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Comparing the cardiac autonomic activity profile of daytime naps and nighttime sleep

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ABSTRACT

Heart rate variability (HRV) is a reliable technique to evaluate autonomic activity and shows marked changes across a night of sleep. Previous nighttime sleep findings report changes in HRV during non-rapid eye movement sleep (NREM), which have been associated with cardiovascular health benefits. Daytime sleep, however, has been linked with both positive and negative cardiovascular outcomes. Yet, no studies have directly compared HRV profiles during an ecologically-valid daytime nap in healthy, well-rested adults to that of nighttime sleep. Using a within-subjects design, 32 people took a daytime nap and slept overnight in the lab at least one week apart; both sleep sessions had polysomnography, including electrocardiography (ECG), recorded. We measured inter-beat intervals (RR), total power (TP), low frequency power (LF; .04–.15 Hz), and high frequency power (HF; .15–.40 Hz) components of HRV during NREM and rapid eye movement (REM) sleep. Compared to the nap, we found longer RR intervals and decreased heart rate during the night for both Stage 2 and SWS and increased TP, LF and HF power during nighttime Stage 2 sleep only; however, no differences in the LFHF ratio or normalized HF power were found between the nap and the night. Also, no differences in REM sleep between the nap and night were detected. Similar relationships emerged when comparing the nap to one cycle of nighttime sleep. These findings suggest that longer daytime naps, with both SWS and REM, may provide similar cardiovascular benefits as nocturnal sleep. In light of the on-going debate surrounding the health benefits and/or risks associated with napping, these results suggest that longer daytime naps in young, healthy adults may support cardiac down-regulation similar to nighttime sleep. In addition, napping paradigms may serve as tools to explore sleep-related changes in autonomic activity in both healthy and at-risk populations.

1. Introduction

Sleep influences the cardiovascular system, with distinct changes in cardiac activity occurring at sleep onset and within individual sleep stages (Trinder et al., 2012). Fluctuations in cardiac activity across sleep and wake are understood to homeostatically regulate the balance between the sympathetic and parasympathetic branches of the autonomic nervous system (ANS), where waking is primarily supported by sympathetic activity and sleep is dominated by parasympathetic/vagal activity. Increases in parasympathetic activity during nighttime sleep have been correlated with reduced risk for cardiovascular disease (Thayer et al., 2010), leading researchers to describe nighttime sleep as a “cardiovascular holiday” (Trinder et al., 2012). In contrast, the relationship between cardiac autonomic activity and daytime sleep is not as clear. Epidemiological studies have reported that frequent napping is associated with both increased (Leng et al., 2014) and decreased (Naska et al., 2007) risk for cardiovascular mortality. While discrepancies between studies may be due to a range of confounding factors, including comorbid psychological and health problems as well as demographic and population differences; due to the lack of empirical investigations, the reasons for these discrepancies between studies is not known. However, circadian influences on both sleep (Borbély and Achermann, 1999) and cardiovascular activity (Guo and Stein, 2003) suggest daytime naps may confer less restorative benefit than a night of sleep; and though a similar cardiac profile between the two sleep phases has been suggested in healthy adults (Cellini et al., 2016, 2017), no study has directly compared the cardiac profile of a nap to a night of sleep, which is the goal of the current study.

The lack of empirical investigations into the relationship between napping and cardiovascular modulation is surprising considering that cardiovascular disease is the leading cause of death in the United States (Benjamin et al., 2017) and, according to the National Sleep

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Foundation, 53% of adults in America nap at least once in a 7-day period for at least 15 min a day (National Sleep Foundation, 2014). In other healthy, adult samples, 60–75% of adults' nap at least one time in a 7-day week with average nap durations of about 70 min (Dinges, 1992; Pilcher et al., 2010). Considering napping is a common practice, it is important to determine if and under which conditions naps have the potential to either exacerbate or reduce the risk for cardiovascular disease. Additionally, experimental studies increasingly use naps to assess the effect of sleep for cognitive fitness and memory consolidation. Even more, cardiac, autonomic activity during naps has recently been shown to be a strong predictor of memory improvement (Whitehurst et al., 2016). Therefore, a thorough, direct assessment comparing the autonomic profile of a nap and nighttime sleep is necessary.

A typical measure of cardiac, autonomic activity is heart-rate variability (HRV), a non-invasive, well-established technique, extracted from the beat-to-beat interval (RR interval) and detected from an electrocardiogram (ECG). Two oscillatory components that characterize the RR interval –the low frequency component (LF; .04–.15 Hz) and the high frequency component (HF; .15–.40 Hz) –have been independently tied to underlying ANS mechanisms (Tobaldini et al., 2013). The HF component is partially representative of parasympathetic outflow and vagal activity (Berntson et al., 1997), and while consensus is lacking regarding the interpretation of the LF component (Billman, 2013), it is purported to account for sympathetic activity and some vagal modulations (Stein et al., 1994; Berntson et al., 1997; Malliani et al., 1991).

In healthy adults, sleep onset is characterized by a reduction in overall heart rate (i.e., a lengthening in the RR interval) and increased HF power (Trinder et al., 2012). As people cycle through non-rapid eye movement sleep (NREM), comprised of stages 1–3 [(with Stage 3 commonly referred to as slow wave sleep SWS)], studies have shown a continued lengthening of RR intervals, decreased LF, but not HF power, and decreased LF/HF ratios compared to both wake and REM sleep (Trinder et al., 2001). These findings indicate an overall reduction in cardiovascular output and dominance of parasympathetic and vagal activity during NREM sleep.

Here, we utilized a within-subjects design with each subject undergoing a 2-hour daytime nap opportunity and a night of sleep in the lab, at least one week apart, while being monitored with polysomnography (PSG), including electrocardiography (ECG). We assessed cardiac autonomic activity within each sleep stage (Stage 2, SWS, and REM) of the nap and night condition. We anticipated similar autonomic activity for all HRV variables across both nap and night conditions.

2. Materials and methods

Thirty-two healthy, non-smoking participants between the ages of 18 to 35 (M_{age} = 21.00 ± 2.4 years, 16 Females) with no personal history of neurological, psychological, or other chronic illness provided informed consent, which was approved by both the Western Institutional Review Board and the University of California, Riverside Human Research Review Board. Participants received monetary compensation and/or course credit. Individuals were excluded from participation if they reported: irregular sleep/wake cycles; a sleep disorder (determined during an in-person interview); personal or familial history of diagnosed psychopathology; substance abuse/dependence; loss of consciousness greater than 2 min or a history of epilepsy; current use of psychotropic medications; and any cardiac or respiratory illness that may affect cerebral metabolism.

2.1. Protocol

All participants wore an actigraph and completed daily sleep diaries for one week prior to in-lab visits to ensure participants were not sleep-deprived, received an average of 7 h of sleep per night, and spent at least 6.5 h in bed the night prior to each visit. Each participant had a daytime nap and a nighttime sleep session (on different days) monitored with PSG and ECG. For the nap session, participants were allotted 2 h of time in bed between 1:30–3:30PM and a trained sleep technician monitored all naps in real-time to ensure subjects were asleep within 30 min of lights out. Midday naps, which provide normal, healthy sleepers the opportunity to fully cycle through Stage 2, SWS, and REM, were used to optimize the chance that all subjects would obtain adequate time in each sleep stage. Nighttime sleep was recorded from subjects on a day other than their nap day (at least 1 week apart and counterbalanced) to eliminate confounds due to reductions in sleep pressure after the nap.

2.2. Polysomnography (PSG)

PSG data were collected using Astro-Med Grass Heritage Model 15 amplifiers with Grass GAMMA software. Scalp electroencephalographic (EEG) and electrooculagraphic (EOG) electrodes were referenced to unlinked contralateral mastoids (F3/A2, F4/A1, C3/A2, C4/A1, P3/A2, P4/A1, O1/A2 O2/A1, LOC/A2, ROC/A1). Two submental muscle tone electromyographic (EMG) electrodes were attached under the chin and referenced to each other. High pass filters were set at .3 Hz and low pass filters at 35 Hz for EEG and EOG. Raw data were visually scored in 30-sec epochs according to the Rechtschaffen & Kales' (1968) manual.

2.3. Cardiac assessment

Electrocardiogram (ECG) data were collected at 256 Hz sampling rate using a modified Lead II Einthoven configuration. R-wave peaks were detected automatically by Kubios HRV Analysis Software 2.0 (Matlab, Kuopio, Finland), visually examined, and edited for artifacts. An independent lab tool was employed to perform the HRV analysis of the R-waves series through both nap and nighttime sleep periods according to the Task Force of the European Society of Cardiology and North American Society of Pacing and Electrophysiology guidelines (Camm et al., 1996). Missing and ectopic beats were corrected via cubic spline interpolation. Inter-beat intervals were computed and a third order polynomial filter was applied on the time series to remove non-stationary trend components and increase estimation of the LF component (Tarvainen et al., 2014; Weber et al., 1992). An autoregressive model (model order set at 16; Boardman et al., 2002) was employed to quantify the absolute spectral power (ms^2) in the LF and HF frequency bands and the total power (TP), which reflects total HRV. The LF, HF, and TP measures had skewed distributions and as such were transformed by taking the natural logarithm. From these variables we derived the HF normalized units (HF_{nu} = (HF [ms^2]/ HF [ms^2] + LF [ms^2])*100) and the LFHF ratio (LF / HF). Since the LF normalized units are mathematically reciprocal to HF_{nu} (i.e. LF_{nu} =1- HF_{nu}), to avoid redundancy, we computed only the HF_{nu} index. Additionally, we included the RR interval as a major index of cardiac autonomic control (Pinna et al., 2007) as well as heart rate (HR), measured as beats per minutes (bpm), in our analyses.

For the analysis of RR, HR and frequency-domain HRV measures during different sleep stages, consecutive, artifact-free windows of undisturbed sleep were selected across both the night and nap. Each window was 3-min in duration and the 2-min preceding and the entire 3-min epoch were free from stage transitions. Windows were identified and averaged within Stage 2, SWS and REM sleep. Epochs of Stage 1 and wake after sleep onset were not analyzed (Trinder, et al., 2001).

2.4. Data reduction and statistical analyses

Our method of HRV analyses emphasizes consolidated sleep stages. As a result, for subjects that could not attain at least 5 min of undisturbed sleep in any given sleep stage, we were unable to calculate HRV parameters. This disproportionately impacted the nap condition with four subjects not acquiring enough consolidated SWS and three

subjects not acquiring enough consolidated REM. Additionally, the ECG lead was disturbed in three nighttime and nine nap recordings, and as a result, the RR interval could not be calculated during SWS or REM. These records were removed from those analyses. Lastly, one subject was removed as an outlier (values greater than 2.5 standard deviations from the mean). The final sample size for nighttime records was as follows Stage 2 = 29; SWS = 28; REM = 29 and the final sample sizes for the nap records was as follows: Stage 2 = 30; SWS = 18; REM = 19.

First, to ensure that the cardiac profile in our sample was similar to that previously reported, we performed repeated measures ANOVAs on the nighttime records for each of the HRV variables (RR, HR, LF, HF, TP, HFnu, LFHF) with sleep stage (Stage 2, SWS, and REM) as a within-subjects factor. The Huynh-Feldt correction was applied when sphericity assumptions were violated. Tukey's HSD test with the Bonferonni correction were used for post-hoc comparisons and partial eta squared (η_p^2) is reported for effect size.

Here, our main goal was to directly assess if HRV parameters differ during a nap compared to nighttime sleep. To examine this goal, we conducted three multivariate ANOVA's for each sleep stage (Stage 2, SWS, REM) with HRV parameters (HR, RR, LF, HF, TP, HFnu, LFHF) as dependent variables and condition (Nap vs Night) as a fixed factor. We included minutes in each sleep stage (Stage 2, SWS, REM) as a covariate in each model and report partial eta squared (η_p^2) for effect size.

Due to homeostatic and circadian controls of sleep, there are variations in the amount of sleep present in each sleep cycle, with the first sleep cycle comprised primarily of SWS sleep, the last sleep cycle typically presenting with increased REM and intermediate cycles having comparable amounts of both SWS and REM (see Borbély and Achermann, 1999 for full description). To determine if cardiac autonomic activity during a daytime nap is analogous to a cycle of nighttime sleep, we conducted secondary analyses where we compared each of our HRV parameters during the nap to a sole sleep cycle. Here, we used the second cycle of nighttime sleep to be able to compare the nap to adequate amounts of each nighttime sleep stage: Stage 2 (N = 29), SWS (N = 26), and REM sleep (N = 27; for definition of cycles, please see Feinberg and Floyd, 1979). We utilized the same statistical MANOVA models as in the full night analyses. Here, we also controlled for the number of minutes contributing to the HRV analysis in that cycle of sleep.

3. Results

3.1. HRV profile during nighttime sleep

Sleep architecture for both nights and naps can be found in Table 1. Nighttime sleep showed significant differences in RR intervals across sleep stages, $p < 0.001$, $\eta_p^2 = 0.30$ with longer RR intervals during NREM Stage 2 when compared to REM ($p < .001$), as well as longer RR intervals during Stage 2 when compared to SWS ($p < 0.001$). However, we found no difference between RR intervals during SWS and REM (p = 0.42). A similar relationship was found for HR with higher heart rate in SWS (p = 0.001) and REM (p < 0.001) when compared to Stage 2, however no differences between SWS and REM (p = 0.12). We also found changes in TP across nighttime sleep states, $p < 0.001$, $\eta_p^2 = 0.48$, with higher TP in both Stage 2 ($p < .001$) and REM ($p < .001$) when compared to SWS. Changes in TP across sleep states was derived from changes in both LF ($p < 0.001$, $\eta_p^2 = 0.53$) and HF activity ($p = 0.002$, $\eta_p^2 = 0.22$). Specifically, LF activity was lowest in SWS and significantly lower during SWS when compared to both Stage 2 ($p < 0.001$) and REM ($p < 0.001$), and HF activity was lower in SWS when compared to Stage 2 (p = 0.001). No differences emerged between Stage 2 and REM sleep for either LF (p = 0.73) or HF activity (p = 0.74). These changes in raw power were also reflected in both HFnu ($p < 0.001$, $\eta_p^2 = 0.61$) and LFHF ($p < 0.001$, $\eta_p^2 = 0.56$). For HFnu, we found increased power during SWS compared to both Stage 2 ($p < .001$) and REM ($p < .001$), and increased activity in Stage 2 compared to REM ($p < 0.001$). In contrast, for LFHF, the lowest ratio emerged during SWS, which was significantly lower than both Stage 2 ($p < .001$) and REM sleep ($p < 0.001$). Stage 2 also had a lower LFHF ratio when compared to REM ($p < .001$). For nap sleep stage comparisons see Supplemental Fig. 1.

In line with previous literature, these data replicate the relative dominance of parasympathetic (HF_{nu}) activity during NREM when compared to REM sleep, which is driven by a reduction in LF activity and an increase in HF activity during NREM sleep (Table 2, *see* Fig. 1*a-f*).

3.2. HRV profile of a nap vs. nighttime sleep: Whole night analyses

Comparing nap vs. night, after controlling for minutes in each sleep stage, we found differences in Stage 2 across a nap and a night, $p = 0.05$, $\eta_p^2 = 0.21$. Specifically, during Stage 2 sleep, we found significantly longer RR intervals ($p = 0.001$, $\eta_p^2 = 0.16$) and lower HR ($p = 0.002$, $\eta_p^2 = 0.15$) at night compared to the nap. Additionally, there was higher TP ($p = 0.01$, $\eta_p^2 = 0.11$), LF ($p = 0.04$, $\eta_p^2 = 0.07$) and HF ($p = 0.03$, $\eta_p^2 = 0.08$) activity during the night compared to the nap. No differences in the LFHF ratio $p = 0.47$, $\eta_p^2 = 0.009$ or HF_{nu} power $p = 0.47$, $\eta_p^2 = 0.09$ emerged between the nap and night for Stage 2 sleep. For SWS, differences between RR intervals also emerged; a MANOVA showed longer RR intervals during nighttime sleep when compared to the nap, $p = 0.01$, $\eta_p^2 = 0.14$ coupled with a higher heart rate during the nap compared to the night ($p = 0.04$, $\eta_p^2 = 0.09$). No other differences between a nap and a night in SWS were present. Additionally, none of the seven cardiac parameters differed across a night and a nap for REM sleep ($p = 0.59$, $\eta_p^2 = 0.10$).

Table 1
Demographics.

	Mean (*SD*)	N (%)
Age (years)	20.77 (*2.20*)	
BMI (kg/m^2)	23.26 (*3.31*)	
Female/Male		16 / 16 (*50 / 50*)
Ethnicity		
African-American		2 *(6)*
Asian		4 *(13)*
Hispanic		15 *(47)*
Caucasian		3 *(9)*
Mixed (2 or more)		4 *(13)*
Other		4 *(13)*

Note: BMI = Body Mass Index; SD = Standard Deviation.

Table 2
Descriptive statistics for nap and nighttime sleep.

	Nap			Night		
	Mean	Min	Max	Mean	Min	Max
TST	96.36 (19.56)	36.50	116.50	480.53 (66.67)	162.00	571.00
Stage 1	14.34 (17.29)	3.00	101.00	7.02 (5.13)	0	20.50
Stage 2	50.45 (21.06)	0	83.00	256.23 (51.64)	96.50	356.50
SWS	17.86 (14.61)	0	54.00	96.08 (32.45)	24.00	147.00
REM	13.70 (12.85)	0	54.00	121.20 (39.50)	40.50	217.00
WASO	12.36 (16.20)	0.50	67.50	30.63 (11.50)	14.00	52.50
SL	5.92 (6.28)	1.00	27.50	9.52 (12.12)	0.50	63.50
SE %	83.52 (16.58)	30.40	96.80	92.15 (3.18)	83.30	97.20

Note: Mean (Standard Deviation); TST = Total Sleep Time; SWS = Slow Wave Sleep; REM = Rapid Eye Movement sleep; WASO = Wake After Sleep Onset (calculated as the minutes of wake after first epoch of sleep); SL = Sleep Latency (calculated as the time to first epoch of sleep); SE = Sleep Efficiency. All stats are represented in minutes besides SE which is in percentage.

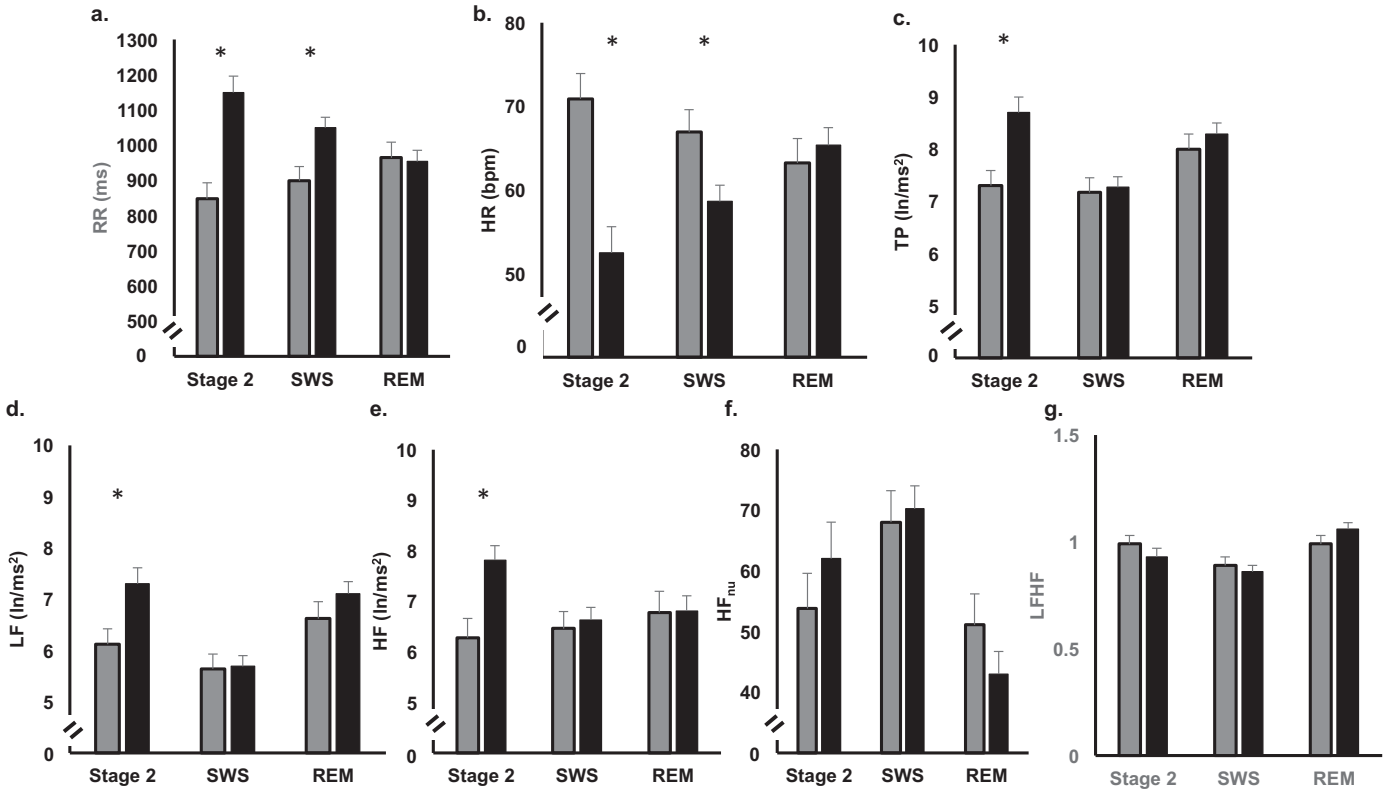

Fig. 1. a-g. Cardiac HRV parameters across a daytime nap (grey bars) and nighttime sleep (black bars). Asterisks indicate significant differences ($p < 0.05$) between nap and night within the sleep stage for the given HRV parameter. Error bars represent standard error of the mean.

3.3. HRV profile of a nap vs. nighttime sleep: cycle analyses

Next, we examined how the daytime nap compared to the second cycle of nighttime sleep. Similar to the whole night analyses, for Stage 2, we found significant differences between the second cycle of nighttime sleep and a daytime nap ($p = 0.009$, $\eta_p^2 = 0.28$) with a lower heart rate at night compared to the nap ($p = 0.002$, $\eta_p^2 = 0.16$), as well as higher LF ($p = 0.02$, $\eta_p^2 = 0.09$), HF ($p = 0.04$, $\eta_p^2 = 0.07$) and TP ($p = 0.03$, $\eta_p^2 = 0.08$) activity. Importantly, for Stage 2 sleep, we found no difference between LFHF ($p = 0.46$, $\eta_p^2 = 0.01$) or HFnu ($p = 0.30$, $\eta_p^2 = 0.02$). Even more, no differences between the second cycle of SWS ($p = 0.18$, $\eta_p^2 = 0.21$) or REM sleep ($p = 0.73$, $\eta_p^2 = 0.07$) and the daytime nap were detected. Reliability between cycles for nighttime sleep can be found in Supplemental Table 1.

4. Discussion

In the current study, we directly compared alterations in cardiac, autonomic activity during both a daytime nap and nighttime sleep in a within-subjects design. Similar to previous studies, nighttime sleep showed dominance of parasympathetic activity during NREM sleep when compared to REM, which was reflected in decreased LF power and reduced LFHF ratios, as well as increased HF and HF_{nu} power during both Stage 2 and SWS when compared to REM.

Comparing a nap to a night, in Stage 2 only, we found higher TP, as well as higher HF and LF activity during nighttime sleep compared with naps, and in Stage 2 and SWS, we found longer RR intervals and lower heart rates at night compared to the night. No differences in REM sleep were found between night and daytime sleep, and importantly, no differences in either the LFHF ratio or HFnu power were present for any of the sleep stages. These relationships were maintained in our cycle analyses where we compared HRV features during the second cycle of nighttime sleep to respective aspects of the nap. This suggests that the relative increases in parasympathetic activity present during nighttime sleep, and related to cardiovascular health, are also present during a daytime nap. These data suggest that, like a night of sleep, a nap can serve as a "mini-cardiovascular" break.

4.1. HRV: Naps vs Nocturnal sleep

Our results extend previous studies on daytime sleep (Cellini et al., 2016, 2017), and the role of circadian effects on sleep-dependent cardiac profiles (Boudreau et al., 2013). One comparable study (Boudreau et al., 2013) used a forced 72-hour ultradian sleep/wake protocol and found that there are less sleep-pronounced changes in RR, HF, LF and LFHF ratios during the day when compared to the night. We found a similar effect here, with shorter RR intervals, higher TP, LF and HF activity during the day compared to the night. However, importantly, changes in TP, LF and HF activity were only found to differ in Stage 2 sleep and were not as widely prevalent across sleep stages as previously reported (Boudreau et al., 2013). However, due to the strict circadian control of the Boudreau et al. (2013) study (one hour nap opportunities every other hour for three days), the amount of sleep observed (mean nap time 27 min) was limited, which consequently truncated the amount of time in each sleep stage used for analyses. Furthermore, sleep-deprivation may have played a role in the prior results. Here, using a paradigm in which well-rested subjects were provided a 2-hour daytime nap opportunity, we expand upon these prior findings and suggest instead that daytime circadian effects may emerge the most strongly during lighter, Stage 2 sleep. On the other hand, during deeper sleep states (SWS and REM), sleep regulatory effects appear more pronounced. This suggests that longer daytime naps that include SWS and REM sleep (e.g., 45–90 min) may show more cardiac benefits.

4.2. Cardiovascular health, cognitive fitness and napping behaviors

There has been a significant debate in the literature surrounding the benefits of napping for health and cognition, with some research showing that napping is correlated with increased nighttime sleep disruption (Owens et al., 2010), excessive daytime sleepiness and depression (Foley et al., 2007), as well as cardiovascular mortality (Leng et al., 2014); while others report associations between napping with normal nighttime sleep, as well as distinct cognitive and health benefits including increased cognitive alertness, decreased daytime sleepiness, and decreased risk for heart attack and coronary death (Jung et al., 2013; Leng et al., 2014; Dhand and Sohal, 2006; Gillberg et al., 1996; Naska et al., 2007). These discrepancies may be related to a range of factors, including comorbid psychological and health problems and demographic differences as well as cultural context. In addition, reasons for napping may predict differences in nap benefits. In a college sample, Duggan et al. (2016) used factor analysis to categorize people's reasons for napping into a five-factor DREAM model: "Dysregulative" (napping due to disturbed sleep schedules), "Restorative" (napping to prepare or make up for sleep loss), "Emotional" (napping due to stress, depression or boredom), "Appetitive" (napping for enjoyment), and "Mindful" (napping to increase cognitive ability). These factors were compared with self-reported sleep, psychological, and physical health. The findings suggest that only Emotional reasons for napping were associated with negative health outcomes (Duggan et al., 2016). No studies have examined whether nap habits and reasons for napping may be related to health predictors. Additionally, current studies on napping do not discern between planned and accidental naps. This may be an important distinction as it has been shown that planned naps are associated with cognitive and health benefits, while accidental naps are suggestive of an underlying sleep or health disorder (Dhand and Sohal, 2006). Taken together, these results suggest that understanding why and how people are napping is essential when considering potential health or psychological outcomes.

4.3. Limitations

The current sample was comprised of young, healthy adults with no history of physical or psychiatric illness. Many studies reporting negative associations between napping and health outcomes utilize older and/or unhealthy populations (Jung et al., 2013; Leng et al., 2014). Considering cardiac activity is a significant factor for populations at risk for cardiovascular disease (Legramante and Galante, 2005) and HRV assessments during sleep have been shown to be useful in risk stratification, or determination of an individual's need for preventative treatment (Stein and Pu, 2012), it would be helpful to measure HRV profiles during daytime sleep in clinical samples to gain a better understanding of sleep and cardiovascular modulations in healthy and at-risk populations. Additionally, research has shown that hormonal fluctuations associated with female's menstrual phase can interact with both nap (Genzel et al., 2012) and nighttime sleep features (Baker and Driver, 2007). In future investigations, accounting for menstrual cycle related fluxes is an important consideration.

Another limitation of this study is the reduction in subject numbers due to HRV methodological constraints. Specifically, standard practice for HRV analyses (Camm et al., 1996; Cellini et al., 2015) requires assessment of HRV over a five-minute period of a consistent sleep stage. Due to the large amount of sleep transitions present in a daytime nap, this approach limits the total amount of data available and likely impacts statistical power. Specifically, 5 min of undisturbed sleep in each sleep stage is not as common in daytime naps and reduces the total sample. While we don't believe that the reduction in power was a substantially limiting factor in the current study, in order to retain more statistical power, future studies should explore alternative methodological approaches to HRV assessment.

Conflicts of interest

None.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.nbscr.2018.03.001.

References

Baker, F.C., Driver, H.S., 2007. Circadian rhythms, sleep and the menstrual cycle. Sleep Med. 8, 613–622. http://dx.doi.org/10.1016/j.sleep.2006.09.011.
Berntson, G.G., et al., 1997. Heart rate variability: origins, methods and interpretive caveats. Psychophysiology 34, 623–648.
Benjamin, E.J., et al., 2017. On behalf of the American Heart Association Statistics Committee and Stroke Staitstics Subcommittee. Heart disease and stroke statistics—2017 update: a report from the American Heart Association. Circulation. http://dx.doi.org/10.1161/CIR.0000000000000485.
Billman, G.E., 2013. The LF/HF ratio does not accurately measure cardiac sympatho-vagal balance. Front. Physiol. 4, 26.
Boardman, A., Schlindwein, F.S., Rocha, A.P., Leite, A., 2002. A study on the optimum order of autoregressive models for heart rate variability. Physiol. Meas. 23, 325–336.
Borbély, A.A., Achermann, P., 1999. Sleep homeostasis and models of sleep regulation. J. Biol. Rhythm. 14, 557–568.
Boudreau, P., Yeh, W.-H., Dumont, G.A., Boivin, D.B., 2013. Circadian variation of heart rate variability across sleep stages. Sleep 36, 1919.
Camm, A., Malik, M., Bigger, J., Breithardt, G., Cerutti, S., Cohen, R., Kleiger, R., 1996. Heart rate variability: standards of measurement, physiological interpretation and clinical use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Circulation 93, 1043–1065.
Cellini, N., Torre, J., Steggno, L., Sarlo, M., 2017. Cardiac autonomic activity during daytime nap in young adults. J. Sleep Res. http://dx.doi.org/10.1111/jsr.12539.
Cellini, N., Whitehurst, L.N., McDevitt, E.A., Mednick, S.C., 2016. Heart rate variability during daytime naps in healthy adults: autonomic profile and short-term reliability. Psychophysiology 53, 473–481.
Dhand, R., Sohal, H., 2006. Good sleep, bad sleep! The role of daytime naps in healthy adults. Curr. Opin. Pulm. Med. 12, 379–382.
Dinges, D.F., 1992. Adult napping and its effects on ability to function. In: Stampi, C. (Ed.), Evolution, Chronobiology, and Functions of Polyphasic and Ultrashort Sleep. Birkhauser, Boston, pp. 118–134.
Duggan, K.A., McDevitt, E.A., Whitehurst, L.N., Mednick, S.C., 2016. To nap, perchance to DREAM: a factor analysis of self-reported reasons for napping. Behav. Sleep Med. http://dx.doi.org/10.1080/15402002.2016.1178115.
Feinberg, I., Floyd, T.C., 1979. Systematic trends across the night in human sleep cycles. Psychophysiology 16, 283–291. http://dx.doi.org/10.1111/j.1469-8986.1979.tb02991.x.
Foley, D.J., Vitello, M.V., Bliwise, D.L., Ancoli-Israel, S., Monjan, A.A., Walks, J.K., 2007. Frequent napping is associated with excessive daytime sleepiness, depression, pain, and nocturia in older adults: findings from the National Sleep Foundation '2003 Sleep in America' poll. Am. J. Geriatr. Psychiatry 15, 344–350.
Genzel, L., Kiefer, T., Renner, L., Dressler, M., 2012. Sex and modulatory menstrual cycle effects on sleep related memory consolidation. Psychoneuroimmunology 37, 987–998. http://dx.doi.org/10.1016/j.psyneuen.2011.11.006.
Gillberg, M., Kecklund, G., Axelsson, J., Akerstedt, T., 1996. The effects of a short daytime nap after restricted night sleep. Sleep 19, 570–575.
Guo, Y., Stein, P.K., 2003. Circadian rhythm in the cardiovascular system: chronocardiology. Am. Heart J. 145, 779–786.
Legramante, J.M., Galante, A., 2005. Sleep and hypertension: a challenge for the autonomic regulation of the cardiovascular system. Circulation 112, 786–788.
Leng, Y., Wainwright, N.W., Cappuccio, F.P., Surtees, P.G., Hayat, S., Luben, R., Khaw, K.-T., 2014. Daytime napping and the risk of all-cause and cause-specific mortality: a 13-year follow-up of a British population. Am. J. Epidemiol. 179, 1115–1124.
Malliani, A., Pagani, M., Lombardi, F., Cerutti, S., 1991. Cardiovascular neural regulation explored in the frequency domain. Circulation 84, 482–492.
Naska, A., Oikonomou, E., Trichopoulou, A., Psaltopoulou, T., Trichopoulos, D., 2007. Siesta in healthy adults and coronary mortality in the general population. Arch. Int. Med. 167, 296–301.
National Sleep Foundation, 2014. Sleep Health Index. ⟨http://sleepfoundation.org/sleep-health-index⟩.
Owens, J.F., Buysse, D.J., Hall, M., et al., 2010. Napping, nighttime sleep, and cardiovascular risk factors in mid-life adults. J. Clin. Sleep Med. 6, 330–335.
Pilcher, J.J., Michalowski, K.R., Carrigan, R.D., 2010. The prevalence of daytime napping and its relationship to nighttime sleep. Behav. Med. 27, 71–76.
Pinna, G., Maestri, R., Torunski, A., Danilowicz-Szymanowicz, L., Szwoch, M., La Rovere, M., Raczak, G., 2007. Heart rate variability measures: a fresh look at reliability. Clin.

Sci. 113, 131–140.
Stein, P.K., Pu, Y., 2012. Heart rate variability, sleep and sleep disorders. Sleep Med. Rev. 16, 47–66.
Stein, P.K., Bosner, M.S., Kleiger, R.E., Conger, B.M., 1994. Heart rate variability: a measure of cardiac autonomic tone. Am. Heart J. 127, 1376–1381.
Tarvainen, M.P., Niskanen, J.P., Lippponen, P.O., Ranta-aho, Karjalainen, P.A., 2014. Kubios hrv-heart rate variability analysis software. Comp. Methods Prog. Biomed. 113, 210–220.
Thayer, J.F., Yamamoto, S.S., Brosschot, J.F., 2010. The relationship of autonomic imbalance, heart rate variability and cardiovascular disease risk factors. Int. J. Cardiol. 141, 122–131.
Tobaldini, E., Nobili, L., Strada, S., Casali, K.R., Braghiroli, A., Montano, N., 2013. Heart rate variability in normal and pathological sleep. Front. Physiol. 4. http://dx.doi.org/10.3389/fphys.2013.00294.
Trinder, J., Kleiman, J., Carrington, M., Smith, S., Breen, S., Tan, N., Kim, Y., 2001. Autonomic activity during human sleep as a function of time and sleep stage. J. Sleep Res. 10, 253–264. http://dx.doi.org/10.1046/j.1365-2869.2001.00263.x.
Trinder, J., Waloszek, J., Woods, M., Jordan, A., 2012. Sleep and cardiovascular regulation. Pflügers Arch. - Eur. J. Physiol. 463, 161–168. http://dx.doi.org/10.1007/s00424-011-1041-3.
Weber, E.J.M., Molenaar, C.M., van der Molen, M.W., 1992. A nonstationarity test for the spectral analysis of physiological time series with an application to respiratory sinus arrythmia. Psychophysiol 29, 55–65.
Whitehurst, L.N., Cellini, N., McDevitt, E.A., Duggan, K.A., Mednick, S.C., 2016. Autonomic activity during sleep predicts memory consolidation in humans. PNAS 113, 7272–7277.