

Contents lists available at [ScienceDirect](https://www.sciencedirect.com)

Canadian Journal of Diabetes

journal homepage:
www.canadianjournalofdiabetes.com


Original Research

The Concept of Sleep Ability and its Effect on Diabetes Control in Adults With Type 2 Diabetes

Azusa Oda MSN^{a,*}; Michiko Inagaki PhD^b; Keiko Tasaki PhD^b; Kiyoko Matsui PhD^b; Tomomi Horiguchi PhD^b; Miki Koike MSN^a

^a Division of Health Sciences, Graduate School of Medical Science, Kanazawa University, Kodatsuno, Kanazawa, Ishikawa, Japan

^b Faculty of Health Sciences, Institute of Medical, Pharmaceutical and Health Sciences, Kanazawa University, Kodatsuno, Kanazawa, Ishikawa, Japan



Key Messages

- Few studies to date have examined the relationship between the various individual components of sleep and diabetes control.
- Canonical correlation analysis identified components of sleep ability representing quality, quantity, maintenance, regularity and recognition of sleep.
- A significant association between components of diabetes control and sleep ability indicates that improving sleep ability may improve diabetes control.

ARTICLE INFO

Article history:

Received 28 February 2018

Received in revised form

9 August 2018

Accepted 6 November 2018

Keywords:

diabetes control

Pittsburgh Sleep Quality Index

Problem Areas in Diabetes questionnaire

sleep

type 2 diabetes

ABSTRACT

Objectives: Few published studies have examined the effects of various components of sleep on the control of type 2 diabetes. This study aimed to construct a concept of *sleep ability* and examine its effect on diabetes control in adults with type 2 diabetes.

Methods: Participants were 37 outpatients, 41 to 73 years of age, who had type 2 diabetes. Participants monitored their sleep for 14 days using a sleep meter, and they completed questionnaires concerning quality of life (Problem Areas in Diabetes), self-care (Self-Care Agency Questionnaire) and sleep quality (Pittsburgh Sleep Quality Index). Data on glycated hemoglobin levels and body mass index were also collected. Canonical correlation analysis and exploratory selection were used to investigate the relationships between the variables involved in diabetes control and sleep ability.

Results: Using canonical correlation analysis and exploratory selection, sleep ability was found to be composed of the Pittsburgh Sleep Quality Index score, objective total sleep time, wake after sleep onset, bedtime standard deviation, wake-up time standard deviation and the absolute value of the difference between subjective and objective sleep efficiency. A significant correlation was found between components of diabetes control (glycated hemoglobin levels, body mass index, quality-of-life evaluation from Problem Areas in Diabetes and self-care evaluation from the Self-Care Agency Questionnaire) and sleep ability (canonical correlation coefficient [R_C] = 0.755, $p=0.006$).

Conclusions: The significant elements of sleep ability represented the quality, quantity, maintenance, regularity and recognition of sleep, and each element made a large contribution to diabetes control. We conclude, therefore, that improving sleep ability may lead to good diabetes control.

© 2018 Canadian Diabetes Association.

R É S U M É

Objectifs : Peu d'études publiées ont porté sur l'examen des effets des divers éléments du sommeil sur la maîtrise du diabète de type 2. La présente étude avait pour objectif l'élaboration d'un concept sur l'« aptitude au sommeil » et l'examen de son effet sur la maîtrise du diabète chez les adultes atteints du diabète de type 2.

Mots clés :

maîtrise du diabète

questionnaire Pittsburgh Sleep Quality

Index

questionnaire Problem Areas in Diabetes

* Address for correspondence: Azusa Oda, MSN, Division of Health Sciences, Graduate School of Medical Science, Kanazawa University, 920-0942, 5-11-80, Kodatsuno, Kanazawa, Ishikawa, 090-5681-3203, Japan.

E-mail address: aznurse440@gmail.com

1499-2671/© 2018 Canadian Diabetes Association.

The Canadian Diabetes Association is the registered owner of the name Diabetes Canada.

<https://doi.org/10.1016/j.cjcd.2018.11.007>

Méthodes : Il s'agissait de 37 participants en consultation externe qui étaient âgés de 41 à 73 ans et qui avaient le diabète de type 2. À l'aide d'un appareil de monitoring du sommeil, les participants ont pu surveiller leur sommeil durant 14 jours et ont rempli les questionnaires sur la qualité de vie (Problem Areas in Diabetes), sur les autosoins (Self-Care Agency Questionnaire) et sur la qualité du sommeil (Pittsburgh Sleep Quality Index). Nous avons collecté les données sur les taux d'hémoglobine glyquée et l'indice de masse corporelle. Nous avons utilisé l'analyse canonique des corrélations et la sélection exploratoire pour examiner les relations entre les variables impliquées dans la maîtrise du diabète et l'aptitude au sommeil.

Résultats : À l'aide de l'analyse canonique des corrélations et de la sélection exploratoire, nous avons observé que l'aptitude au sommeil était composée du score au Pittsburgh Sleep Quality Index, de la durée objective totale de sommeil, du réveil après l'endormissement, de l'écart-type de l'heure du coucher, de l'écart-type de l'heure du réveil et de la valeur absolue de la différence entre l'efficacité subjective et objective du sommeil. Nous avons observé une corrélation importante entre les éléments de la maîtrise du diabète (taux d'hémoglobine glyquée, indice de masse corporelle, évaluation de la qualité de vie selon le Problem Areas in Diabetes et évaluation des autosoins selon le Self-Care Agency Questionnaire) et de l'aptitude au sommeil (coefficient de corrélation canonique $[R_c] = 0,755$, $p = 0,006$).

Conclusions : Les éléments importants de l'aptitude au sommeil représentaient la qualité, la quantité, le maintien, la régularité et la reconnaissance du sommeil, et chaque élément contribuait grandement à la maîtrise du diabète. Par conséquent, nous concluons que l'amélioration de l'aptitude au sommeil peut mener à une bonne maîtrise du diabète.

© 2018 Canadian Diabetes Association.

Introduction

The term diabetes control is often used synonymously with blood glucose control and glycated hemoglobin (A1C) values. However, previous studies have revealed that multifaceted approaches are necessary for diabetes control. The American Association of Diabetes Education has proposed core performance outcomes for the self-management of diabetes, which emphasized the importance of comprehensive diabetes control (1,2). When considering diabetes care, it is, therefore, important to adopt a comprehensive definition of diabetes control.

Recently, sleep care has received attention as a component of diabetes care that complements diet, exercise and medication therapies, which are the 3 pillars of diabetes treatment. Sleep care is a matter of great importance in patients with diabetes who struggle with the medical approach to diabetes control (3). Moreover, impaired sleep causes daytime sleepiness and has been associated with decreased self-management in patients with type 2 diabetes (4).

Poor sleep adversely affects diabetes control by leading to an increase in morning cortisol levels and a decrease in insulin sensitivity and glucose effectiveness (5). Likewise, sleep restriction is associated with a reduction in levels of the appetite-suppressing hormone leptin (6) and an elevation in levels of the appetite-enhancing hormone ghrelin (7,8). Sleep restriction affects energy balance and results in weight gain because of increased appetite, and significant weight gain, in turn, results in insulin resistance, along with diabetes risk (9). Conversely, a decrease in insulin response to glucose causes sympathetic stimulation and parasympathetic inhibition, resulting in poor sleep (10).

When considering the relationship between type 2 diabetes and sleep, both the quality and the quantity of sleep are important (11). Sufficient sleep is essential for overall physical, cognitive and emotional well-being (12), and sleep quality is a significant predictor of A1C levels (13). Thus, the quality and quantity of sleep may have profound effects on patients with type 2 diabetes and should, therefore, be routinely assessed in clinics (14,15).

Matches and mismatches between subjective and objective assessments of sleep have been discussed extensively. Patients with insomnia have been shown to have a tendency to underestimate total sleep time and sleep efficiency (16,17). However, there are only a few reported studies concerning the existence of any such discrepancy in sleep recognition and the role of actual elements of

sleep, such as the expansion of sleep onset latency (SOL), wake after sleep onset (WASO) (18) and fragmentation of sleep (19) in patients with type 2 diabetes. Furthermore, even among studies that have considered each element of sleep, few have recognized sleep as a comprehensive concept in the same way they recognize diabetes control. By perceiving sleep as a composite concept, we propose that multifaceted approaches to sleep in patients with type 2 diabetes could be adopted, leading to good diabetes control.

We have named this composite concept of sleep that may lead to good diabetes control *sleep ability*. In this study, we aimed to construct the concept of sleep ability by identifying the elements of sleep that affect type 2 diabetes. By encouraging patients with type 2 diabetes to adopt sufficient, good-quality sleep, we hope to improve the control of diabetes comprehensively.

Methods

Participants

Outpatients with type 2 diabetes who attended clinics at 2 hospitals in Ishikawa Prefecture, Japan, were identified and asked whether they would give permission to review their medical records, whether they would complete 3 questionnaires and whether they would monitor their sleep for 14 days using a sleep meter. Patients who agreed to all 3 of these requests were enrolled in the study, which included 35 men and 9 women aged 41 to 73 years. Of these 44 participants, we excluded 7 from whom fewer than 3 days of sleep monitoring data were obtained, leaving a total of 37 participants and 440 days of sleep monitoring data for analysis.

Independent variable set sleep ability

Participants were asked to complete the Pittsburgh Sleep Quality Index (PSQI), 1 of the most widely used standardized measures to assess the quality of sleep (20,21). The PSQI consists of 7 categories (C1, sleep quality; C2, sleep latency; C3, sleep duration; C4, habitual sleep efficiency; C5, sleep disturbance; C6, use of sleeping medication; and C7, daytime dysfunction) and evaluates sleep quality according to the total score for the 7 categories. A score of 6 or higher is deemed to indicate sleeping problems. In addition to total point score, we extracted data on subjective total sleep time (sTST) and subjective sleep efficiency (sSE) from categories C3 and C4.

Participants monitored their sleep objectively for 14 days by using an Omron sleep sensor (HSL-101; Omron, Kyoto, Japan). This sensor uses weak radio waves to detect body movements (chest movement, turning, etc.) in order to record periods of sleeping and waking remotely from the bedside. According to the manufacturer's protocol, the usefulness and validity of the instrument in adults have been confirmed (22). Specifically, this instrument has the same degree of coincidence and correlation with polysomnography, so it can be assumed that the detection rate of sleeping and waking periods is sufficient (22). The operation of the sleep sensor was fully explained to each participant, who were asked to activate and deactivate the device by pushing the start/stop button when getting into bed and waking up. After 14 days, sleep sensors were collected, and data on length and depth of sleep were analyzed in chronological order using the WellnessLINK website (23). Variables obtained were bedtime duration (BT), objective total sleep time (σ TST), deep sleep time (DST), sleep onset latency (SOL) and wake after sleep onset (WASO), recorded in units of 1 min. Using these data, objective sleep efficiency (oSE) was calculated using the formula $(\sigma$ TST/BT) \times 100. Bed and waking times were extracted from the graph generated from the data by the WellnessLINK website, and standard deviations of bedtime (BSD) and waking times (WSD) were calculated.

To examine the discrepancy between subjective and objective sleep, the absolute value of the difference between subjective and objective total sleep time (ABS[TST]) and between subjective and objective sleep efficiency (ABS[SE]) were calculated using the formula $|sTST - \sigma$ TST|, $|sSE - oSE|$.

We designated the composite concept of sleep affecting diabetes control as the independent variable set as *sleep ability*. This included 13 elements of sleep data: PSQI, σ TST, sSE, BT, σ TST, DST, SOL, WASO, oSE, BSD, WSD, ABS(TST) and ABS(SE).

Dependent variable set diabetes control

For the purpose of this study, we included A1C, body mass index (BMI), the Problem Areas in Diabetes Survey (PAID) score (24,25) and the Self-Care Agency Questionnaire (SCAQ) score (26) in the concept of diabetes control. These were indicators of glycemic control, weight control, quality of life (QOL) with diabetes and self-care behaviours, respectively (1,27).

The PAID is a 20-item diabetes-specific scale developed to measure the emotional burden related to living with diabetes (25). This measure reflects anxiety about dietary behaviour, hypoglycemia, complications and glycemic control in patients with diabetes (24). It was initially developed to measure the emotional burden of factors affecting the treatment of diabetes, but as data accumulated, it also has become a method for evaluating the QOL of patients with diabetes. In the Japanese version of PAID (28), each item can be rated on a 5-point Likert scale ranging from 1 (no problem) to 5 (serious problem), and the higher the total scale score, the more serious the emotional burden of diabetes and the lower the QOL.

The SCAQ is a 30-item scale measuring the ability of patients with chronic diseases to administer self-care. It includes 4 subscales: 1) ability to perform self-care operations; 2) ability to adjust one's own physical condition based on personal weakness; 3) ability to concentrate one's attention on self-care; and 4) ability to receive valid support (26). Each item is rated on a 5-point Likert scale ranging from 1 to 5; the higher the total scale score, the higher the ability for self-care.

Statistical analysis

Canonical correlation analysis is a multivariate analysis of the association between sets of multiple independent and dependent variables (29). Each set can be named. The canonical correlation

coefficient (R_c) maximizes the correlation between the canonical variate obtained by synthesizing multiple independent and dependent variables. The purpose of our study was to construct an independent variable set named *sleep ability* that affected the dependent variable set, A1C, BMI, PAID and SCAQ scores, which was named *diabetes control*. Canonical correlation analysis was used to determine the relationships among these variables and to identify which variables had the strongest correlations. The relationships between the 2 sets of variables were interpreted using canonical loadings in order to determine how much each variable contributed to its own set and using canonical cross-loadings in order to determine how much each variable contributed to the other set.

The significance level (p) of the correlation was set at 0.05, and a loading >0.30 was regarded as an important contribution. All statistical analyses were performed using IBM SPSS Statistics 24 (IBM, Armonk, New York, United States).

Ethical considerations

This study was conducted with the approval of the Kanazawa University Medical Ethics Review Committee and complied with the principles of the Declaration of Helsinki. The researcher explained to participants 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. Prior written consent was obtained from all participants.

Results

Table 1 shows the participants' characteristics. The mean \pm standard deviation (SD) of A1C was $7.8 \pm 1.6\%$, and 62.2% of participants exceeded the target value of 7.0% set for the prevention of complications by the Japanese Diabetes Society (30). The mean \pm SD of the PSQI was 8.4 ± 4.7 points, which was significantly higher than the 6-point cut-off point considered for impaired sleep ($p=0.004$).

The independent variable set

Data for the 13 elements were standardized because the units of some variables differed. Data were analyzed, applying the assumptions for canonical correlation analysis, and independent variables were narrowed down from 13 elements that were the first draft of sleep ability (Figure 1).

The Shapiro-Wilk test was performed to determine whether each variable was normally distributed, which is a prerequisite for canonical correlation analysis (29,31). This revealed that some variables were not normally distributed. Therefore, quantile-quantile plots and histograms were constructed, and they indicated that oSE, sSE and WSD were not normally distributed. However, WSD did appear to be normally distributed on variable transformation. It was, therefore, included in the analysis, whereas oSE and sSE were excluded from further analysis. All other variables appeared to have approximately normal distributions, so no other variables were transformed.

Multicollinearity was considered when selecting variables (31). Multiple regression analysis was performed with 1 of the remaining 11 variables designated as the dependent variable and others as the independent variables. The variance inflation factor was obtained for each, with a variance inflation factor ≥ 10 indicating the possibility of multicollinearity. We excluded 4 elements (σ TST, DST, SOL and ABS[TST]) with a variance inflation factor ≥ 10 because they indicated multicollinearity. Multicollinearity also occurs when there is a strong correlation among variables. Therefore, we calculated the Pearson correlation coefficient for each set of variables and excluded any variable with $r \approx 1$. At this time, no variables

Table 1
Characteristics of the sample

| Characteristics | n | % | Mean (SD) | Range |
|--|-----------|------------------|--------------|------------|
| Sex (male/female) | 29/8 | 78.4/21.6 | | |
| Age (years) | | | 59.3 (9.8) | 41–73 |
| Diabetes duration (years) | | | 9.8 (5.0) | 1–23 |
| Main treatment: Internal medicine | 15 | 40.5 | | |
| Insulin | 20 | 54.1 | | |
| Incretin | 2 | 5.4 | | |
| Complication: Presence (M/F) | 15 (11/4) | 40.5 (73.3/26.7) | | |
| Obstructive sleep apnea: Presence (M/F) | 4 (3/1) | 10.8 (75.0/25.0) | | |
| Total sleep time: Underestimation/overestimation | 11/26 | 29.7/70.3 | | |
| Sleep efficiency: Underestimation/overestimation | 15/22 | 40.5/59.5 | | |
| Sleep ability | | | | |
| Pittsburgh Sleep Quality Index | | | 8.4 (4.7) | 2–22 |
| Subjective total sleep time (h:min) | | | 6:27 (1:25) | 4:00–10:00 |
| Subjective sleep efficiency (%) | | | 85.7 (16.9) | 22.7–100.0 |
| Bedtime duration (h:min) | | | 6:41 (1:26) | 3:51–9:31 |
| Objective total sleep time (h:min) | | | 5:43 (1:21) | 2:40–9:03 |
| Deep sleep time (h:min) | | | 1:53 (0:41) | 0:49–3:20 |
| SOL sleep onset latency (h:min) | | | 0:34 (0:19) | 0:09–1:27 |
| Wake after sleep onset (h:min) | | | 0:34 (0:32) | 0:02–3:07 |
| Objective sleep efficiency (%) | | | 85.5 (9.0) | 56.0–98.0 |
| Bedtime standard deviation (h:min) | | | 1:03 (0:40) | 0:14–3:06 |
| Wake-up time standard deviation (h:min) | | | 0:49 (0:41) | 0:15–3:06 |
| Absolute value of difference between subjective and objective total sleep time (h:min) | | | 1:13 (1:11) | 0:03–6:20 |
| Absolute value of difference between subjective and objective sleep efficiency | | | 12.2 (12.5) | 0.77–65.6 |
| Diabetes control | | | | |
| Glycated hemoglobin (%) | | | 7.8 (1.6) | 5.4–12.3 |
| Body mass index (kg/m ²) | | | 26.5 (5.3) | 16.8–37.7 |
| Problem areas in diabetes | | | 47.1 (12.8) | 22–72 |
| Self-care Agency Questionnaire | | | 107.2 (17.4) | 72–139 |

were excluded, and 7 elements were left and identified as the components of the independent variable set that comprised the second draft of sleep ability (Fig. 1).

The dependent variable set

The same procedure was performed for the dependent variables A1C, BMI, PAID and SCAQ, and all were found to be normally distributed with no multicollinearity.

Canonical correlation analysis

Table 2 shows the canonical correlation coefficient, canonical weights, canonical loading and canonical cross-loading from the results of the canonical correlation analysis.

Canonical correlation analysis was performed on the dependent and independent variable sets. BT was excluded by exploratory selection from the candidates for the independent variable set that comprised the second draft of sleep ability, and PSQI, oTST, WASO, BSD, WSD and ABS(SE) were set as independent variables. Then, 1 significant canonical variate was found between *diabetes control* and *sleep ability* ($R_C=0.755$; $p=0.006$) (Table 2).

Weighting of variables

The canonical weights of all dependent variables were negative. The canonical weight of WSD was positive, and those of all the other independent variables were negative (Table 2).

Contribution of variables to their own set

In the dependent variable set diabetes control, the absolute value of the canonical loadings of A1C, BMI and PAID scores were

high (0.913, 0.579 and 0.505, respectively), while the absolute value of the canonical loading of the SCAQ score was low (0.003). These results indicated that A1C, BMI and PAID made large contributions, and SCAQ made a smaller contribution to diabetes control. Furthermore, the canonical loadings of A1C, BMI and PAID scores were negative, while the canonical loading of the SCAQ score was positive, indicating that diabetes control was good. In the independent variables set of sleep ability, the large absolute value of the canonical loadings of all independent variables (>0.30) indicated that all were appropriate components of sleep ability. In particular, the large absolute values of the canonical loadings of ABS(SE), PSQI and WSD indicated that these factors made a major contribution to sleep ability. In addition, the canonical loadings of PSQI, WASO, BSD, WSD and ABS(SE) were negative, while the canonical loading of oTST was positive, indicating a high level of sleep ability. The significant canonical correlation between the dependent and independent variable sets indicated that a high level of sleep ability led to a good level of diabetes control.

Contribution of variables to the other set

In the diabetes control set, the canonical cross-loadings of A1C, BMI and PAID score were negative values, whereas the absolute values were large (0.689, 0.437 and 0.382, respectively), so these low values indicated a great contribution to a high level of sleep ability. In the sleep ability set, the canonical cross-loadings of ABS(SE), PSQI, WSD, BSD and WASO were negative, while the absolute values were large (0.668, 0.655, 0.390, 0.376 and 0.335, respectively), so these low values indicated a great contribution to a high level of diabetes control.

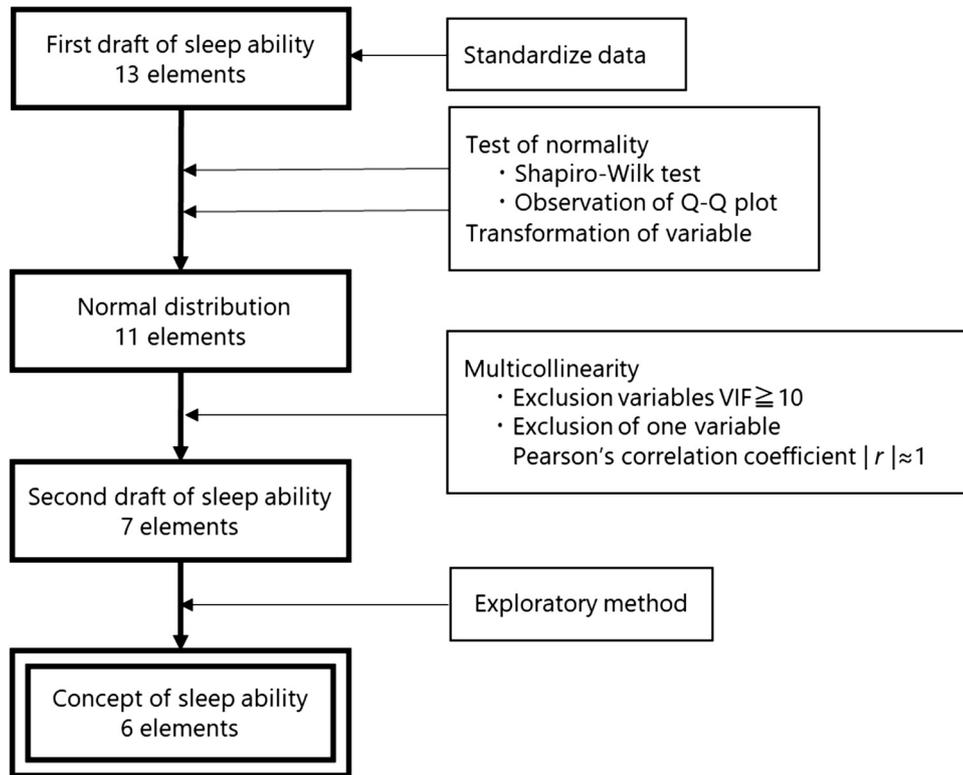


Figure 1. Process for determining sleep ability. Q-Q plot, Quantile-quantile plot; VIF, variance inflation factor.

Discussion

This study aimed to construct a concept of sleep ability that might affect diabetes control in adults with type 2 diabetes by using canonical correlation analysis to identify the elements of sleep ability. We found that sleep ability is composed of 6 elements of sleep: quality of sleep, quantity of sleep, maintenance of sleep, regularity of bedtime, regularity of wake-up time and recognition of sleep, all of which led to good diabetes control. Although relationships between the mechanisms of poor sleep quality or quantity and diabetes have been clarified, our study demonstrated a new approach to show that other aspects of sleep are related to diabetes control, thereby establishing a link between the concept of sleep and the control of type 2 diabetes.

Table 2
Canonical correlation between diabetes control and sleep ability

| | Canonical weights | Canonical loadings | Canonical cross-loadings |
|------------------|-------------------|--------------------|--------------------------|
| Diabetes control | | | |
| A1C | -0.802 | -0.913 | -0.689 |
| BMI | -0.153 | -0.579 | -0.437 |
| PAID | -0.356 | -0.505 | -0.382 |
| SCAQ | -0.222 | 0.003 | 0.002 |
| Sleep ability | | | |
| PSQI | -0.543 | -0.868 | -0.655 |
| oTST | -0.103 | 0.322 | 0.243 |
| WASO | -0.079 | -0.444 | -0.335 |
| BSD | -0.082 | -0.498 | -0.376 |
| WSD | 0.058 | -0.517 | -0.390 |
| ABS(SE) | -0.582 | -0.885 | -0.668 |

A1C, glycated hemoglobin; ABS(SE), absolute value of difference between subjective and objective sleep efficiency; BMI, body mass index; BSD, bedtime standard deviation; o, objective; PAID, Problem Areas in Diabetes; PSQI, Pittsburgh Sleep Quality Index; SCAQ, Self-Care Agency Questionnaire; TST, total sleep time; WASO, wake after sleep onset; WSD, wake-up time standard deviation.
Note: Canonical correlation coefficient: $R_c=0.755$ ($p=0.006$).

Sleep and type 2 diabetes are also related to each other at a molecular level. In a recent study, serum interleukin-6 and tumor necrosis factor-alpha levels were significantly higher in participants with sleep disorders, and the concentrations of both these cytokines were much higher in patients with diabetes and with sleep disorders (32). On the other hand, in metabolic disorders such as type 2 diabetes, the increased production of cytokines (e.g. interleukin-6 and tumor necrosis factor-alpha) may promote and contribute to the development of behavioural symptoms, including sleep problems (33). Therefore, the relationship between sleep disorders and diabetes can be explained by the elevation of serum cytokines. Sleep can be considered a behavioural risk factor for the development of diabetes; therefore, the promotion of lifestyle modifications for adequate sleep could serve as a primary preventive measure against the development of diabetes (34).

In this study, the element that greatly contributed to the 2 sets of variables, the independent and the dependent sets, was the absolute value of the difference between subjective and objective sleep efficiency. In considering the discrepancy in sleep, we used TST and SE, which were available both for subjective and objective measures. SE expresses the rate of actual sleep and combines both quantitative and qualitative aspects of sleep. In this study, ABS(SE), calculated from SE, was extracted as an element of sleep ability through an analytic process. This study revealed that overestimation and underestimation of SE exists in adults with type 2 diabetes. We described the values of SE overestimation and underestimation using absolute values: the greater the discrepancy, the more it is regarded as a factor for impaired sleep ability; thus, the discrepancy could be interpreted as poorer diabetes control. The results of this study suggest that neither overestimation nor underestimation is desirable for diabetes control. However, approximately 60% of the participants considered their sleep to be good, even though their sleep was inadequate; that is, in the overestimation of sleep efficiency, more urgent treatments are needed. In such a situation, no medical treatments were pursued

because they regarded their own sleep as being adequate. This suggests the necessity and usefulness of examining sleep perception in adults with type 2 diabetes.

Although there are practical difficulties in monitoring sleep objectively, we propose that objective sleep monitoring be performed at least once for patients with diabetes who do not improve despite diet, exercise, medication and other therapies. Such monitoring may identify misconceptions concerning sleep recognition, in particular, sleep efficiency, and might reveal information necessary for improved diabetes control. In addition, we also propose sleep monitoring as part of early diabetes education that is effective in enhancing patients' understanding of their sleep tendencies in advance in order to enable the correct recognition of adequate or inadequate sleep. Such monitoring has the potential to reveal information that will improve diabetes control.

Our findings showed that the key elements of sleep ability revealed in this study were PSQI value (quality of sleep), objective total sleep time (quantity of sleep), wake after sleep onset (maintenance of sleep), bedtime SD and wake-up time SD (regularity of sleep), and absolute value of difference between subjective and objective sleep efficiency (recognition of sleep). Although previous studies have considered some of these elements, almost all have examined them as discrete factors that indicate poor sleep. In our study, we demonstrated that the interpretability and significance of good sleep quality and quantity, good maintenance of sleep, regularity of bed and waking times and correct assessment of sleep are factors that indicate good diabetes control. However, conversely, if 1 of the elements is missing, sleep ability is not optimal and may not contribute to good diabetes control. We believe that this balance is important in sleep homeostasis.

In summary, interventions to improve sleep ability in adults with type 2 diabetes include ensuring sufficient quality and quantity of sleep, improving the environment for maintenance of sleep, following a regular daily routine and confirming and reviewing self-assessment of sleep.

The novelty of this study is the use of canonical correlation analysis. Canonical correlation analysis is a useful and powerful technique for determining relationships among multiple dependent variables and independent variables (29). In this study, this method was used for both descriptive and predictive purposes. The 2 variable sets were named *diabetes control* and *sleep ability*, and sleep ability was determined to maximize diabetes control. In particular, for chronic diseases such as type 2 diabetes, medical professionals are required to identify patients and their living environments comprehensively and to promote the optimum health of patients. We consider that the analytic method used in this study is well aligned with the purpose of the study, which was to determine the optimal relationship between diabetes control and sleep ability in patients with diabetes.

Limitations

There are a few limitations to the study that should be considered when interpreting the results. The sleep sensor used in this study detected body motions using radio waves to determine sleep state, sleep onset/wake time, halfway awake time, etc. Therefore, it was impossible to determine the respiratory condition, that is, obstructive sleep apnea (OSA), as an objective measurement value. Furthermore, because we confirmed the presence of OSA only by interviewing the participants, its extent could not be defined. Patients with OSA have shorter sleeping times, lower sleep efficiency and increased arousal times after onset of sleep than patients without OSA (35). In addition, in severe OSA, fasting blood glucose levels, insulin resistance and A1C levels increase (36); hence, it could lead to an increased incidence of type 2 diabetes and the possibility of impaired glycemic control. Thus, patients with OSA

may have decreased sleep ability and poor diabetes control. It is, therefore, necessary to investigate variables, including respiratory conditions during sleep, with a larger number of participants.

Conclusions

Through this study, we proposed a concept of sleep ability and, using canonical correlation analysis, we showed that sleep ability has a strong influence on diabetes control in adults with type 2 diabetes. The components—PSQI score, objective total sleep time, wake after sleep onset, bedtime standard deviation, wake-up time standard deviation and absolute value of difference between subjective and objective sleep efficiency—constituted the concept of sleep ability. We clarified that sleep ability comprised quality, quantity, maintenance, regularity and recognition of sleep. We conclude that diabetes may be successfully controlled by providing care that increases the sleep ability of adults with type 2 diabetes.

Acknowledgments

We are grateful to Dr. Nomura Hideki (Kanazawa University) for help with statistical analysis in this study.

Funding

The authors received no grant funding for this research.

Author Disclosures

Conflicts of interest: None.

Author Contributions

All authors participated in the conception and design of the study and approved the final manuscript submitted; AO collected and analyzed the data; MI, MK, KT, KM and TH provided expert analytical advice on the interpretation of the results; AO drafted the manuscript; MI, MK, KT, KM and TH reviewed the manuscript and provided critical insight.

References

- Mulcahy K, Maryniuk M, Peeples M, et al. Diabetes self-management education core outcomes measures. *Diabetes Educ* 2003;29:768–803.
- American Association of Diabetes Educators. Standards for outcomes measurement of diabetes self-management education. *Diabetes Educ* 2003;29:804–16.
- Khosravan S, Alami A, Rahni SG. Effects of continuous care model based non-pharmacological intervention on sleep quality in patients with type 2 diabetes mellitus: A randomized controlled clinical trial. *Int J Commun Based Nurse Midwif* 2015;3:96–104.
- Chasens ER, Korytkowski M, Sereika SM, Burke LE. Effect of poor sleep quality and excessive daytime sleepiness on factors associated with diabetes self-management. *Diabetes Educ* 2013;39:74–82.
- Stamatakis KA, Punjabi NM. Effects of sleep fragmentation on glucose metabolism in normal subjects. *Chest* 2010;137:95–101.
- Chaput JP, Després JP, Bouchard C, Tremblay A. Short sleep duration is associated with reduced leptin levels and increased adiposity: Results from the Quebec Family Study. *Obesity* 2007;15:253–61.
- Taheri S, Lin L, Austin D, Young T, Mignot E. Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. *PLoS Med* 2004;1:e62.
- Spiegel K, Tasali E, Penev P, Van Cauter E. Brief communication: Sleep curtailment in healthy young men is associated with decreased leptin levels, elevated ghrelin levels, and increased hunger and appetite. *Ann Intern Med* 2004;141:846–50.
- Knutson KL, Spiegel K, Penev P, Van Cauter E. The metabolic consequences of sleep deprivation. *Sleep Med Rev* 2007;11:163–78.
- Spiegel K, Leproult R, Van Cauter E. Impact of sleep debt on metabolic and endocrine function. *Lancet* 1999;354:1435–9.
- Tang Y, Meng L, Li D, et al. Interaction of sleep quality and sleep duration on glycemic control in patients with type 2 diabetes mellitus. *Chin Med J* 2014;127:3543–7.

12. Leahy LG. In search of a good night's sleep. *J Psychosoc Nurs Ment Health Serv* 2017;55:19–26.
13. Knutson KL, Ryden AM, Mander BA, Van Cauter E. Role of sleep duration and quality in risk and severity of type 2 diabetes mellitus. *Arch Intern Med* 2006;166:1768–74.
14. Mesarwi O, Polak J, Jun J, Polotsky VY. Sleep disorders and the development of insulin resistance and obesity. *Endocrinol Metab Clin North Am* 2013;42:617–34.
15. Liu Y, Croft JB, Wheaton AG, et al. Association between perceived insufficient sleep, frequent mental distress, obesity and chronic diseases among US adults, 2009 behavioral risk factor surveillance system. *BMC Pub Health* 2013;13:84.
16. Bianchi MT, Williams KL, McKinney S, Ellenbogen JM. The subjective-objective mismatch in sleep perception among those with insomnia and sleep apnea. *J Sleep Res* 2013;22:557–68.
17. Gooneratne NS, Bellamy SL, Pack F, et al. Case-control study of subjective and objective differences in sleep patterns in older adults with insomnia symptoms. *J Sleep Res* 2011;20:434–44.
18. Johnson ST, Thiel D, Al Sayah F, et al. Objectively measured sleep and health-related quality of life in order adults with type 2 diabetes: A cross-sectional study from the Alberta's Caring for Diabetes Study. *Sleep Health* 2017;3:102–6.
19. Trento M, Broglio F, Riganti F, et al. Sleep abnormalities in type 2 diabetes may be associated with glycemic control. *Acta Diabetol* 2008;45:225–9.
20. Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry Res* 1989;28:193–213.
21. Doi Y, Minowa M, Uchiyama M, et al. 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;97:165–72.
22. Hashisaki M, Nakajima H, Tsuchiya N, et al. Comparative evaluation of noncontact sleep sensor with PSG in Japanese. *J Sleep Res* 2012;37:234.
23. Omron Healthcare Company. WellnessLINK. <https://www.wellnesslink.jp/>. Accessed February 16, 2018.
24. Polonsky WH, Anderson BJ, Lohrer PA, et al. Assessment of diabetes-related distress. *Diabetes Care* 1995;18:754–60.
25. Welch GW, Jacobson AM, Polonsky WH. The problem areas in diabetes scale. *Diabetes Care* 1997;20:760–6.
26. Revision of the Self-Care Agency Questionnaire for patients with chronic illness. *Jpn J Nurs Sci* 2001;21:29–39.
27. Beck J, Greenwood DA, Blanton L, et al, on behalf of the 2017 Standards Revision Taskforce. 2017 National Standards for Diabetes Self-Management Education and Support. *Diabetes Care* 2017;40:1409–19.
28. Ishii H, Welch GW, Jacobson A, et al. The Japanese version of Problem Area in Diabetes Scale: A clinical and research tool for the assessment of emotional functioning among diabetic patients in Japanese. *Diabetes* 1999;48(Suppl):SA319.
29. Hair JF, Black WC, Babin BJ, Anderson RE, Tatham RL. *Multivariate data analysis*. 5th ed. Upper Saddle River, New Jersey, United States: Prentice Hall; 1998.
30. Haneda M, Noda M, Origasa H, et al. Japanese Clinical Practice Guideline for Diabetes 2016. *Diabetology Int* 2018;9:1–45.
31. Sherry A, Henson RK. Conducting and interpreting canonical correlation analysis in personality research: A user-friendly primer. *J Pers Assess* 2005;84:37–48.
32. Huili W, Hua Q, Hang W, Baolan JI, Huacong D. Serum brain-derived neurotrophic factor levels and sleep disorders in Chinese healthy and newly diagnosed type 2 diabetic subjects. *J Diabetes* 2017;9:180–9.
33. Lasselin J, Capuron L. Chronic low-grade inflammation in metabolic disorders: Relevance for behavioral symptoms. *Neuroimmunomodulation* 2014;21:95–101.
34. Chao CY, Wu JS, Yang YC, et al. Sleep duration is a potential risk factor for newly diagnosed type 2 diabetes mellitus. *Metabolism* 2011;60:799–804.
35. Aronsohn RS, Whitmore H, Cauter EV, Tasali E. Impact of untreated obstructive sleep apnea on glucose control on type 2 diabetes. *Am J Respir Crit Care Med* 2010;181:507–13.
36. Bozic J, Galic T, Supe-Domic D, et al. Morning cortisol levels and glucose metabolism parameters in moderate and severe obstructive sleep apnea patients. *Endocrine* 2016;53:730–9.