

Sleep Debt and Cumulative Excess Wakefulness

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IN HIS EDITORIAL,¹ CZEISLER DESCRIBES OUR STUDY OF THE NEUROBEHAVIORAL EFFECTS OF CHRONIC SLEEP LOSS AS A MILESTONE. We are pleased that our experiments helped settle the longstanding debate over whether chronic sleep restriction could lead to cumulative waking neurobehavioral impairments. In a heuristic analysis of the cause of these cumulative deficits from sleep loss, we offered different ways to calculate sleep debt.² Czeisler takes issue with the distinction we made between cumulative sleep debt and cumulative excess wakefulness.¹ We note that as commonly conceptualized, however, “sleep debt” is used to describe effects associated with sleep loss, regardless of the reason for the lost sleep.³ If valid, this conceptualization should explain the rate at which performance impairments build up both over days of chronic sleep restriction and over days of total sleep deprivation. To do so using cumulative sleep debt, however, one must posit different neurobehavioral mechanisms for the effects of these two modes of sleep deprivation.^{1,4} We offered the more parsimonious explanation of “cumulative excess wakefulness,” which reconciled psychomotor vigilance task (PVT) performance lapses across days of total sleep deprivation and days of chronic sleep restriction,² without invoking additional mechanisms (or assuming linearity¹).

Czeisler correctly points out that our analysis based on cumulative excess wakefulness presumes that sleep of at least 4h duration “resets the system,”¹ by providing credit for ~16h of wakefulness. Based on our data, we estimated that the duration of stable wakefulness to be gained prophylactically from at least 4h sleep was $\xi=15.84\pm 0.73$ h (mean \pm s.e.).² However, since sleep and wakefulness are organized in circadian cycles, obtaining ~16h of stable wakefulness following 4h of sleep still leaves ~4h of wakefulness per cycle unaccounted for. We hypothesized that this excess wakefulness caused the neurobehavioral deficits we observed. Accordingly, getting ~8h of sleep per circadian cycle would prevent the accumulation of excess wakefulness in the average individual.²

If sleep periods of 4h time in bed (TIB) or more provide ~16h of stable wakefulness, but 0h TIB provides no waking credit, Czeisler appropriately questions whether sleep periods shorter than 4h (and even as brief as microsleeps) would provide ~16h of stable wakefulness,¹ or less.

We have been studying this issue and report here data from $n=9$ subjects in an experiment identical to the 0h TIB condition, except that 2h TIB for sleep was scheduled every night (02:45-04:45). Polysomnographically assessed total sleep time was 1.77 ± 0.05 h (mean \pm s.e.) per day. When we tentatively assumed that these sleep periods would result in $\xi=15.84$ h of stable wakefulness (i.e., the net excess wakefulness would be only $24\text{h}-15.84\text{h}-1.77\text{h}=6.39$ h per day on average), PVT performance impairment in this condition was found to be worse than predicted by cumulative excess wakefulness beyond 15.84h (Figure 1a). It follows that the duration of stable wakefulness gained from 2h TIB must be shorter than 15.84h. We conclude that the hypothetical period of stable wakefulness ξ may progressively shorten when TIB is increasingly reduced below 4h—a conclusion consistent with the results of prophylactic napping studies.⁵ By logical extension, brief epochs of unintended sleep in the 0h TIB condition may have provided some benefit, but of very short duration only.

The three chronic sleep restriction conditions in our study had in common that slow wave sleep (SWS) was preserved. Czeisler proposes that the reason subjects in the 0h TIB condition accumulated performance deficits rapidly was the loss of SWS.¹ Subjects in the 2h TIB condition described above had 0.34 ± 0.05 h (mean \pm s.e.) of SWS per day, which was 29% of the average daily SWS found in the 4h, 6h and 8h TIB conditions.² When we postulated that the amount of stable wakefulness for the 2h TIB condition would be proportionately less (i.e., $\xi=29\% \times 15.84\text{h}=4.59$ h), the PVT performance deficits plotted against cumulative excess wakefulness lined up with the other conditions (Figure 1b). While this does not prove that SWS (or EEG slow wave activity) is essential to the recovery and/or prophylactic function of sleep, it suggests a mechanism by which performance deficits from cumulative sleep loss may be unified across the range from 0h to 8h TIB, requiring no adjustment of the cumulative excess wakefulness hypothesis² and no artificial differentiation of “continuous wake extension.”¹ Thus, we believe that neurobehavioral performance impairment from sleep loss may be parsimoniously understood as reflecting the cost of cumulative excess wakefulness.²

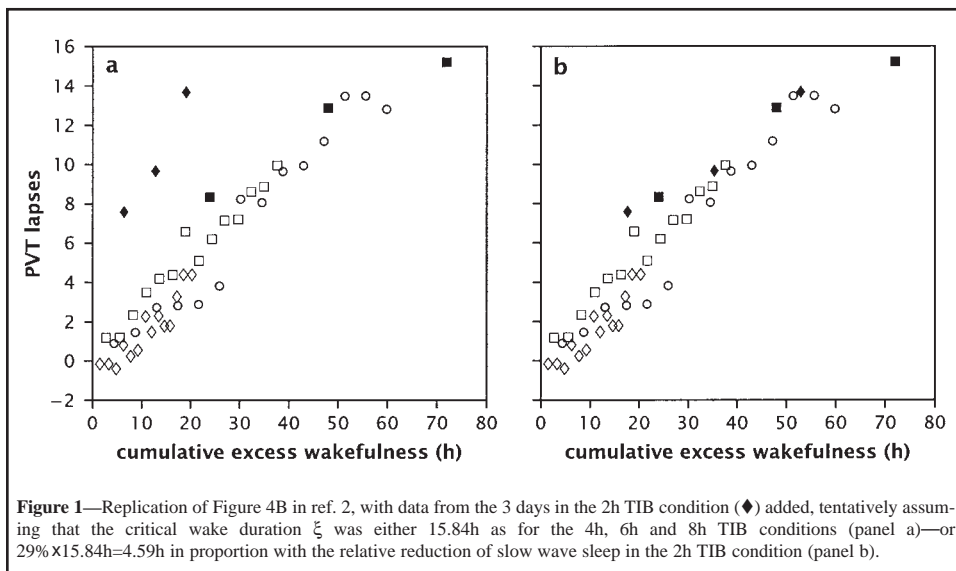


Figure 1—Replication of Figure 4B in ref. 2, with data from the 3 days in the 2h TIB condition (◆) added, tentatively assuming that the critical wake duration ξ was either 15.84h as for the 4h, 6h and 8h TIB conditions (panel a)—or $29\% \times 15.84\text{h}=4.59$ h in proportion with the relative reduction of slow wave sleep in the 2h TIB condition (panel b).

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