Sleep and Vigilance (2022) 6:359–365
https://doi.org/10.1007/s41782-022-00218-z

ORIGINAL ARTICLE

Profiling of Sleep Models Based on Voluntary and Involuntary Sleep in Adults with Type 2 Diabetes

Azusa Oda[1] · Miho Katayama[1] · Ritsuko Aijo[1,2] · Chinatsu Kato[1]

Received: 27 December 2021 / Revised: 22 August 2022 / Accepted: 29 August 2022 / Published online: 12 September 2022

Abstract

Purpose The purpose of this study is to generate the concept of voluntary sleep (V) and involuntary sleep (IV) in sleep, to build sleep models using them, and to profile by diabetes control in adults with type 2 diabetes.

Methods We obtained 595 nights of sleep data from 50 participants. Participants measured sleep with the sleep meter HSL-101 (Omron Healthcare, Kyoto) and answered the Pittsburgh Sleep Quality Index (PSQI). They were operationally defined as V and IV.

Results "V1: sleep self-determination" and "V2: conscious sleep quantity" were generated from voluntary sleep, and "IV1: continuous deep sleep" and "IV2: actual sleep quantity" were generated from involuntary sleep. Using cluster analysis, they were classified into three models, "CL1: sleep satisfaction model", "CL2: short sleep model", and "CL3: dissatisfaction sleep model". When the diabetes controls in each cluster were compared by ANOVA and Bonferroni's test, HbA1c was higher in the order of CL1, CL2, and CL3, and there was a significant difference between CL1 and CL3 ($p=0.029$). Similarly, age was low in the same order and BMI was high in the same order, with a significant difference between CL1 and CL3 ($p=0.030, 0.037$).

Conclusions Sleep in adults with type 2 diabetes could be clustered into three models based on V and IV, each of which could be profiled by a significantly characteristic diabetes control index. It will be possible to identify the patient's sleep model from the diabetes control and appropriately approach voluntary sleep and involuntary sleep.

Keywords Sleep · Diabetes · Pittsburgh sleep quality index · Cluster analysis

1 Introduction

Sleep disorders are a serious problem in today's 24-h society. Inadequate sleep can have a major impact on our society and is regarded as a new public health problem owing to its widespread effects [1, 2]. Too little or too much sleep is associated with significant causes of death, such as cardiovascular disease [3, 4], cerebrovascular disease [5, 6], diabetes [7, 8], and hypertension [3, 9, 10]. In addition, frequent episodes of inadequate sleep are associated with decreased mental and physical well-being [11]. These are captured by the concept of sleep quantity. In addition to quantity, quality is another way that sleep is perceived. The quality of sleep decreases with age [12, 13], and times of inadequate sleep are associated with worse metabolism and decreased activity [14, 15]. Indeed, sleep quantity and quality are composite elements that characterize sleep, and both need to be considered when predicting health outcomes [16].

There is also the concept of regularity in sleep. The circadian clock has been established to be a pervasive biological mechanism, and an irregular sleep schedule is the most common cause of circadian clock disruption [17]. Inconsistent and irregular sleep duration and timing increase the rates of obesity, hypertension, and dyslipidemia [18–21]. Although studies have suggested that poor sleep can impair health in a variety of aspects, most of these researches were based on individual perceptions; these studies treated sleep subjectively, but there are other ways of perceiving sleep objectively [22]. Subjective sleep is primarily evaluated through self-reports, whereas objective sleep is examined by measurements, such as polysomnography and actigraphs [23].

✉ Azusa Oda
azusa.oda@komatsu-u.ac.jp

[1] Faculty of Health Sciences, Department of Nursing, Komatsu University, He 14-1 Mukaimotoorimachi, Komatsu, Ishikawa 923-0961, Japan

[2] Division of Health Sciences, Graduate School of Medical Sciences, Kanazawa University, Kanazawa, Japan

In this study, we used a sleep meter and a self-administered questionnaire to measure sleep in adults with type 2 diabetes. The purpose of this study was to define sleep that can be controlled by oneself as voluntary sleep (V) and sleep that is difficult to control by oneself as involuntary sleep (IV), to construct sleep models using these concepts, and to understand the relationships of these types of sleep with diabetic control in adults with type 2 diabetes.

2 Methods

2.1 Participants

We asked 91 adults with type 2 diabetes and who went to three hospitals in a prefecture to participate in measurement of sleep using a sleep meter and to answer the Pittsburgh sleep quality index (PSQI), which is a self-evaluation of sleep quality. A total of 50 subjects (38 men, 12 women) agreed to participate in the study, and a total of 595 days of sleep were analyzed. No participant had fewer than three days of measurement.

2.2 Ethical Considerations

This study was approved by the local ethics review committee (approval number: 742-1). The researcher explained to the subjects verbally and in writing that participation in the research was voluntary that there would be no disadvantage in refusing to participate, and that personal information was strictly protected. Written informed consent was obtained from all participants.

2.3 Objective Sleep Evaluation

Participants were asked to use a sleep sensor (HSL-101; Omron, Kyoto, Japan) to self-monitor their sleep for 14 nights. The sleep meter was placed next to the bed, and measurements were performed without restraining, attaching to, or contact with the participant. The participants were asked to press a button on the sleep meter when they went to bed and upon waking.

The following sleep parameters were automatically calculated using the sleep meter: bedtime (BT), total sleep time (TST), deep sleep time (DST), sleep onset latency (SOL), and wake after sleep onset (WASO). In addition, sleep efficiency (SE) was calculated as (TST/BT) × 100. Bedtimes and wake-up times were obtained from the sleep graph to calculate the bedtime standard deviation (BSD) and wake-up time standard deviation (WSD). The absolute value of the difference between self-reported sleep time and TST was designated as absTST. The absolute value of the difference between self-reported sleep efficiency and SE was designated as absSE.

2.4 Subjective Sleep Evaluation

The Japanese version of the PSQI was used to assess subjective sleep [24–26]. The PSQI comprises seven components, as follows: C1, sleep quality; C2, sleep latency; C3, sleep duration; C4, habitual SE; C5, sleep disturbance; C6, use of medication for sleep; and C7, daytime dysfunction. The scores for these components were added together to obtain the global score. The cutoff was 6 points, and scores ≥ 6 represented poor sleep quality. Subjective bedtime (sBT), subjective TST (sTST), and subjective SE (sSE) were extracted from the C3 and C4 scores.

2.5 Steps in Establishing Sleep Models

Adults with type 2 diabetes were classified according to their sleep types, and a three-step analysis was performed to characterize their diabetic control. As the first step, sleep elements were operationally divided into V and IV elements V was a subjective sleep item that can be consciously controlled, and its components were BSD, WSD, absTST, absSE, sBT, sTST, and sSE. IV was an objective sleep item that was difficult to control consciously, and its components were BT, TST, DST, SOL, WASO, and SE. Bartlett's sphericity test validated the principal component analysis, and a principal component analysis was performed for each. As the second step, a k-means cluster analysis was performed using the integrated principal component score and the PSQI score as the variables. The k-means method is a clustering algorithm that specifies the number of clusters and collects and classifies data with similar attributes. In this study, we increased the number of clusters in order from three and continued the analysis until the composition of one or more clusters became homogenous and interpretable. Thereafter, we obtained the sleep models. Finally, we examined whether there was a difference in diabetes control among the clustered sleep models and performed profiling. Analysis of variance and Bonferroni's test were performed to compare sleep models.

3 Results

3.1 Participants and Sleep Measurements

Table 1 shows the participants' basic attributes and the values for each measurement. The mean total PSQI score was 8.06 ± 4.48, which was significantly higher than the cutoff point ($p = 0.002$).

Table 1 Characteristics and measurements of participants

	n	Mean (SD)	Range
Sex (Male/female)	38/12		
Age (years)		60.50 (10.11)	35–73
HbA1c (%)		7.90 (1.58)	5.4–12.3
BMI (kg/m^2)		26.37 (5.18)	16.85–37.72
Diabetes duration (years)		9.36 (5.04)	1–23
PSQI		8.06 (4.48)	2–22
Voluntary sleep			
WSD: wake-up time standard deviation (h:mm)		0:50 (0:37)	0:15–3:06
BSD: bedtime standard deviation (h:mm)		0:56 (0:38)	0:12–3:06
sTST: subjective total sleep time (h:mm)		6:30 (1:24)	4:0–10:00
sSE: subjective sleep efficiency (%)		86.48 (13.70)	46.67–100.00
absTST: absolute value of difference between TST and sTST (h:mm)		1:11 (1:04)	0:03–6:20
absSE: absolute value of difference between SE and sSE (%)		11.29 (8.70)	0.77–41.77
Involuntary sleep			
SE: sleep efficiency (%)		87.15 (8.54)	56.09–98.03
WASO: wake after sleep onset (h:mm)		0:30 (0:30)	0:01–3:07
DST: deep sleep time (h:mm)		2:01 (0:41)	0:49–3:20
TST: total sleep time (h:mm)		5:53 (1:25)	2:40–9:03
SOL: sleep onset latency (h:mm)		0:33 (0:18)	0:09–1:27

BMI body mass index, *PSQI* Pittsburgh Sleep Quality Index, *SD* standard deviation

Table 2 Principal component analysis of voluntary sleep

		V1	V2
KMO	0.708		
Bartlett's test	< 0.001		
Principal component loading			
WSD		0.879	0.060
BSD		0.778	0.146
absTST		0.752	0.421
absSE		0.738	−0.342
sTST		0.146	0.828
sSE		−0.477	0.737

KMO Kaiser–Meyer–Olkin sampling adequacy, *V1* sleep self-determination, *V2* conscious sleep quantity, *WSD* wake-up time standard deviation, *BSD* bedtime standard deviation, *absTST* absolute value of difference between total sleep time and subjective total sleep time, *absSE* absolute value of difference between sleep efficiency and subjective sleep efficiency, *sTST* subjective total sleep time, *sSE* subjective sleep efficiency

3.2 Voluntary and Involuntary Sleep Models

On principal component analysis, sBT was excluded from V and BT was excluded from IV. V included six variables (i.e., BSD, WSD, sTST, sSE, absTST, and absSE) and IV included five variables (TST, DST, SOL, WASO, and SE).

The results for V are shown in Table 2. Kaiser–Meyer–Olkin (KMO) sampling adequacy was 0.708 and Bartlett's test of sphericity was significant ($p < 0.001$), demonstrating the validity of the principal component analysis. The principal components were calculated until the eigenvalue was ≥ 1. The cumulative contribution ratio was 71.412%. The main variables of the first principal component were WSD, BSD, absTST, and absSE, with principal component loadings of 0.879, 0.778, 0.752, and 0.738, respectively. These components indicated the timing of going to bed, waking, and recognizing sleep. Therefore, the first principal component was named V1 (i.e., sleep self-determination), which accounted for 45.605% of the total variance. The main variables of the second principal component were sTST and sSE, with principal component loadings of 0.828 and 0.737, respectively. These indicated the length and efficiency of sleep. Therefore, this second principal component was named V2 (i.e., conscious sleep quantity).

The results for IV are shown in Table 3. The KMO sampling adequacy was 0.493 and Bartlett's test of sphericity was significant ($p < 0.000$). The principal components were calculated until the eigenvalue was ≥ 1. The cumulative contribution ratio was 78.782%. The main variables of the first principal component were SE, WASO, and DST, with principal component loadings of 0.946, 0.825, and 0.748, respectively. These components were difficult to consciously control and included SE, depth, and persistence. Therefore, this first principal component was named IV1 (i.e., continuous deep sleep), which accounted for 53.333% of the total variance. The main variables of the second principal component

Table 3 Principal component analysis of involuntary sleep

		IV1	IV2
KMO	0.493		
Bartlett's test	< 0.001		
Principal component loading			
SE		0.946	−0.131
WASO		−0.825	0.310
DST		0.748	0.434
TST		0.499	0.769
SOL		−0.533	0.616

KMO Kaiser–Meyer–Olkin sampling adequacy, *IV1* continuous deep sleep; IV2, actual sleep quantity, *SE* sleep efficiency, *WASO* wake after sleep onset, *DST* deep sleep time, *TST* total sleep time, *SOL* sleep onset latency

were TST and SOL, with principal component loadings of 0.769 and 0.616, respectively. These were considered to be components that appeared unintentionally. Therefore, this second principal component was named IV2 (i.e., actual sleep quantity).

3.3 Clustering by Sleep Indicators

A *k*-means cluster analysis was performed based on PSQI, V1, V2, IV1, and IV2, which were indicators of sleep. The number of clusters was increased from three, and analysis continued until the composition of one or more clusters became interpretable. As a result, the best fit was classified into three clusters. These clusters were significantly different in terms of PSQI, V1, and V2 (Table 4).

The configuration of each cluster is shown in a radar chart (Fig. 1). To note the condition featuring the best sleep, sleep quality was set to the maximum PSQI score of 21 at the edge of the plot, and V1 values were multiplied by − 1.

Cluster 1 ($n = 17$) had good sleep quality (PSQI 4.24 ± 1.35) and tended to be good in all components of V and IV. Therefore, it was named CL1 (i.e., satisfactory sleep model). Cluster 2 ($n = 23$) had poor sleep quality (PSQI 8.48 ± 1.56) and was as good as cluster 1 in terms of V1 and IV1 but had low levels of sleep quantity, which were common with V2 and IV2. Therefore, it was named CL2 (i.e., poor sleep quantity model). Cluster 3 ($n = 8$) had extremely poor sleep quality (PSQI 16.25 ± 2.92), with V1 and IV1 being particularly poor. These patients had poor sleep quality, with no sleep satisfaction in either V or IV. Therefore, it was named CL3 (i.e., insufficient sleep quality model).

Table 4 Comparison of five sleep indicators between each model

	CL1	CL2	CL3	*p* value
n	17	23	8	
PSQI	4.24	8.48	16.25	< 0.001
V1	−0.131	−0.245	0.983	0.007
V2	0.619	−0.333	−0.357	0.004
IV1	0.074	0.108	−0.767	0.077
IV2	0.271	−0.295	0.087	0.199

CL1 satisfactory sleep model, *CL2* poor sleep quantity model, *CL3* insufficient sleep quality model, *PSQI* Pittsburgh Sleep Quality Index, *V1* sleep self-determination, *V2* conscious sleep quality, *IV1* continuous deep sleep, *IV2* actual sleep quantity

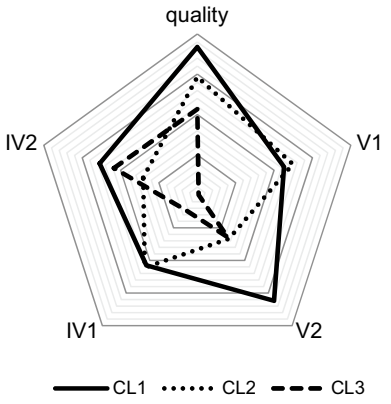

Fig. 1 Radar chart of sleep models. * Quality was calculated as 21 minus the Pittsburgh sleep quality index score. *V1* sleep self-determination, *V2* conscious sleep quantity, *IV1* continuous deep sleep, *IV2* actual sleep quantity, *CL1, Cluster 1* satisfactory sleep model, *CL2, Cluster 2* poor sleep quantity model, *CL3, Cluster 3* insufficient sleep quality model

3.4 Profiling of Sleep Models by Diabetes Control

HbA1c, age, diabetes duration, and body mass index (BMI) were compared among the three sleep models. Table 5 shows the comparison of HbA1c, age, diabetes duration, and BMI among the three sleep models. Compared to CL3, CL1 had significantly lower HbA1c ($7.36 \pm 1.06\%$ vs. $9.14 \pm 2.23\%$, $p = 0.029$); were significantly older (64.24 ± 7.04 years vs. 53.25 ± 9.82 years, $p = 0.031$; and had significantly lower BMI (24.85 ± 4.03 vs. 30.45 ± 4.70, $p = 0.039$). There were no significant differences in diabetes duration among the clusters.

In summary, CL1 was characterized by good HbA1c, older age, and low BMI; CL2 was characterized by moderate HbA1c, age, and BMI; and CL3 was characterized by high HbA1c, young age, and high BMI.

4 Discussion

Sleep disorders, such as reduced sleep quantity and quality, have been reported in most patients with type 2 diabetes [7, 8, 27]. One of authors of this study ([28] and unpublished results) revealed that differences in sleep perception and

Table 5 Comparison of diabetes control between each model

	CL1 ($n=17$) Mean (SD)	CL2 ($n=23$)	CL3 ($n=8$)	CL1 vs CL2 p value	CL2 vs CL3	CL3 vs CL1
HbA1c	7.36 (1.06)	7.89 (1.54)	9.14 (2.23)	n.s	n.s	0.033
Age	64.24 (7.04)	59.30 (10.87)	53.25 (9.82)	n.s	n.s	0.031
Duration	10.41 (6.28)	9.30 (4.23)	8.00 (3.96)	n.s	n.s	n.s
BMI	24.85 (4.03)	26.18 (5.46)	30.45 (4.70)	n.s	n.s	0.039

CL1 satisfactory sleep model, *CL2* poor sleep quantity model, *CL3* insufficient sleep quality model, *BMI* body mass index, *SD* standard deviation, *n.s.* not significant

actuality in diabetics and variability in wake-up and bedtime affect HbA1c. These findings indicate that conscious sleep and unconscious sleep might affect diabetes control. In this study, the sleep structure of adults with type 2 diabetes was clarified by dividing it into V and IV. We found three sleep models by clustering the structure as an index and profiled the models according to diabetes control. There were three unique points in this study.

First, sleep in adults with type 2 diabetes was classified as V or IV. Evidence-based sleep management skills should be handled by community health professionals, such as sleep specialists who develop care models and primary care providers who implement sleep health programs [29, 30]. By doing so, patients reported a reduction in anxiety over sleep problems, feeling better in managing their sleep, and having a sense of control over their sleep [29]. The reason for classifying sleep as V or IV was to clarify the necessity of intervention. Because V can be adjusted consciously, it is expected to be improved by improving the sleep management ability of the patient. IV is beyond the conscious reach of individuals and requires appropriate intervention by medical professionals. Clarifying the responsibility for sleep management enables focused management. Sleep control may be promoted by leaving as much control as possible to the patients themselves.

In this study, V was divided into orthogonal components of V1 (i.e., sleep self-determination) and V2 (i.e., conscious sleep quantity) by principal component analysis. V1 and V2 were thought to represent the voluntary elements of sleep quality and quantity, respectively. Similarly, IV was divided into orthogonal components of IV1 (i.e., continuous deep sleep) and IV2 (i.e., actual sleep quantity), and IV1 and IV2 represented the involuntary elements of sleep quality and quantity, respectively. The results of this study suggested that the concepts of V and IV have both qualitative and quantitative aspects and that these can be applied to evaluate a wide range of sleep elements.

Clustering of clinical phenotypes and symptom patterns of diseases have been widely used [31]. Such profiling may ultimately help to better understand the disease and to predict therapeutic response [32]. In this study, the patients with type 2 diabetes were clustered into three models, as follows: CL1 (i.e., satisfactory sleep model), CL2 (i.e., poor sleep quantity model), and CL3 (i.e., insufficient sleep quality model). These were characterized by a balance of V, IV, and self-assessment of sleep. Based on these results, we believed that it is possible to stratify the risk of sleep disorders by classifying sleep conditions into more uniform clusters. This study suggested that using actual V and IV for clustering could identify patients at high risk for diabetes and may provide personalized medical treatment guidance [33]. Our results suggested that maintaining good sleep has beneficial metabolic effects, which may enhance the current prevention strategies that primarily focus on promoting sufficient sleep and other healthy lifestyles to prevent metabolic diseases [17]. Our data provided evidence that the components of V and IV were associated with a favorable metabolic profile for diabetes and suggested possible sleep interventions to promote good sleep.

In addition, cluster profiling was able to explain the three models based on clinical indicators, such as HbA1c, BMI, and age, which are important in type 2 diabetes. HbA1c and BMI are components of diabetes control and had been canonically correlated with sleep ability [28], thereby, strengthening the robustness of CL1. Our results showed that sleep quality was better with lower HbA1c and lower BMI. Therefore, the quality of diabetes control can be inferred by classification of sleep characteristics and construction of a sleep model, and diabetes may be controlled by sleep intervention. In the future, sleep control for the care of adults with type 2 diabetes should focus on voluntary and involuntary movements, determine sleep items that can be intervened on, and modeling and profiling sleep in relation to diabetes. In terms of clinical application, a patient's sleep model may be identified based on the diabetes control status and appropriately apply it to self-correctable sleep and sleep requiring intervention.

5 Limitation

In this study, we investigated the presence or absence of the common sleep disorder sleep apnea syndrome (SAS), but we did not consider participants with SAS during analysis. In

addition, discrimination between V and IV was subjective. In future studies, a larger sample size may enable statistical classification. Finally, this study was conducted for more than a year, and there may have been seasonal fluctuations in sleep; this is a factor that needs to be considered in future studies.

6 Conclusions

Using the concept of V and IV and self-evaluation, three models of sleep quality and quantity were created in adults with type 2 diabetes. The participants in the satisfactory sleep model had acceptable diabetes control, whereas those in the insufficient sleep quality model had poor diabetes control. Sleep intervention may be an important part of care for adults with type 2 diabetes.

Acknowledgements The authors acknowledge the study participants for their contributions in making this study possible. The authors received no grant funding for this research. This work was supported by JSPS KAKENHI Grant Number 20K19180.

Author contributions AO designed the study, developed and managed the main database, interpreted the analysis, drafted the paper, and MK contributed to the design of the study and interpretation of the data. RA and CK critically revised the manuscript for important intellectual content. All authors have read and approved the final manuscript.

Declarations

Conflict of interest The authors declare no conflicts of interest.

Ethics committee permission This study was conducted with the approval of the Kanazawa University Medical Ethics Review Committee (Approval Number: 742-1).

Research involving human participants All procedures performed in this study were in accordance with the ethical standards of institutional research committee and with the 1964 Helsinki Declaration and its later amendments.

Informed consent All participants provided signed written informed consent before participation in this study.

References

1. Chattu VK, Manzar MD, Kumary S, Burman D, Spence DW, Pandi-Perumal SR. The global problem of insufficient sleep and its serious public health implications. Healthcare (Basel). 2018;7(1):1. https://doi.org/10.3390/healthcare7010001.
2. Hillman D, Mitchell S, Streatfeild J, Burns C, Bruck D, Pezzullo L. The economic cost of inadequate sleep. Sleep. 2018. https://doi.org/10.1093/sleep/zsy083.
3. Covassin N, Singh P. Sleep duration and cardiovascular disease risk: epidemiologic and experimental evidence. Sleep Med Clin. 2016;11(1):81–9. https://doi.org/10.1016/j.jsmc.2015.10.007.
4. Pergola BL, Moonie S, Pharr J, Bungum T, Anderson JL. Sleep duration associated with cardiovascular conditions among adult Nevadans. Sleep Med. 2017;34:209–16. https://doi.org/10.1016/j.sleep.2017.03.006.
5. Qureshi AI, Giles WH, Croft JB, Bliwise DL. Habitual sleep patterns and risk for stroke and coronary heart disease: a 10-year follow-up from NHANES I. Neurology. 1997;48(4):904–11. https://doi.org/10.1212/wnl.48.4.904.
6. Fang J, Wheaton AG, Ayala C. Sleep duration and history of stroke among adults from the USA. J Sleep Res. 2014;23(5):531–7. https://doi.org/10.1111/jsr.12160.
7. Shan Z, Ma H, Xie M, Yan P, Guo Y, Bao W, Rong Y, Jackson CL, Hu FB, Liu L. Sleep duration and risk of type 2 diabetes: a meta-analysis of prospective studies. Diabetes Care. 2015;38(3):529–37. https://doi.org/10.2337/dc14-2073.
8. Lin CL, Tsai YH, Yeh MC. Associations between sleep duration and type 2 diabetes in Taiwanese adults: a population-based study. J Formos Med Assoc. 2016;115(9):779–85. https://doi.org/10.1016/j.jfma.2016.01.013.
9. Clark AJ, Salo P, Lange T, Jennum P, Virtanen M, Pentti J, Kivimäki M, Rod NH, Vahtera J. Onset of impaired sleep and cardiovascular disease risk factors: a longitudinal study. Sleep. 2016;39(9):1709–18. https://doi.org/10.5665/sleep.6098.
10. Gottlieb DJ, Redline S, Nieto FJ, Baldwin CM, Newman AB, Resnick HE, Punjabi NM. Association of usual sleep duration with hypertension: the sleep heart health study. Sleep. 2006;29(8):1009–14. https://doi.org/10.1093/sleep/29.8.1009.
11. Steptoe A, O'Donnell K, Marmot M, Wardle J. Positive affect, psychological well-being, and good sleep. J Psychosom Res. 2008;64(4):409–15. https://doi.org/10.1016/j.jpsychores.2007.11.008.
12. Landry GJ, Best JR, Liu-Ambrose T. Measuring sleep quality in older adults: a comparison using subjective and objective methods. Front Aging Neurosci. 2015;7:166. https://doi.org/10.3389/fnagi.2015.00166.
13. Brandolim Becker N, Jesus SN, Viseu JN, Stobäus CD, Guerreiro M, Domingues RB. Depression and quality of life in older adults: mediation effect of sleep quality. Int J Clin Health Psychol. 2018;18(1):8–17. https://doi.org/10.1016/j.ijchp.2017.10.002.
14. Kim YJ, Yeom HE. Interactive impact of sleep duration and sleep quality on the risk of developing metabolic syndrome in Korean adults. Healthcare (Basel). 2020;8(2):186. https://doi.org/10.3390/healthcare8020186.
15. Štefan L, Sporiš G, Krističević T, Knjaz D. Associations between sleep quality and its domains and insufficient physical activity in a large sample of Croatian young adults: a cross-sectional study. BMJ Open. 2018;8(7): e021902. https://doi.org/10.1136/bmjopen-2018-021902.
16. Zeng Y, Wu J, Yin J, Chen J, Yang S, Fang Y. Association of the combination of sleep duration and sleep quality with quality of life in type 2 diabetes patients. Qual Life Res. 2018;27(12):3123–30. https://doi.org/10.1007/s11136-018-1942-0.

17. Huang T, Redline S. Cross-sectional and prospective associations of actigraphy-assessed sleep regularity with metabolic abnormalities: the multi-ethnic study of atherosclerosis. Diabetes Care. 2019;42(8):1422–9. https://doi.org/10.2337/dc19-0596.
18. Patel SR, Hayes AL, Blackwell T, Evans DS, Ancoli-Israel S, Wing YK, Stone KL, Osteoporotic Fractures in Men (MrOS) and Study of Osteoporotic Fractures (SOF) Research Groups. The association between sleep patterns and obesity in older adults. Int J Obes (London). 2014;38(9):1159–64. https://doi.org/10.1038/ijo.2014.13.
19. Nedeltcheva AV, Scheer FA. Metabolic effects of sleep disruption, links to obesity and diabetes. Curr Opin Endocrinol Diabetes Obes. 2014;21(4):293–8. https://doi.org/10.1097/MED.0000000000000082.
20. Sohail S, Yu L, Bennett DA, Buchman AS, Lim AS. Irregular 24-hour activity rhythms and the metabolic syndrome in older adults. Chronobiol Int. 2015;32(6):802–13. https://doi.org/10.3109/07420528.2015.1041597.
21. Lunsford-Avery JR, Engelhard MM, Navar AM, Kollins SH. Validation of the sleep regularity index in older adults and associations with cardiometabolic risk. Sci Rep. 2018;8(1):14158. https://doi.org/10.1038/s41598-018-32402-5.
22. Forner-Cordero A, Umemura GS, Furtado F, Gonçalves BDSB. Comparison of sleep quality assessed by actigraphy and questionnaires to healthy subjects. Sleep Sci. 2018;11(3):141–5. https://doi.org/10.5935/1984-0063.20180027.
23. Matthews KA, Patel SR, Pantesco EJ, Buysse DJ, Kamarck TW, Lee L, Hall MH. Similarities and differences in estimates of sleep duration by polysomnography, actigraphy, diary, and self-reported habitual sleep in a community sample. Sleep Health. 2018;4(1):96–103. https://doi.org/10.1016/j.sleh.2017.10.011.
24. Doi Y, Minowa M, Uchiyama M, Okawa M, Kim K, Shibui K, Kamei Y. Psychometric assessment of subjective sleep quality using the Japanese version of the Pittsburgh sleep quality index (PSQI-J) in psychiatric disordered and control subjects. Psychiatry Res. 2000;97(2–3):165–72. https://doi.org/10.1016/s0165-1781(00)00232-8.
25. Doi Y, Minowa M, Okawa M, Uchiyama M. Development of the Japanese version of the Pittsburgh sleep quality index. Jpn J Psychiatry Treat. 1998;13(6):755–63 (**in Japanese**).
26. Buysse DJ, Reynolds CF 3rd, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh sleep quality index: a new instrument for psychiatric practice and research. Psychiatry Res. 1989;28(2):193–213. https://doi.org/10.1016/0165-1781(89)90047-4.
27. Barakat S, Abujbara M, Banimustafa R, Batieha A, Ajlouni K. Sleep quality in patients with type 2 diabetes mellitus. J Clin Med Res. 2019;11(4):261–6. https://doi.org/10.14740/jocmr2947w.
28. Oda A, Inagaki M, Tasaki K, Matsui K, Horiguchi T, Koike M. The concept of sleep ability and its effect on diabetes control in adults with type 2 diabetes. Can J Diabetes. 2019;43(5):329–35. https://doi.org/10.1016/j.jcjd.2018.11.007.
29. Gregory P, Morgan K, Lynall A. Improving sleep management in people with Parkinson's. Br J Community Nurs. 2012;17(1):14–20. https://doi.org/10.12968/bjcn.2012.17.1.14.
30. Wesensten, NJ, & Balkin, TJ (2013). The challenge of sleep management in military operations. US Army Med Dep J. 2013; 109–118.
31. Won JC, Im YJ, Lee JH, Kim CH, Kwon HS, Cha BY, Park TS. Clinical phenotype of diabetic peripheral neuropathy and relation to symptom patterns: cluster and factor analysis in patients with type 2 diabetes in Korea. J Diabetes Res. 2017;2017:5751687. https://doi.org/10.1155/2017/5751687.
32. Carpenter JS, Robillard R, Lee RS, Hermens DF, Naismith SL, White D, Whitwell B, Scott EM, Hickie IB. The relationship between sleep-wake cycle and cognitive functioning in young people with affective disorders. PLoS ONE. 2015;10(4): e0124710. https://doi.org/10.1371/journal.pone.0124710.
33. Ye L, Pien GW, Ratcliffe SJ, Björnsdottir E, Arnardottir ES, Pack AI, Benediktsdottir B, Gislason T. The different clinical faces of obstructive sleep apnoea: a cluster analysis. Eur Respir J. 2014;44(6):1600–7. https://doi.org/10.1183/09031936.00032314.