocturnal extension and diurnal naps) on days when work or domestic duties are not mandated indicative of a need to compensate sleep debt by obtaining extra (recovery) sleep? Or is it merely a social decision to consume sleep we don’t need (i.e., what was originally labeled “optional sleep” but now is referred to as “adaptable sleep”)? In other words, what is the biological significance of an ability to obtain extra sleep relative to habitual sleep duration?

Klerman and Dijk1 took an approach to studying habitual sleep duration similar to the way in which Kleitman used the phrase “sleep debt” to characterize what results when one reduces nocturnal sleep time, then attempts to reverse the effects by extending sleep to “liquidate the debt” (p. 317).6 This original conceptualization of sleep debt to explain periodic extension of sleep duration remains relevant to both the fundamental question of how much sleep people need to avoid a cumulative sleep debt, and which aspects of sleep physiology are essential for recovery of healthy waking neurobehavioral and physiological functions. The experimental effects of sleep extension have received scant attention, but the few studies that have been published suggest extended sleep duration can normalize wakefulness10 and metabolic responses,11 and perhaps even expose the basal sleep need of an individual.12

The experimental results of Klerman and Dijk1 provide some of the first evidence that inter-individual variation in well-documented habitual bedrest duration may harbor substantial sleep debt, rather than simply reflecting natural biological variability in basal sleep need. These investigators quantified habitual bedrest duration in 17 healthy young adults using daily diaries, time-stamped call-ins, and actigraphy over a 3-week period, and then evaluated the relationship between HBD on the final week and daytime sleepiness (MSLT) after 1 night in the laboratory. They found that individuals with shorter habitual bedrest durations during the 3 weeks prior to laboratory testing fell asleep more quickly and frequently during the MSLT (often less than 5 minutes) than did those with a longer HBDs, suggesting that sleep debt—as determined by daytime sleep propensity—increased as habitual bedrest duration decreased. The finding is consistent with other evidence that habitual short sleepers tend to have a higher homeostatic sleep pressure than longer sleepers,13 and that they would benefit from increased time in bed for sleep.14 This is an important observation, but it does not tell us whether these short sleepers are able to resist the elevated sleep drive they experience (i.e., have a greater tolerance to sleep debt), or whether they are vulnerable to the sleep debt and experience greater functional deficits and risks associated with sleep debt. Distinguishing between these two alternatives would require other measures of waking functions (e.g., cognitive performance, metabolic outcomes). If healthy adults with short-duration HBD carry a higher homeostatic sleep drive because they are resistant to sleep loss, then there should be no cognitive performance (or metabolic) changes over time at habitual bedrest duration. On the other hand, if the higher sleep debt of short-duration HBD subjects manifests in waking functions, then there should be clear evidence of cognitive performance (or metabolic) changes over time at typical HBD. In this eventuality, the question is how these individuals maintain such reduced sleep durations over the long term.

Although the MSLT data of Klerman and Dijk1 are consistent with the hypothesis that shorter-duration HBD in healthy young adults may harbor sleep debt that reduces daytime alertness, it is also possible that short-duration HBD individuals may demonstrate ease of falling asleep on the MSLT, but experience no difficulty remaining awake and performing alertly in the face of

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SLEEP, Vol. 28, No. 10, 2005

1209 Editorial—Dinges

To be theoretically comprehensive, we must also allow that recovery from sleep debt may occur without sleep, but there is as yet no definitive evidence to support this.
elevated sleep debt. Recent research on repeated exposure to sleep deprivation has provided evidence of trait-like differential vulnerability to the effects of sleep loss, especially on objective neurobehavioral measures of alertness, but this differential vulnerability appeared to be unrelated to subjects’ habitual bedrest durations in the narrow range of HBD studied. It would be valuable to know whether the ability to resist cognitive performance decrements accompanied short HBD in Klerman and Dijk’s study, or whether cognitive performance showed an inverse relationship to habitual bedrest duration similar to what was found for sleep propensity.

Following MSLT testing after habitual bedrest duration, Klerman and Dijk measured polysomnography and subjective sleepiness and tiredness during each of 3 days in which subjects were sequestered for sleep during 12 hours nocturnal time in bed and 4 hours diurnal time in bed. The investigators reasoned that if an individual carried sleep debt coming into repeated days of sleep extension, there would be an initial increase in TST, which would decrease during subsequent days. In contrast, if an individual’s HBD accurately reflected sleep need, and additional sleep is optional, any excess sleep should be identical during all extended sleep opportunities. The results were relatively clear. Subjects slept an average of 4.9 hours more than their habitual bedrest duration during the first day of increased sleep extension, and a majority slept more than their HBD on the second and third days of sleep extension. On the third day, however, total sleep time was negatively associated with habitual bedrest duration. That is, subjects with shorter HBD slept longer than those with longer HBD, suggesting that sleep was still being recovered in short-HBD volunteers. According to the logic of the experiment, “individuals with short HBD carry a larger sleep debt”(p. 1239). Also supporting the conclusion that the extended sleep was affording recovery, was the fact that volunteers reported increased levels of subjective alertness during the bedrest extension days. However, the interpretation of these results would be enhanced if we knew subjects’ steady-state total sleep time, which would require more days of sleep extension.

Interestingly, total sleep time, but not slow wave sleep or REM sleep had a significant relationship with habitual bedrest duration across the days of bedrest extension. Klerman and Dijk did not present stage 2 non-REM sleep results separate from their amalgam with slow wave sleep. Based on the slow wave sleep and REM sleep increments, stage 2 sleep must have comprised a great deal of the extra sleep obtained during sleep extension days. Perhaps stage 2 sleep provides substantial recovery relative to sleep debt induced by habitually reduced sleep. Stage 2 typically comprises approximately 50% of sleep, and it is this stage more than any other that is reduced when sleep is chronically restricted. On the other hand, total sleep time remains the most parsimonious physiological construct for understanding sleep debt.

Remarkably, Klerman and Dijk found that even after 3 days of extended sleep opportunities, total sleep time was considerably longer than habitual bedrest duration in many of their young adult subjects. In fact some subjects with short HBD had not reached a steady state of sleep duration after 3 days (or 48 total extra hours) of sleep opportunity, although the rate of change in total sleep time declined across the days of bedrest extension, prompting the investigators to speculate that the time course of recovery from chronic sleep debt may take more than 2 or 3 days of extended sleep.

REFERENCES

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SLEEP, Vol. 28, No. 10, 2005