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Naps not as effective as a night of sleep at dissipating sleep pressure

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Abstract: The two-process model of sleep posits that two processes interact to regulate sleep and wake: a homeostatic (Process S) and a circadian process (Process C). Process S compensates for sleep loss by increasing sleep duration and intensity. Process C gates the timing of sleep/wake favouring sleep during the circadian night in humans. In this study, we examined whether taking six naps throughout a 24-hr period would result in the same amount of dissipation of homeostatic pressure at the end of the day as a night of sleep, when time in bed is equivalent. Data from 46 participants (10-23 years; mean = 14.5 [$\pm$ 2.9]; 25 females) were analysed. Slow-wave energy, normalized to account for individual differences in slow-wave activity, was used as a measure of sleep homeostasis. In the nap condition, slow-wave energy of six naps distributed equally during a 24-hr period was calculated. In the baseline condition, slow-wave energy was measured after 9-hr time in bed. A paired t-test was used to compare nap and baseline conditions. A linear regression was used to examine whether slow-wave energy varied as a function of age. Slow-wave energy was greater during baseline than the nap condition ($p < .001$). No association between age and slow-wave energy was found for baseline or nap conditions. Our findings indicate that multiple naps throughout the day are not as effective at dissipating sleep pressure as a night of sleep. This is likely due to the influence of the circadian system, which staves off sleep during certain times of the day.

DOI: <https://doi.org/10.1111/jsr.13295>

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ZORA URL: <https://doi.org/10.5167/uzh-207889>

Journal Article

Accepted Version

Originally published at:

Tarokh, Leila; Van Reen, Eliza; Achermann, Peter; Carskadon, Mary A (2021). Naps not as effective as a night of sleep at dissipating sleep pressure. *Journal of Sleep Research*, 30(5):e13295.

DOI: <https://doi.org/10.1111/jsr.13295>

Naps not as Effective as a Night of Sleep at Dissipating Sleep Pressure

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Summary

The two-process model of sleep posits that two processes interact to regulate sleep and wake: a homeostatic (Process S) and a circadian process (Process C). Process S compensates for sleep loss by increasing sleep duration and intensity. Process C gates the timing of sleep/wake favoring sleep during the circadian night in humans. In this study we examined whether taking six naps throughout a 24-h period would result in the same amount of dissipation of homeostatic pressure at the end of the day as a night of sleep, when time in bed (TIB) is equivalent. Data from 46 participants (10 to 23 years; mean = 14.5 (± 2.9); 25 females) were analyzed. Slow wave energy (SWE), normalized to account for individual differences in slow-wave activity, was used as a measure of sleep homeostasis. In the nap condition SWE of six naps distributed equally during a 24-h period was calculated. In the baseline condition, SWE was measured after 9-h TIB. A paired t-test was used to compare nap and baseline conditions. A linear regression was used to examine whether SWE varied as a function of age. SWE was greater during baseline than the nap condition ($p < 0.001$). No association between age and SWE was found for baseline or nap conditions. Our findings indicate that multiple naps throughout the day are not as effective at dissipating sleep pressure as a night of sleep. This is likely due to the influence of the circadian system, which staves off sleep during certain times of day.

Keywords: Slow wave energy, slow wave activity, sleep homeostasis, adolescence, naps, circadian, Sleep EEG

Introduction

Two processes gate the timing and duration of sleep – a homeostatic and a circadian process (Achermann and Borbely, 2017). The homeostatic process is dependent on prior wake – sleep history and dissipates sleep pressure by both sleep duration and intensity. Following a night of truncated sleep, the amount of deep sleep, indexed by EEG slow wave activity (SWA; NREM sleep EEG power 0.4 to 4.6 Hz), increases proportional to the amount of sleep lost (reviewed in (Achermann and Borbely, 2017)). Concurrently, sleep duration is prolonged, however not hour-for-hour to the amount that was lost. This gap is in part due to the influence of the circadian system that pushes the organism towards waking in the morning hours and at particular times of the day, called wake-maintenance zones. The degree to which sleep loss is compensated by increased sleep intensity or duration is still unclear, although to a large degree compensation is related to intensity (Achermann and Borbely, 2017).

Data show that daytime naps, of sufficient duration containing slow waves dissipate sleep pressure and result in less SWA the following night (Werth et al., 1996). Furthermore, other evidence shows that daytime naps and overnight sleep have somewhat overlapping functions. For example, sleep dependent memory consolidation is found for naps and overnight sleep, and naps have been suggested as effective as a night of sleep for such memory consolidation (Mednick et al., 2003).

Questions have arisen regarding the benefit or necessity of one long consolidated sleep episode at night from recent historical reports that our human ancestors may have split sleep into two episodes. Such reports question whether split nocturnal sleep is a more appropriate sleep behavior. Experimental support for this hypothesis comes from a study by Wehr in 1992, in which participants were confined to a dark room for 14 h per night (Wehr, 1992). Wehr found that under these conditions, after compensating for existing sleep debt before entering the study, participants' sleep was divided into two bouts of several hours (evening, morning bouts), separated by 1 to 3 hours of waking in between.

The aim of the current study was to examine whether six naps distributed throughout 24 hours are as effective at dissipating sleep pressure as a night of sleep. To this end, we use a composite measure of sleep duration and intensity, namely slow wave energy (SWE, i.e. cumulative SWA) to measure the degree of dissipated sleep pressure (e.g., Achermann and Borbely, 1990). Based on previous research showing a strong circadian influence on sleep duration and SWA (e.g., Lazar et al., 2015), we hypothesize that six naps will not be as effective as a night of sleep in dissipating sleep pressure.

Methods

Data from 46 participants between ages 10 and 23 years (mean age = 14.5 ($\pm$ 2.9) years; 25 females) were analyzed. All participants or their parents (assent was obtained from participant) gave informed consent and the Institutional Review Board (IRB) for the Protection of Human Subjects of Lifespan Hospital approved the study. Exclusion criteria included chronic or current illness, evidence of learning disability, sleep disorder, and personal or family history of psychopathology. Additionally, individuals with a pattern of insufficient sleep or excessive daytime sleepiness were not included in the study. A schematic of the study protocol can be found in Figure 1. Briefly, sleep at home for two weeks consisting of 10 h time in bed (TIB) was monitored using actigraphy, sleep diaries, and daily phone calls to the lab's answering machine at rise and sleep time. Subsequent to this, two nights of sleep consisting of 10 h TIB were monitored using polysomnography in the lab. On the third in-lab day, participants went to bed two hours later and were awakened 3 hours earlier, resulting in 5 h TIB. On days 4 to 6 participants slept for 1.5 h and were awake for 2.5 h six times during each 24 h period. Following the nap protocol, participants were kept awake for approximately 32 h and then permitted to sleep ad lib.

Polysomnographic recordings (four EEG derivations, EMG and EOG) were performed. Data were sampled at 250 Hz and scored in 30-s epochs according to standardized criteria (Rechtschaffen and Kales, 1968). EEG derivation C3/A2 was analyzed and power density spectra were calculated for 30-s epochs (Hanning window, average of six 5-s epochs). To account for artifacts, mean slow wave activity (power in the 0.6 to 4.6 Hz range; SWA) per five-minute interval was determined based on artifact free 30-s epochs and multiplied by the duration of NREM sleep in

this interval. These 5-minute intervals of SWA were cumulated over all naps (1.5 h naps x 6 times per day = 9 h time in bed TIB) resulting in a SWE value for each 24-h interval. Because TIB was 10 h on the baseline (night 2) night, and thus one hour longer than total TIB in the nap protocol, SWE on the baseline night was calculated for the first 9 hours after lights off. Thus, for both naps and baseline sleep SWE was computed over approximately 9 hours TIB.

Because our sample included of a broad age range, and developmental decline in SWA is well documented across adolescent development (e.g., (Tarokh and Carskadon, 2010)), we normalized all data points to the mean SWE at the end of the sleep opportunity on baseline and adaptation nights for each subject. Thus, SWE at the end of the baseline night is close to one. The mean normalization value was $178,780 \mu V^2 \cdot \text{min}$ (SD= 118,890), and was correlated with age ($r=-0.56$; $p < 0.0001$).

For this analysis, a repeated measure ANOVA was used to compare SWE on the three napping (in-lab day 6 to 9) days and the baseline night of sleep (four factors: nap day 1, 2, 3 and baseline sleep). In the case of a significant main effect, pairwise comparisons were performed with paired t-tests. A correlation analysis was used to examine whether normalized SWE was associated with age.

Results

On average, normalized SWE at the end of the baseline night was 1.03 (SD= 0.09 and was higher than SWE at the end of the nap protocol (nap day 1: 0.92 (SD=0.19); nap day 2: 0.93 (SD=0.21); nap day 3: 0.86 (SD=0.22)). The difference between the baseline and nap protocol was significant for the first ($t(45)= -3.45$; $p = 0.001$), second ($t(45)= -2.811$; $p = 0.007$), and third ($t(45) = -4.73$; $p < 0.0001$) day of naps (Figure 1). There were no differences between the three nap days (nap 1 versus 2, $t(45) = -0.39$, $p = 0.69$; nap 1 versus 3, $t(45) = 1.65$, $p = 0.10$).

We then investigated whether normalized SWE showed systematic changes with age. Since there were no difference between napping conditions, we analyzed the third day of naps since this day was furthest from the short night of sleep following the baseline night. We found no age association for the baseline ($r=0.08$; $p = 0.61$) or nap ($r=0.002$; $p=0.99$) conditions.

Sleep stages and SWE for the baseline night and three nap days can be found in Table 1. As expected in the nap protocol, sleep duration was longer at clock times corresponding to the biological night but short during the wake maintenance zone (Strogatz et al., 1987) (naps 2 and 3). This finding was also apparent in our measure of SWE, which showed a dip during the wake maintenance zone (Table 1). We note that there was a slight shift in phase due to the forced de-synchrony protocol. On average dim light melatonin onset which was captured on the short night (see Figure 1) was at 21.36 (standard deviation = 0.89; range = 18.65 to 23.46) and circadian period was 24.23 (standard deviation = 0.31; range = 23.49 to 25.10).

Discussion

We find that several naps distributed throughout the day are not as effective as a night of sleep at dissipating sleep pressure. To date, most nap studies have examined the impact of a single midday nap, typically 30 to 90 minutes in duration, on alertness, memory consolidation and subsequent nighttime sleep. These studies generally report a beneficial impact of napping on alertness and memory consolidation (e.g., (Hilditch et al., 2017) (Mednick et al., 2002)), however, nighttime sleep following a nap is often lighter (e.g., less slow wave sleep) as compared to when no naps are taken (e.g., (Werth et al., 1996)).

The current study contrasts the efficacy of multiple naps distributed evenly across a 24-h period to a night of sleep. As expected, during certain times of day (i.e., the wake maintenance zone prior to nighttime sleep), sleep efficiency was low due to the influence of the circadian clock, resulting in diminished TST in the nap condition. The sleep lost at these sleep opportunities was not entirely compensated by a rise in SWA in subsequent sleep episodes, resulting in less SWE at the end of 24-h period of the nap protocol as compared to a night of sleep. While the forced desynchrony protocol resulted in a slight shift in phase, given that the protocol spanned three days, this shift was small and unlikely to influence the results. Indeed, SWE did not differ on the first versus the last day of naps.

Several studies from the 1980s addressed the ability of humans to sleep polyphasically. In one study of nine adults, participants spent 72-h in isolation in underground apartments shielded from all time cues and were prohibited from reading, writing, listening to music and strenuous exercise. Under these conditions, not only was sleep duration longer (mean = 10.4 versus 7-8 hours under normal conditions) but multiple sleep episodes occurred in addition to the major sleep episode defined as sleep > 6 h during the biological night (Campbell and Zulley, 1985). The findings from this study were later confirmed and expanded (Wehr, 1992). Another study of polyphasic sleep examined sleep and performance in 99 sailors in solo or double-handed ocean sailing races, an extreme condition under which long sleep bouts are not possible because the sailor must keep the ship on course for several days at a time (Stampi, 1988). Under these conditions, mean TST was 6.3 h (SD 1.77 h), however, the average duration of each episode was 2.02 h (SD 1.7 h). Importantly, the sailors were able to maintain performance and maintain this schedule over long periods (20 to 49 days). Unfortunately, in the current study we do not include measures of performance and as has previously been shown (e.g., Van Dongen et al., 2003) performance and SWA may show differential recovery timelines.

The perceived benefits of sleeping less by fragmenting sleep episodes has attracted interest due to internet proliferation of interest in polyphasic sleep with the goal of reducing total sleep time. The recommendations for polyphasic sleep come in different forms, and have been covered by media outlets (e.g., (Ducharme, 2018)) who have reported that polyphasic sleep schedules have caught on among those in high pressure jobs (e.g., Silicon valley). The most popular of such schedules, the Uberman schedule consists of six 20-min naps spaced equally across the day, with anecdotal claims from proponents that such schedules increase focus and productivity despite diminished TST (Moyer, 2019). In addition to a large body of evidence showing the importance of adequate and appropriately timed sleep on wellbeing, our study which mimics the timing of the Uberman naps (albeit with significantly more sleep opportunity) provides further direct evidence refuting this claim.

When we look at sleep physiology and the presumed role of sleep intensity in recovery, one marker of this process, SWE that is accumulated across multiple naps,

shows that sleep pressure is not entirely dissipated under napping conditions as compared to a baseline night of sleep. In other words, sleep loss cannot be entirely compensated by an increase in SWA during naps timed to coincide with the trough in the circadian system, and further recovery time at an appropriate time of day is needed. Taken together, our novel study design allows us to add to the existing literature about the interaction of the circadian and homeostatic systems, finding that sleep lost at peaks in the circadian timing system cannot be compensated by an increase in SWA at naps coinciding with the circadian trough. Thus, both sleep duration and intensity are important for achieving sleep dependent recovery as indexed by SWE.

	BSL	Nap 1	Nap 2	Nap 3	Nap 4	Nap 5	Nap 6
TST (min)	545 ($\pm$ 30)	67.39 ($\pm$ 12.5)	44.84 ($\pm$ 22.5)	32.23 ($\pm$ 19.7)	78.06 ($\pm$ 4.9)	81.05 ($\pm$ 3.8)	79.86 ($\pm$ 7.8)
Awake^a (min)	29 ($\pm$ 29) ^a	22.08 ($\pm$ 12.5)	44.91 ($\pm$ 22.6)	57.46 ($\pm$ 19.8)	11.54 ($\pm$ 5.1)	8.32 ($\pm$ 3.7)	9.75 ($\pm$ 8.0)
Stage 2 (%)	51 ($\pm$ 5)	41($\pm$ 9)	41($\pm$ 15)	47 ($\pm$ 19)	39 ($\pm$ 11)	34 ($\pm$ 8)	39 ($\pm$ 9)
SWS (%)	21 ($\pm$ 7)	35 ($\pm$ 12)	33 ($\pm$ 18)	26 ($\pm$ 19)	40 ($\pm$ 15)	27 ($\pm$ 12)	32 ($\pm$ 10)
REM sleep (%)	19 ($\pm$ 3)	11 ($\pm$ 7)	7 ($\pm$ 7)	3 ($\pm$ 5)	11 ($\pm$ 8)	26 ($\pm$ 11)	18 ($\pm$ 8)
SWE (μV²)	189,280 ($\pm$ 129,520)	31,693 ($\pm$ 25,941)	22,205 ($\pm$ 19,211)	11,717 ($\pm$ 11,855)	42,002 ($\pm$ 33,271)	26,754 ($\pm$ 20,050)	31,366 ($\pm$ 18,531)

Table 1: Sleep stages averaged over subjects and three days of napping (standard deviations are across subjects). ^aWaking after sleep onset for baseline sleep; for naps, the total amount of waking during the scheduled sleep opportunities is reported. SWS: slow wave sleep (stages 3 and 4). Slow wave energy (SWE) is computed for the baseline night and for each nap opportunity as described in the methods.

Figure 1: Study protocol. Two nights of 10-h sleep opportunity (adaption followed by a baseline night) were followed by a short night of sleep (short night) consisting of 5-h TIB. Following this short night, participants spent 3 days on an intermittent nap schedule (Nap day 1 to 3) with 1.5-hours sleep opportunity (Nap 1 to 6) followed by 2.5-hours waking period. In this figure, sleep opportunities are shown in black.

Figure 2 Violin plots showing slow-wave energy (SWE; normalized, see Methods) at the end of the nap protocols and 9-h after sleep onset in on the baseline night (BSL). Median values are shown in red, while the mean is in black. The thickness of the plot indicates the distribution of values. SWE was significantly higher in the baseline as compared to the three nap conditions indicating that more dissipation of sleep pressure occurred. The nap conditions were not significantly different from each other. The * denotes significance at $p < 0.05$.

Acknowledgements

The authors thank Jena Burner, Margaret Gordon-Fogelson, Erin McInrue, Gretchen Surhoff, Jared Saletin, David Bushnell, Ellyn Ferriter, Jon Lassonde, Sharon Driscoll, Marcy D'Uva, Ashten Bartz, Dave Barker, and Caroline Gredvig. This work was supported by grants from the National Institute of Mental health (5R01MH076969 to MAC) and the Swiss National Science Foundation (SNSF grant 320030_130766 to PA).

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