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Editorial

The Guilleminault et al. article on the timing of sleep opportunities in a seven-night sleep restriction, in addition to showing that sleep timing is critical to the tolerability of sleep restriction, also reports important individual differences to sleep deprivation tolerability [1]. Of itself, the finding that the timing of sleep opportunity is critical to sleep restriction tolerability is noteworthy. This article reports that sleep efficiency for a sleep period centered at midnight was low, while that centered at 04:00 h in the early morning was quite high. These differential nocturnal sleep efficiencies affected levels of daytime alertness; the higher sleep efficiency was associated with greater alertness. The authors note in discussing this finding that these healthy young college students were most probably phase-delayed. The point is made that the early morning sleep opportunity was likely centered over the circadian nadir of core body temperature in these young adults, while in the midnight sleep opportunity it was likely still rising. Any number of studies have shown that the ability to sleep, regardless of the intensity of the homeostatic sleep drive, is determined by circadian timing across the 24-h day [2], but this study goes a step beyond and shows that within the dark period there are significant individual differences.

Of particular interest in this article are the individual differences in restriction tolerability reported by the authors. The possible sources of such individual variability are worth discussing. The first type of individual differences might be described as state differences. The possibility of differences in basal level of sleepiness-alertness is highly probable. Among a large sample of healthy young adults, similarly recruited from a university community, with stable nightly sleep schedules of a little more than 7 h, without sleep complaints, or reported daytime sleepiness, the average daily sleep latency on a multiple sleep latency test (MSLT) varied from 2 to 20 min [3]. Such differences had an impact on the tolerability of a sleep phase advance in another study. Sleepy individuals at baseline had relatively high sleep efficiencies on the 4-h phase advance, while the sleep efficiencies of alert individuals were low [4]. Secondly, there may be basal differences in current circadian phase. This study tried to control for such differences by requiring a set bedtime for 1 week prior to the experiment.

The second class of individual differences can be described as trait differences. One's optimal circadian phase for sleep and wakefulness can be considered a trait variable, in contrast to the state variable current circadian phase. In this study, the Horne and Osberg questionnaire was used to screen for morningness–eveningness extremes, a variable previously shown to be under genetic influence [5]. Another potential explanatory trait variable, but one that has received very little experimental attention, is differences in the sensitivity and responsivity of the sleep homeostat; that is, how large a sleep deficit the system can tolerate and how robustly the sleep homeostat produces sleep when detecting a deficiency. In a 2-week sleep extension study of sleepy, healthy normals, the majority of individuals slept during the extended bedtime, which produced a normalization of their MSLT [6]. But a small subset did not sleep during the added bedtime and their MSLT did not improve. Among other things, this difference could be due to a reduced sensitivity or responsivity of their sleep homeostat. A final trait variable that might be considered is a difference in compensatory mechanisms in response to an accumulated sleep deficiency. Some studies have shown that compensation to the cognitive and behavioral effects of sleep restriction does occur, particularly when the sleep loss accumulates at a slow rate [7]. Individual differences in compensatory capacities have not been specifically identified, but most probably do exist.

Among the interesting questions is the extent to which the genetic code for these trait differences will become known. While to date no studies in humans have shown a genetic difference in sleep homeostasis (i.e., as defined by delta power), there have been such demonstrations in inbred mouse strains [8]. Sleep need is a construct that refers to the set point around which the sleep homeostat regulates daily sleep time and sleep need (i.e., the set point) and is hypothesized to vary among individuals and to be genetically determined. Similarly, the sensitivity and responsivity of the sleep homeostat could also be genetically determined.

With sleep, as in all physiological systems, the critical question is always how much of the variance is attributable to genetics and how much to the environment. The influence of societal causes of sleep deprivation on some individuals (e.g., medical residents, shift workers) has to be balanced with the increasing evidence of genetic modulation of both

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circadian and homeostatic processes. The Guilliminault et al. article highlights the large individual differences, which are the result of these two factors.

References

[1] Guilleminault C, Powell NB, Martinez S, Kushida C, Raffray T, Palombini L, Philip P. Preliminary observations on the effects of sleep time in a sleep restriction paradigm. Sleep Med 2003;4:177–84.
[2] Dijk D-J, Czeisler CA. Contribution of the circadian pacemaker and the sleep homeostat to sleep propensity, sleep structure, electroencephalographic slow waves and sleep spindle activity in humans. J Neurosci 1995;15:3526–38.
[3] Levine B, Roehrs T, Zorick F, Roth T. Daytime sleepiness in young adults. Sleep 1988;11:39–46.
[4] Roehrs T, Salin-Pascual R, Merlotti L, Rosenthal L, Roth T. Phase advance in moderately sleepy and alert normals. Sleep 1996;19: 417–22.
[5] Vink JH, Groot AS, Kerkhoff GA, Boomsma DI. Genetic analysis of morningness and eveningness. Chronobiol Int 2001;18:809–22.
[6] Roehrs T, Shore E, Papineau K, Rosenthal L, Roth T. A two-week sleep extension in sleepy normals. Sleep 1996;19:576–82.
[7] Drake C, Roehrs T, Burduvali E, Bonahoom A, Rosekind M, Roth T. Effects of rapid versus slow accumulation of eight hours of sleep loss. Psychophysiology 2001;38:979–87.
[8] Franken P, Cholbet D, Tafti M. The homestatic regulation of sleep need is under genetic control. J Neurosci 2001;21:2610–21.

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