Integrating nap and night-time sleep into sleep patterns reveals differential links to health-relevant outcomes

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SUMMARY
Both night-time sleep and nap behaviour have been linked consistently to health outcomes. Although reasons for napping are usually tied to night-time sleep, the majority of studies assess their effects independently. The current study thus aimed to examine the health relevance of patterns of sleep behaviour that take into account both night-time and daytime sleep habits. Night-time sleep, recorded during 7 days via actigraphy from 313 participants (aged 34–82 years) of the Midlife in the United States II Biomarker study, was assessed. Blood and urine specimens were assayed for noradrenaline, interleukin-6 and C-reactive protein. Participants self-reported nap behaviour, depressive symptoms, perceived chronic stress and the presence of medical symptoms and conditions. Overall, nappers ($n = 208$) showed elevated waist–hip ratios, C-reactive protein and interleukin-6 levels compared to non-nappers and reported more physiological symptoms and conditions (all $P \leq 0.019$). Within nappers, cluster analysis revealed three patterns of sleep behaviour—infrequent nappers with good night-time sleep, frequent nappers with good night-time sleep and nappers with poor night-time sleep. Nappers with poor night-time sleep thereby exhibited elevated noradrenaline levels, depressive symptoms and perceived stress scores compared to other groups (all $P \leq 0.041$). These findings support the idea that nap–health relationships are complex, in that frequency of napping and accumulation of nap sleep is not related linearly to health consequences. Assessing nap behaviour in conjunction with night-time sleep behaviour appeared crucial to elucidate further the health relevance of napping, particularly in terms of psychological health outcomes, including chronic stress and depressive symptoms.

INTRODUCTION

Sleep is crucial for maintaining both physical and mental health. Consequently, sleep problems and short sleep have been linked to depression (Goldstein and Walker, 2014; Luyster *et al.*, 2012), as well as cardiovascular morbidity, hypertension and obesity (Magee *et al.*, 2008; Mullington *et al.*, 2009). Furthermore, sleep-restricted individuals show elevated inflammatory processes, as indicated by elevated interleukin (IL)-6 and C-reactive protein (CRP) levels (Ferrie *et al.*, 2013; Haack *et al.*, 2007; Van Leeuwen *et al.*, 2009). However, more sleep does not necessarily equal better overall health. In fact, oversleeping has been shown to have negative physical and psychological health implications as well (Jennum *et al.*, 2014; Prather *et al.*, 2015). These findings emphasize the importance of maintaining a delicate balance in order to reap the full health benefits of sleep. Importantly, this does not apply only to night-time sleep, but to sleep occurring at any time during a 24-h period (Evans and Rogers, 1994; Torsvall *et al.*, 1989).

In addition to sleeping at night, approximately 50% of Americans report sleeping during the day (National Sleep Foundation, 2013). Although such a frequent behaviour, its health consequences are not well understood. Naps have been shown to improve cognitive performance, reduce subjective sleepiness and improve mood in healthy subjects and, furthermore, to restore IL-6 levels following sleep deprivation (Faraut *et al.*, 2011; Luo and Inoue, 2000; Milner and Cote, 2009; Vgontzas *et al.*, 2007). However, in midlife adults, napping is associated with higher body mass index

(BMI) and central adiposity (Owens *et al.*, 2010), and in older adults, frequent napping is associated with depressive symptoms, an increased risk for type 2 diabetes, and excess weight (Foley *et al.*, 2007; Hays *et al.*, 1996; Xu *et al.*, 2010).

Notably, variation in nap duration and frequency as well as methodological issues can complicate the determination of health consequences. For example, while laboratory studies can manipulate circumstances to address how napping contributes to health outcomes, they may not be generalizable to natural sleep behaviour. Conversely, observational sleep studies provide insight into naturally occurring sleep behaviour, but often exclude nap information, use incidence of napping as a classifier only or focus upon how previous sleep affects subsequent napping, and vice versa. While these approaches are paramount to understanding the interplay between sleep and health, napping is treated as a behaviour distinct from night-time sleep. However, quantity and quality of night-time sleep may influence reasons for napping and thereby affect its links to health outcomes differentially. For example, naps taken to compensate for poor night-time sleep may differ in their health consequences from naps taken for enjoyment. This raises the question of to what extent napping behaviour must be considered in conjunction with night-time sleep behaviour in order to understand sleep–health relationships fully.

To address this question, the current study aimed to assess relationships between health and naturally occurring sleep behaviour patterns by conceptualizing (i) napping as sleep behaviour occurring in conjunction with night-time sleep and (ii) health as chronic health conditions, operationalized as chronic psychological health outcomes (e.g. depressive symptoms), physiological health mediators (e.g. inflammatory marker IL-6) and physiological health outcomes (e.g. total number of symptoms and conditions reported). To relate current findings to the recent literature, health effects of napping were determined by comparing napping and non-napping individuals. To address the main aim of the study, interindividual differences in patterns of daytime and night-time sleep combinations within napping individuals were related to the same above-mentioned health outcomes.

The data utilized came from the Midlife in the United States II (MIDUS II) Biomarker study. Participants provided 7 days' worth of sleep and nap information, psychological health questionnaires and blood samples for biomarker analysis. This data set allows capturing an individual's sleep habits by assessing actual sleep behaviour during the course of a week by actigraphy (night-time sleep) and diary (napping) as well as assessing a wide range of physiological and psychological health issues.

MATERIAL AND METHODS

Participants

Participants in this study were a subset of 368 healthy adults who took part in the MIDUS II Biomarker study as well as the follow-up actigraphy sleep study at the University of Wisconsin, Madison (Ryff *et al.*, 2013). Participants were excluded from analysis for having incomplete data ($n = 23$), abnormally high waist–hip ratios ($n = 2$; > 4 standard deviations (SD) above the mean), abnormally high overnight urinary noradrenaline secretion [$n = 1$; 8 standard deviations (SD) above the mean], disordered sleep behaviour ($n = 1$; total nap time 7 SD above the mean) and acute infection, i.e. IL-6 > 10.0 pg/ml (n = 7) or CRP > 10.0 μg mL^{-1} ($n = 22$) (e.g. Clyne and Olshaker, 1999). In total, 313 participants met the above requirements and were included in analyses. Participants were divided further into two groups—those who reported napping at least once ($n = 208$) and those who did not report napping ($n = 105$) during the study period. The study protocol was approved by the Institutional Review Board of the University of Wisconsin–Madison, and informed written consent was obtained for all participant.

Protocol

Participants were recruited from the MIDUS II Biomarker Project study population. Upon arrival at the University of Wisconsin, participants underwent a physical examination and a medical history interview with a clinician and completed a set of self-report questionnaires assessing demographic, psychological and medical variables before an overnight stay. During this stay, 12-h overnight urinary samples were collected. Fasting blood samples were obtained the next morning between 06:30 and 07:00 hours. Participants then received detailed instructions on the at-home data collection portion, including self-reported sleep assessments and an Actiwatch to be worn around the wrist for seven consecutive days and nights (see Dienberg Love *et al.* (2010) for more details).

Measures

Actigraphy

Sleep behaviour data were collected using actigraphs (Actiwatch®-64; Mini Mitter, Bend, OR, USA, now Philips Respironics). Respondents began recording at 07:00 hours on the first Tuesday morning after their overnight stay and continued for seven consecutive nights, indicating when they started trying to fall asleep (bedtime) and when they arose in the morning (rise time) by pressing the event marker button on the Actiwatch. Upon return of the Actiwatches to MIDUS project staff, event marker and sleep diary information assisted automatic detection of sleep intervals by manufacturer algorithms (Actiware 5, Philips Respironics) (Ancoli-Israel *et al.*, 2003). Anomalous or incomplete actigraphy intervals were reviewed further by study staff and hand-scored or deleted as appropriate. Average sleep duration, efficiency and latency were determined by averaging respective values from each of the seven study nights for each participant.

Nap reporting

Napping was assessed by the MIDUS Biomarker Project Daily Sleep Diary, a daily sleep diary, which was completed at bedtime on each of the seven study days. Participants reported whether napping occurred on any given day and, if so, listed the nap(s) duration. No participants reported multiple naps at any given day; thus, nap frequency is equivalent to number of days a participant reported a nap. Naps of a self-reported duration of 1 min or more were considered for analyses. Total nap time was determined by summing the duration of each nap occurring during the study week to provide a cumulative measure of weekly napping.

Biomarkers/health mediators

Noradrenaline (NE) and cortisol levels were assessed in one overnight 12-h urine sample collected during the in-laboratory overnight stay. NE concentrations were determined by high-pressure liquid chromatography, while urinary free cortisol and d(3)-cortisol levels were measured by enzymatic colorimetric assay and liquid chromatography-tandem mass spectrometry using multiple reaction monitoring in positive mode (Taylor *et al.*, 2002). All cortisol values reported as <0.019 μg day^{-1} were adjusted to 0.019 μg dL^{-1}. A cholesterol panel and the inflammatory markers CRP and IL-6 were assessed in fasting blood samples taken between 06:30 and 07:00 hours following the in-laboratory overnight stay. Serum and plasma was isolated from the respective samples, aliquoted, frozen at -80°C, shipped on dry ice to the MIDUS Biocore Laboratory and stored at -80°C until assayed. IL-6 was measured using the Quantikine® high-sensitivity enzyme-linked immunosorbent assay (ELISA) kit (R&D Systems, Minneapolis, MN, USA) and CRP was measured using the BNII nephelometer from Dade Behring (Deerfield, IL, USA) utilizing a particle-enhanced immunonepholometric assay. CRP values <0.15 μg dL^{-1} were adjusted to 0.14 μg dL^{-1}. To determine the ratio between high-density lipoprotein cholesterol (HDL) and low-density lipoprotein cholesterol (LDL), first HDL cholesterol was determined using a lipid assay (Roche Diagnostics, Indianapolis, IN, USA). HDL values >120 mg dL^{-1} were adjusted to 121 mg dL^{-1}. Next, an estimation of LDL-cholesterol level was made using the Fridewald formula from direct measurements of total cholesterol, triglycerides and HDL cholesterol (Ryff *et al.*, 2013). Lastly, a HDL/LDL ratio was computed by dividing HDL values with LDL values.

Waist–hip ratio was measured by study clinicians using a Gulik II tape measure during the participants' visits to UW-Madison. Abdominal obesity is commonly defined by a waist–hip ratio above 0.90 for males and above 0.85 for females (World Health Organization, 2011).

Self-report measures

The Center for Epidemiologic Studies Depression (CESD) scale was utilized to measure depressive symptoms (Radloff, 1977). The CESD is a well-validated and reliable ($\alpha = 0.89$) instrument composed of 20 items assessing the frequency of depression-related symptoms during the last month. For the purposes of the current study, CESD items related directly to sleep were excluded from the total score in order to avoid CESD sleep items driving associations between sleep and depressive symptoms.

The Perceived Stress Scale (PSS) is a widely used reliable ($\alpha = 0.86$) 10-item self-report questionnaire measuring chronic stress. The questions are general in nature and avoid content specific to subpopulations (Cohen *et al.*, 1983). Responses assess frequency of feelings of stress during the last month, with a higher PSS score indicating higher perceived chronic stress.

The 'Symptoms and Conditions' category of the MIDUS Biomarker Project Medical History questionnaire was utilized to assess participants' health status. The total number of symptoms and conditions per participant was calculated by summing the presence of physician-confirmed self-report of symptoms and conditions.

Statistical analyses

A three-step approach was used to address the study aims. All analyses controlled for age, gender and race. First, nappers were compared to non-nappers with regard to demographics and sleep parameters using chi-squared tests or analysis of covariance (ANCOVA). Hierarchical regression analyses were computed to test associations between napping behaviour and health outcomes as well as to explore the potential mediating effects of average night-time sleep duration on these associations. To capture the complexity of sleep behaviour, while at the same time avoiding computing multiple sets of regressions, a two-step cluster analysis was chosen to address the second aim of the study (King, 1967). The cluster analysis combined self-report nap variables (nap frequency, total nap time) as well as actigraphy-determined night-time sleep variables (average sleep duration, average sleep efficiency, sleep latency). Schwarz's Bayesian information criterion (BIC) was used for establishing the optimal number of clusters and analysis of variance (ANOVA) for the classification relevance of variables. Clusters were tested for associations with health-relevant outcome variables using ANCOVAs. All analyses were computed in SPSS version 22.0. Statistical significance was assumed at $P \leq 0.05$.

RESULTS

Nappers versus non-nappers

Nappers and non-nappers were compared on all study variables. Results are summarized in Table 1 and show no differences in terms of gender, race, marital status or age (all $P > 0.23$). While nappers were more likely to be unemployed compared to non-nappers ($P = 0.019$), no differences were observed with regard to level of education ($\chi^2 = 7.86$,

Table 1 Differences between nappers (n = 208) and non-nappers (n = 105) in demographic variables and actigraphy-assessed sleep during 7 days

	Nappers	*Non-nappers*	F/χ^2	*P*
Demographics				
Gender %				
Men/women	45/55	40/60	0.63	0.43
Race %				
Caucasian/African American/Other	73/24/3	80/19/1	2.88	0.24
Employment status %				
Employed/unemployed/not answered	64/35/1	77/22/1	5.61*	0.019
Marital status %				
Married or cohabitating/no longer married/never married	67/24/9	65/21/14	2.04	0.36
Average age in years [standard deviation (SD)]	54.5 (11.7)	52.4 (11.1)	2.36	0.12
Age range (min/max)	34/82	34/81	–	–
Sleep averaged during 7 days (mean and SD)				
Sleep duration (min)	367.5 (62.3)	391.6 (59.7)	8.73*	0.003
Sleep latency (min)	29.3 (27.8)	23.3 (24.6)	2.51	0.11
Sleep efficiency (%)	80.12 (9.9)	83.06 (9.1)	4.58*	0.03
Nap length (min)	19.81 (19.8)	–	–	–
24-h sleep duration (min)	387.3 (61.7)	391.6 (59.7)	0.10	0.75

*signifies significant differences between groups (p<.05).

Table 2 Differences between nappers (n = 208) and non-nappers (n = 105) in health mediators, psychological health outcomes and physiological health outcomes

	Nappers Mean (SD)	*Non-nappers Mean (SD)*	β	*SE*	*B*	*P*
Health mediators						
Urine noradrenaline	2.13 (1.39)	2.22 (1.75)	0.02	0.19	0.08	0.68
Urine cortisol (μg dL^{-1})	1.25 (1.52)	1.34 (1.27)	00.01	0.17	0.04	0.82
Serum IL-6 (μg mL^{-1})	2.87 (1.75)	2.27 (1.63)	0.13*	0.20	0.47	0.019
Blood C-reactive protein (μg mL^{-1})	2.59 (2.35)	1.72 (1.56)	0.19*	0.25	0.88	0.001
Blood cholesterol HDL/LDL ratio	0.62 (0.43)	0.56 (0.29)	0.06	0.05	0.05	0.24
Waist–hip ratio	0.91 (0.09)	0.88 (0.10)	0.12*	0.01	0.02	0.005
Psychological health outcomes						
Depressive symptoms (CESD)	5.02 (6.49)	4.20 (6.26)	0.06	0.76	0.77	0.32
Perceived stress (PSS)	22.14 (5.88)	21.93 (6.42)	0.03	0.72	0.35	0.63
Physiological health outcomes						
Total no. symptoms and conditions	3.58 (2.59)	2.73 (1.98)	0.12*	0.26	0.64	0.016

CESD, Center for Epidemiologic Studies Depression scale; PSS, Perceived Stress Scale; LDL, low-density lipoprotein; HDL, high-density lipoprotein; SD, standard deviation; SE, standard error * P < .05.

P = 0.73) or income as assessed in MIDUS I (t = 1.06; P = 0.29). Sleep latency and duration within a 24-h period were similar in both groups (all $P > 0.10$); however, nappers had significantly less sleep per night (approximately 20 min; P = 0.003) and had significantly less efficient sleep (average sleep efficiency: P = 0.03).

Descriptives of health-related outcome variables are summarized in Table 2. IL-6 levels, CRP levels, total number of symptoms and conditions, as well as waist–hip ratios, were significantly higher in nappers compared to non-nappers (see Fig. 1). Similar findings were observed when using BMI as an outcome variable instead of waist–hip ratio (P = 0.001). No group differences were found for urinary endocrine markers, HDL/LDL ratio, depressive symptoms or perceived chronic stress levels (all $P > 0.14$).

To address the possibility that differences between nappers and non-nappers in waist–hip ratio, IL-6 and CRP are due to current health problems, analyses were repeated controlling for number of symptoms and conditions; however, none of the effects changed. Conversely, controlling for obesity (BMI) eliminated the significant difference between nappers and non-nappers for IL-6 (F = 1.33, P = 0.25), but did not affect between-group effects for other variables. Additionally, controlling for employment status did not affect between-group effects for any variables. Lastly, none of the above findings changed when controlling for effects of nighttime sleep duration, i.e. significant differences in health outcomes between nappers and non-nappers remained significant and non-significant differences remained non-significant.

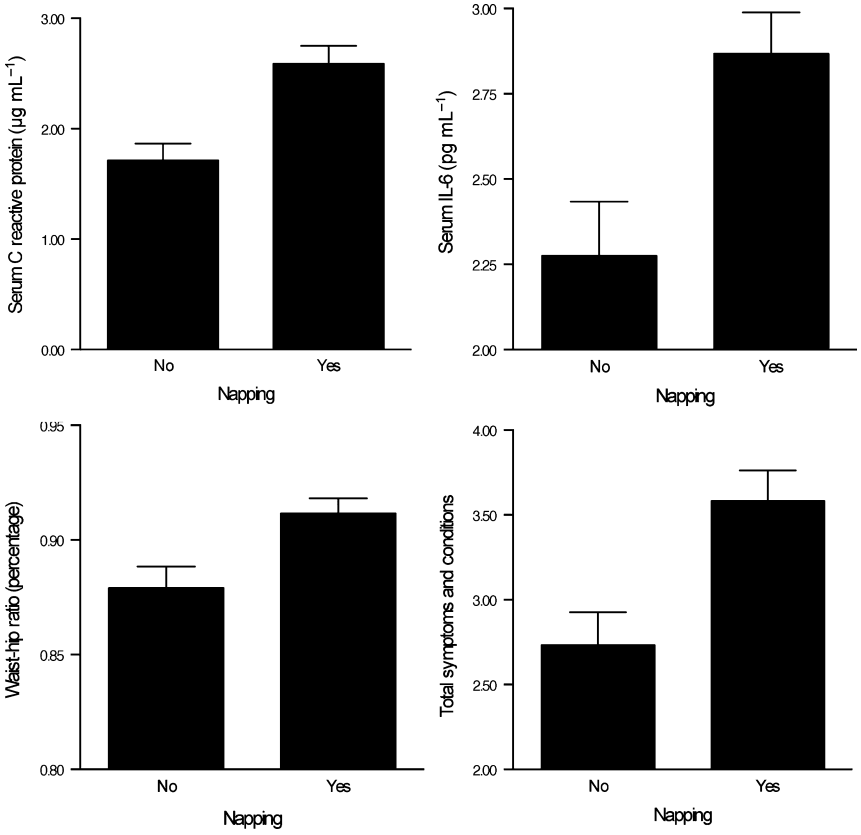

Figure 1. Participants napping at least once a week showed significantly elevated C-reactive protein levels and interleukin-6 levels, higher waist–hip ratios and a greater number of total medical symptoms and conditions compared to non-nappers.

Sleep patterns

To test the possibility that, within nappers only, specific patterns of sleep behaviour would be linked differentially to health measures, self-reported nap behaviour and sleep behaviour variables determined by actigraphy were next subjected to a two-step cluster analysis resulting in a three-cluster solution (BIC: 569.76; BIC ratio changes: 0.65; average silhouette: 0.5). ANOVA confirmed centroid differences for actigraphy sleep and nap variables and between-cluster differences in sleep and demographic variables are summarized in Table 3.

Table 3 Three-cluster solution for actigraphy-assessed night-time sleep and diary-assessed daytime nap behaviour

	Cluster 1 (n = *113)*	*Cluster 2* (n = *68)*	*Cluster 3* (n = *27)*	F/χ^2
Demographics				
Gender %				
Men/Women	27/73	62/38	74/26	30.14*[*]
Race %				
Caucasian/African American/Other	76/22/2	71/22/7	63/37/0	7.73[NS]
Employment status %				
Employed/unemployed/not answered	69/31/0	59/40/1	59/41/0	2.01[NS]
Marital status %				
Married or cohabitating/no longer married/never married	74/21/5	62/25/13	52/33/15	7.29[NS]
Average age (SD)	53.69 (11.07)	55.00 (12.69)	56.67 (11.73)	0.79[NS]
Age range (min/max)	34/80	35/82	38/81	–
Sleep during 7 days				
Nap frequency	1.57 (0.72)	4.65 (1.41)	2.56 (1.37)	171.53*
Total nap time (min)	59.11 (45.12)	277.48 (152.50)	121.22 (76.88)	107.46*
Average sleep duration (min)	389.57 (50.19)	363.96 (53.85)	284.36 (57.17)	44.28*
Average sleep latency (min)	20.35 (13.29)	23.93 (16.29)	79.95 (40.14)	103.58*
Average sleep efficiency (%)	83.93 (5.97)	81.28 (6.23)	61.30 (9.24)	131.35*

NS, not significant; SD, standard deviation.
*$P < 0.001$.

Participants in the three clusters did not differ in terms of age, marital status, education level, employment status or race (all $P > 0.30$). However, cluster 1 was dominated by women, while the two genders were distributed evenly in clusters 2 and 3. Individuals in cluster 1 reported sleeping approximately 6.5 h on average per night and napping less frequently (less than twice a week) than participants in other clusters. Participants in cluster 2, in contrast, napped four to five times per week and slept for approximately 6 h per night. Cluster 3 participants slept less, on average (approximately 5 h per night), compared to clusters 1 and 2 and napped between two and three times per week. Additionally, participants in cluster 3 had worse sleep efficiency and average sleep latency four times greater than that of participants in clusters 1 or 2.

Differences in health indicators between sleep clusters

No significant differences between clusters were observed for most of the biomarkers and total number of symptoms and conditions (all $P > 0.10$). However, urinary NE levels differed between clusters, with nappers in cluster 3 showing higher NE levels compared to nappers in clusters 1 or 2 ($F = 4.70$; $P = 0.01$) (see Fig. 2). Furthermore, individuals in the three clusters differed significantly in terms of depressive symptoms ($F = 4.03$; $P = 0.019$) and perceived chronic stress ($F = 3.23$; $P = 0.041$), such that participants in cluster 3 reported the highest levels of depressive symptoms and perceived chronic stress while participants in cluster 1 had the lowest CESD and PSS scores (see Fig. 2). This effect pattern did not change when controlling for number of symptoms and conditions or when controlling for obesity, with the exception of the latter reducing the significant PSS finding to a trend ($F = 2.87$, $P = 0.06$).

DISCUSSION

The current study investigated the associations between sleep habits and health outcomes in American adults, taking into account night-time sleep assessed via actigraphy as well as self-reported nap behaviour. Interestingly, of 313 participants, 208 reported napping between one and seven times during the study week. Overall, individuals who reported napping exhibited elevated levels of the inflammatory biomarkers IL-6 and CRP, greater waist–hip ratios and a larger number of total symptoms and conditions compared to non-nappers. Within nappers, three distinct patterns of sleep behaviour emerged that were characterized further by differential associations with NE, chronic stress levels and depressive symptoms.

Sleep, demographic and health differences in nappers and non-nappers

Approximately 66% of participants in the current study reported napping during the course of a week. As such, our findings are in line with earlier studies reporting prevalence rates of napping in healthy adult populations in the range of 45–80% (Milner and Cote, 2009; Pilcher *et al.*, 2001), with higher rates being observed in adults above 60 years than adults under 35 years of age (Buysse *et al.*, 1992; Ohayon *et al.*, 2001; Yoon *et al.*, 2003). Similarly, they replicate a lack of differences between nappers and non-nappers in terms of gender (Owens *et al.*, 2010; Yoon *et al.*, 2003), race (Owens *et al.*, 2010) and average hours of sleep per 24-h period (Jean-Louis *et al.*, 2000; Lauderdale *et al.*, 2008). As such, nap behaviour shown in the current study appears to be representative of sleep habits observed in other studies and the general population.

With regard to health-related outcome measures, nappers exhibited significantly higher IL-6 and CRP levels as well as higher waist–hip ratios and more medical symptoms and conditions. These findings are in line with observations from earlier studies linking napping to obesity as well as inflammation-related diseases (Foley *et al.*, 2007; Hays *et al.*, 1996; Owens *et al.*, 2010; Xu *et al.*, 2010). Importantly, inflammation has been shown consistently to lead to

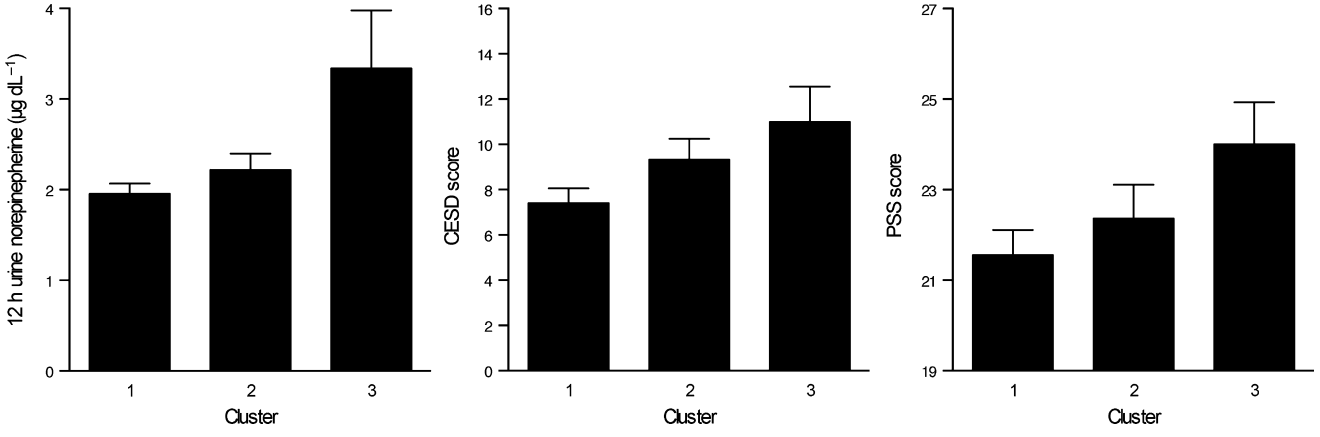

Figure 2. Differences in health indicators between sleep clusters showing insomnolent sleepers (cluster 3) exhibiting the highest levels of urinary noradrenaline, depressive symptomatology [assessed by the Center for Epidemiologic Studies Depression (CESD) scale] and perceived chronic stress [assessed by the Perceived Stress Scale (PSS)]. Predominantly 'night-time sleepers' describes participants in cluster 1, 'siesta sleepers' describes participants in cluster 2 and 'insomnolent sleepers' describes participants in cluster 3.

pathological sleepiness (Cho *et al*., 2013), while obesity has been related to daytime fatigue (Vgontzas *et al*., 1998). Thus, feelings of fatigue may drive the urge to nap. Additionally, inflammatory processes are linked closely to obesity (Park *et al*., 2005) and both inflammation as well as adiposity are, in turn, associated with overall morbidity and mortality, particularly from cardiovascular diseases, diabetes and other chronic diseases (Wang *et al*., 2011). However, controlling for number of medical conditions did not abolish the link between napping and markers of inflammation or obesity, suggesting a more complex role of napping.

Differential health effects of sleep patterns

Given nappers' elevated health risks as well as earlier findings suggesting non-linear associations between napping and health outcomes, we next investigated the potential of sleep pattern-dependent health effects within nappers. Cluster analysis thereby revealed three natural groupings (clusters) of daytime and night-time sleep behaviour. One group of individuals (cluster 1) slept predominantly at night, and only napped infrequently during the study period. Participants in this cluster exhibited sleep parameters comparable to non-nappers, such as high sleep efficiency and duration, and good self-reported sleep satisfaction. Participants in cluster 2 napped more days than not during the study week. This nap behaviour resembles the siesta, which is defined commonly as a traditional mid-day short nap practised by many cultures worldwide. However, considering that time of day when naps were taken was not recorded in the present MIDUS II subproject, it is important to note that 'siesta nappers' may not be taking mid-afternoon naps, or be napping at a consistent time of day across the cluster. In addition, when compared to individuals in cluster 1, these siesta sleepers did not exhibit any detriments in sleep latency or efficiency. A third group of participants (cluster 3) exhibited the worst night-time sleep of any of the groups and napped less than siesta sleepers, but more than predominantly night sleepers. It could be speculated that individuals in this third group are insomnolent sleepers, in that they do not gain enough sleep regardless of time of day.

While nappers exhibited elevated inflammation, obesity and more medical conditions compared to non-nappers, these health indicators were not associated differentially with sleep behaviour patterns in the three groups of nappers. This suggests that, contrary to earlier reports (Dinges, 1992), the occurrence of napping *per se* may be indicative of increased health problems related to obesity and inflammation, independent of specific manifestations of sleep habits.

Contrarily, sleep behaviour differences within nappers were related to NE levels and mental health outcome variables. Insomnolent sleepers not only showed the highest NE levels, but reported the highest number of depressive symptoms and perceived the highest level of stress compared to predominantly night or siesta sleepers. The current study is not the first to observe the co-occurrence of elevated NE and mental health effects in the context of sleep. A study by Mezick *et al.* (2009) showed that NE was associated with higher sleep fragmentation and moderated by negative affect (Mezick *et al*., 2009). Furthermore, napping has been associated repeatedly with depression (Foley *et al*., 2007). The current findings suggest that frequency of napping *per se* may not be responsible for this effect, as siesta sleepers showed similar napping frequency without elevated depressive symptoms or perceived stress levels. Instead, the poor night-time sleep shown by insomnolent sleepers may be detrimental in terms of elevated stress and depressive symptoms independently of napping behaviour. Put differently, although napping has been linked previously to increased risk for mental health problems, daytime sleep behaviour may not be related to an increased risk for depression provided that night-time sleep quality is high. These findings lend support for an emphasis on considering sleep as a complex behaviour consisting of interdependent daytime and night-time occurrences of varying lengths and quality.

Limitations

The current findings should be assessed in light of several limitations. Primarily, participants were not distributed equally into clusters. While the overall number in the present study was sufficiently large to investigate sleep and health relationships, the smaller number for insomnolent sleepers precluded the assessment of gender, age or race as moderators. Secondly, the current study used cluster analysis to detect sleep behaviour patterns. While cluster analysis allows for the grouping of variables, it does not provide information on why certain variables may be clustered, and future studies will have to characterize the determinants of distinct sleep behaviour patterns. Thirdly, considering time of day when napping occurred may be a promising additional factor when assessing health effects of sleep behaviour, given that naps taken in the evening may be more detrimental for night-time sleep quality (Milner and Cote, 2009). Moreover, self-reported nap behaviour was not compared with actigraphy data to confirm nap frequency or duration; nor did participants record whether naps were premeditated or spontaneous, information that may be helpful in determining individual differences within nappers. Fourthly, habitual napping is a potential marker for sleep apnea (Masa *et al*., 2006). Future studies would benefit from including diagnostic assessment of sleep apnea. Fifthly, self-reported sleep assessed during the month prior to the study (PSQI: Pittsburgh Sleep Quality Index) correlated significantly with the equivalent actigraphy-based sleep measures (data not reported). However, multiple weeks of actigraphy data collection would be needed to evaluate how well 1 week of sleep assessment reflects an individual's sleep habits. Lastly, statistical and conceptual concerns precluded the assessment of links between sleep behaviour patterns and specific disease patterns or syndromes, representing another promising avenue for future studies.

CONCLUSION AND OUTLOOK

In summary, napping was a prevalent behaviour in the assessed study population, with the majority of nappers exhibiting shorter night-time sleep duration compared to non-nappers. Independently of medical conditions or health effects of shorter night-time sleep duration, however, individuals who napped were more likely to show elevated inflammatory processes and adiposity, suggesting that napping may indicate greater distinct health risks over and above being a consequence of ill health. When focusing on nappers only, those showing a combination of less frequent napping and poor night-time sleep exhibited higher chronic stress, depressive symptoms and elevated NE levels. While napping can be a consequence of stress and depression negatively affecting night-time sleep, the current findings suggest that individuals who nap may not be at a greater risk for depression as long as their night-time sleep is sufficient in quantity and quality. Therefore, the present study is a first step towards determining which sleep behaviour changes may be promising for decreasing negative health effects. In order to help tease apart which sleep behaviours are benign and which should be avoided, the next important step will be to identify the determinants and underlying mechanisms differentiating functional nap and night-time sleep behaviour patterns and sleep patterns that are related to health detriments.

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AUTHOR CONTRIBUTIONS

JKD and JMW jointly developed the research questions, managed the literature searches, undertook the statistical analyses and contributed to and approved the final manuscript.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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