

# Sleep Pattern, Duration and Quality in Relation with Glycemic Control in People with Type 2 Diabetes Mellitus

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## What's Known

- Napping and segmented sleep are associated with better glycemic control in type 2 diabetes.
- There is a linear correlation between sleep duration and better glycemic control.
- Sleep quality is not related to HbA1C.
- There is no association between sleep variables and lipid profile or microalbuminuria.

## What's New

- Glycemic control is better in patients who take a nap.
- Sleep segmentation is linearly correlated with better glycemic control.

## Abstract

Sleep disturbances have been shown to be associated with diabetes control, but the relation between planned wakings or napping with glycemic indices has not been evaluated yet. This study evaluated the relation between sleep quality, duration, and pattern, including daytime napping of people with diabetes and their glycemic control. A cross-sectional correlation research design was used for this study. We enrolled 118 people with type 2 diabetes receiving oral agents without major complications at the Shahid Bahonar Center, Kerman. The age, weight, height, serum HbA1c, as well as other glycemic indices and lipid profile were measured. BMI was also calculated. All participants were requested to fill in the Pittsburgh Sleep Quality Index (PSQI) questionnaire to evaluate their sleep quality. In addition, they were inquired about their sleep schedule during day and night. Pearson correlation and multiple regression analyses were conducted to examine the correlation between HbA1c and sleep pattern variables. The variables were also compared between participants with or without napping using t-test. All analyses were performed with the SPSS version 19 (SPSS, Chicago, IL, USA). The mean age was 58±11 years and mean HbA1c (%) was 7.8±11 (62±13 mmol/mol). Sleep duration and the number of sleep segments significantly predicted HbA1c (F (2,114)=5.232, P=0.007, R<sup>2</sup>=0.084). A one-hour increment in sleep duration was associated with a 0.174% (1.4 mmol/mol) decrement in HbA1c. PSQI score did not contribute to the regression model. Moreover, participants who napped (66%) had a lower HbA1c (7.6±1) compared to others (8.1±1.3) (P=0.04). We concluded that napping and segmented sleep are associated with a better glycemic control in type 2 diabetes and there is a linear correlation between sleep duration and better glycemic control.

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**Keywords** • Sleep • Diabetes mellitus type 2 • Hemoglobin A glycosylated • Split sleep pattern • Nap

## Introduction

After industrialization, lifestyle has changed considerably as well as sleep habits.<sup>1</sup> People who used to get into bed a few hours after sunset, nowadays remain awake enjoying the advantage of the artificial light. Sleep deprivation is now a public health epidemic. In addition, the napping habit is currently not accepted in many cultures. Do these changes in the natural sleep pattern<sup>2</sup>

could predispose to or protect us from medical conditions such as diabetes mellitus?

Type 2 diabetes mellitus or previously named non-insulin dependent diabetes mellitus is a condition in which the body is not able to maintain normal glycaemic hemostasis. Many investigators have tried to establish the association between sleep, including its duration and quality, and diabetes risk of diabetes development or state of glycaemic control in diabetic patients. Glycaemic control includes short-term control, which is evaluated by assessing blood glucose level, and long-term control, which is estimated by checking glycosylated hemoglobin (HbA1c). HbA1c was first described as an unusual hemoglobin in diabetics by Rahbar et al.<sup>3</sup> Subsequently, this protein was presented as a marker of diabetes control during previous weeks or months.<sup>4</sup> Sleep deprivation has been shown to decrease the insulin sensitivity and impair glucose tolerance.<sup>5-7</sup> Very short sleep duration has also been suggested to increase the odds of diabetes in longitudinal studies.<sup>8-12</sup> Likewise, sleep duration is suggested to be related to glycaemic control in people with diabetes.<sup>13,14</sup> In addition, sleep disturbances have been related to diabetes. In a longitudinal study involving 6,599 non-diabetic men aged 44±4 years with 14 years follow-up, Nilsson et al. reported that difficulties in falling asleep and the use of hypnotic drugs are associated with an increased risk of developing diabetes mellitus (odds ratio [OR] 1.52 [95% CI 1.05-2.20]).<sup>15</sup>

Based on some previous studies, napping has been suggested to increase diabetes risk up to 1.4 compared with those who do not nap.<sup>10,16</sup> Xue et al. in a prospective cohort study on 10,143 normal participants reported that daytime napping is associated with a higher risk of developing diabetes in up to nine years follow-up.<sup>3,10</sup> Similar results were obtained from a cross-sectional study by Lam et al. in China.<sup>4</sup>

However, the aforementioned studies have only evaluated the odds of diabetes in the normal population, but the effect of napping on glycaemic control in people with diabetes has not been evaluated. If we find an answer to this question, it may help physicians to guide their diabetic patients with their glycaemic control.

To the best of our knowledge, the association between sleep pattern and diabetes has not been studied yet. Furthermore, in our country, many people have a regular daytime napping habit and planned wakings during nighttime, especially for night prayers. Previous studies have shown a relationship between sleep deprivation and glycaemic control, and thus, we speculated that such wakings might affect

glycaemic control. This study was prospectively performed in a relatively homogenous group of people with diabetes. Sleep data were collected meticulously. We used the validated translation of the standard Pittsburgh Sleep Quality Index (PSQI) questionnaire to evaluate sleep quality.<sup>17</sup> The PSQI is a self-rated questionnaire that evaluates sleep quality and disturbances over the previous month. This questionnaire includes nineteen items, from which seven components are calculated; including sleep duration, sleep latency, habitual sleep efficiency, sleep disturbances, subjective sleep quality, use of sleeping medication, and daytime dysfunction. Global score is the sum of these seven components. The higher the global score, the worse sleep quality is reported.

This study was designed to evaluate the association between sleep qualities, durations, as well as sleep patterns, including daytime napping and wakings during nighttime of the people with diabetes and their glycaemic control and lipid profile.

## Patients and Methods

### Study Population

A cross-sectional correlation research design was used for this study. We enrolled 118 people with type 2 diabetes older than 25 years without major complications affecting sleep, including ischemic heart disease, renal function impairment, and diabetic neuropathy. Participants were selected among patients who were under routine outpatient follow-up for diabetes control in Shahid Bahonar diabetes clinic. All patients at this center are under close follow-up and any alteration in the laboratory data or symptoms that suggest possible diabetes complication are well managed. Therefore, patients with any record of diabetes complication were excluded from this study. All participants were receiving only oral glycaemic agents. The study was conducted at the diabetes clinic of Shahid Bahonar center in Kerman in 2014. Furthermore, participants with a history of severe obesity, obstructive sleep apnea, psychiatric problems, drug addiction, or other medical conditions affecting sleep were also excluded.

### Variables and Methods

Anthropometric characteristics of the participants including height, weight, and age were recorded and BMI was calculated based on "weight (kg)/height<sup>2</sup> (m)". In addition, laboratory data, including serum HbA1c (%), mmol/mol), fasting blood sugar (FBS) (mg/dl), two-hours postprandial blood sugar (BS)

(mg/dl), random urine micro-albumin (mg/L), and lipid profiles were measured for each individual. Patients with abnormal results of creatinine were excluded. We used the validated translation of the standard Pittsburgh Sleep Quality Index (PSQI) questionnaire to evaluate sleep quality.<sup>17</sup>

This questionnaire includes nineteen items, from which seven components are calculated; including sleep duration, sleep latency, habitual sleep efficiency, sleep disturbances, subjective sleep quality, use of sleeping medication, and daytime dysfunction. Each component is calculated based on the responses to some related questions and finally, the sum of scores for these seven components yields a global score. People with scores equal or less than five are categorized as good sleepers and those with scores higher than five are categorized as poor sleepers. The validity and reliability of the questionnaire were 86 and 89, respectively, as reported in a locally published manuscript (Shahrifar et al. 2009).<sup>17</sup> In addition, the participants were inquired about the time they regularly go to bed, the time they get to sleep, time and number of wakings during night, and the napping time and duration.

#### Statistical Analysis

Spearman correlation test was used to measure the correlation between HbA1c and other continuous variables such as age, body mass index (BMI), waist diameter, global PSQI score, sleep segments, and sleep duration. In addition, the status of mean HbA1c was compared between groups with different sleep segments with ANOVA test. Further, mean HbA1c was compared between those who napped and those who did not nap using student t-test. Similar statistical method was performed to compare mean HbA1c between good sleepers (global PSQI equal or less than 5) and poor sleepers (global PSQI higher than 5). Multiple regression analysis was conducted to examine the relationship between HbA1c and various potential predictors. The variables were also compared between participants with or without napping. All analyses were performed with the SPSS version 19 (SPSS, Chicago, IL, USA). P values less than 0.05 were considered significant.

#### Ethics Statement

This study was approved by the institutional review board of the Kerman University of Medical Sciences (KMU). All participants signed informed consent before enrollment.

## Results

A total of 118 participants (90 males and 28 females) with mean±SD age of 58±11 years were enrolled (table 1). Serum creatinine was higher in men (P=0.001) and BMI as well as total cholesterol were higher in women (P=0.03). Mean HbA1c (%) was 7.8±1.1 (62±13 mmol/mol).

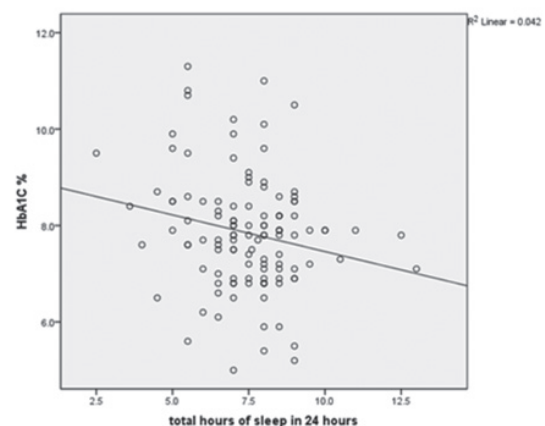
#### Sleep Duration

On average, participants slept 6.6±1.5 hours at night and 1.3±0.6 during daytime. A negative correlation was observed between nighttime sleep duration and HbA1c (r=-0.15, P=0.09, NS). This linear correlation was significant when daytime nap duration was added to nighttime sleep (r=-0.2, P=0.02) (figure 1).

**Table 1:** Demographic and laboratory data of participants, comparing male versus female

	Male	Female	P value
Age	58±11	58±12	0.53
BMI	29±4	31±5	0.03*
Waist (cm)	92±11	97±15	0.11
FBS (mmol/l)	9.3±3.6	9.4±3.4	0.83
BS 2h PP (mmol/dl)	13.3±4.3	13.4±4	0.96
HbA1c (mmol/mol)	62±15	62±12	0.80
HbA1c (%)	7.8±1.4	7.8±1.1	0.80
Serum Cr (µmol/l)	89±13	78±12	0.001
Random urine micro-albumin (mg/l)	27.5±29.3	356±46.4	0.18
Serum triglyceride (mg/dl)	2.1±0.9	1.95±0.8	0.32
Total cholesterol (mg/dl)	4.2±0.9	4.7±1.1	0.03
LDL cholesterol (mg/dl)	2.5±0.9	2.7±0.9	0.29
HDL cholesterol (mg/dl)	1.2±0.3	1.2±0.5	0.49

\*Data are presented as mean±SD



**Figure 1:** Scatter plot showing negative correlation between total hours of sleep in 24 hours and HbA1c of the patients with type 2 diabetes.

### Sleep Patterns

As a scale variable, the number of sleep segments, including nighttime and daytime sleeps showed a linear negative correlation with HbA1c ( $r=-0.23$ ,  $P=0.01$ ). It means that patients who split their sleep in more than one segment during 24 hours have better diabetes control.

As a categorical variable, considering the sleep pattern of the participants during nighttime, 57% slept once at night, 27% in two segments, 13% in three segments, and 3% in four segments. Sixty-six percent of the participants reported that they usually take a daytime nap. HbA1c was compared between the aforementioned groups; mean HbA1c (%) was  $8\pm 1.3$  in individuals who slept once per night,  $7.6\pm 1$  in two segments group,  $7.8\pm 1.2$  in three segments group, and  $6.6\pm 1.5$  in four segments group. In each group, HbA1c was lower in those who took naps (figure 2).

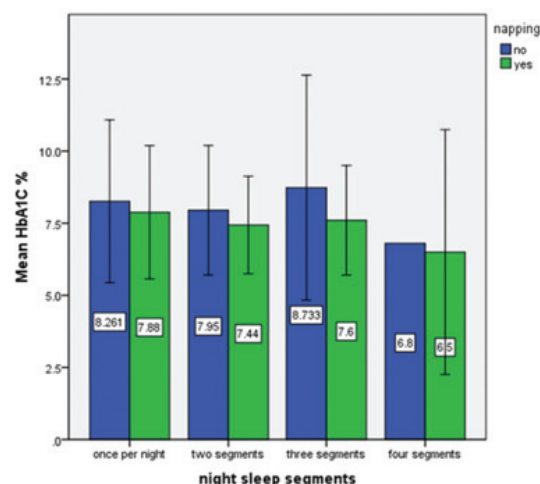
Finally, participants who napped had lower HbA1c ( $7.6\pm 1$ ) compared to others ( $8.1\pm 1.3$ ) ( $t(115)=2.05$ ,  $P=0.04$ ). When we compared those who have a biphasic nighttime sleep with no daytime nap (two segments of sleep), with those who took a daytime nap in addition to an uninterrupted nighttime sleep (two segments of sleep in global), no difference was observed regarding HbA1c level (both  $7.9\pm 1.1$ ).

### Sleep Quality (PSQI)

The mean global PSQI score was  $7.5\pm 3$  scores. Twenty-six percent of participants were good sleepers (global PSQI equal or less than 5). Mean HbA1c was  $7.7\pm 1.2$  in good sleepers and  $7.9\pm 1.2$  in poor sleepers ( $P=0.51$ , NS). As a scale variable, neither global PSQI score nor its component scores were correlated to HbA1c. Global PSQI showed a linear correlation with age ( $r=0.23$ ,  $P=0.01$ ) and waist diameter ( $r=0.19$ ,  $P=0.04$ ). The global PSQI score was not correlated with BMI, years with diabetes, glycemic indices, serum creatinine, urine micro-albumin, or lipid profiles. A negative linear correlation was observed between PSQI score and sleep duration ( $r=-0.24$ ,  $P=0.007$ ). In other words, sleep quality was better in longer sleep durations and fewer segments.

### Multiple Regression Analysis

The correlation of various sleep variables with HbA1c level is summarized in table 2. A multiple regression was run to predict HbA1c from the total 24 h sleep duration, and the number of 24 h sleep segments. These variables significantly predicted HbA1c,



**Figure 2:** Mean HbA1c in participants with type 2 diabetes with different number of nighttime sleep segments. In each category, mean HbA1c is lower in those who take a daytime nap in addition to their nighttime sleep segments.

**Table 2:** Correlation of different sleep variables with HbA1c level of patients with type 2 diabetes mellitus

Variable	Pearson r	P value
Total 24 h sleep duration*	-0.2	0.02***
Nighttime sleep duration	-0.15	0.09
Total number of 24 h sleep segments**	-0.23	0.01*
Sleep quality (global PSQI score)	0.05	0.58

\*Including daytime nap added to nighttime sleep duration;

\*\*Including daytime nap and nighttime sleep segments;

\*\*\*Significant

( $F(2,114)=5.232$ ,  $P=0.007$ ,  $R^2=0.084$ ). PSQI score did not contribute to the regression model. A one-hour increment in sleep duration was associated with a 0.174% (1.4 mmol/mol) decrement in HbA1c. Furthermore, adding one segment to the sleep pattern was associated with a 0.257% (2.8 mmol/mol) decrement in HbA1c. The predicted equation is presented as:

$$\text{HbA1c (\%)} \text{ predicted} = -0.257 \times \text{sleep segments} - 0.174 \times \text{total sleep duration} + 9.391.$$

Finally, we ran multiple regression test to check if there are any probable confounding factors affecting the relation of these sleep variables with HbA1c level. Variables including age, duration of diabetes, gender, waist diameter, BMI and total cholesterol were entered into the regression model from which only BMI and waist diameter significantly altered the effect size of the previously entered sleep variables ( $F(4,112)=3.833$ ,  $P=0.006$ ,  $R^2=0.124$ ). The new prediction equation is:

$$\text{HbA1c (\%)} \text{ predicted} = -0.294 \times \text{sleep segments} - 0.150 \times \text{total sleep duration} - 0.016 \times \text{waist diameter} + 0.047 \times \text{BMI} + 9.511.$$

### *Association between Sleep Variables and other Metabolic Variables*

There was no association between sleep duration and quality as well as sleep segments or napping with lipid profile status of the patients, including triglyceride level, total cholesterol, LDL cholesterol, and HDL cholesterol. Likewise, random urine micro albumin was not affected by the sleep status of patients.

## Discussion

We observed that HbA1c level had a negative linear correlation with the number of sleep segments and sleep duration. In other words, patients who split their sleep in more than one segment during 24 hours have a lower HbA1c. Likewise, the longer the patients sleep in 24 hours, the better their diabetes is controlled. Although some previous studies have shown that sleep duration is related to the risk of diabetes<sup>8,11,18,19</sup> or worse glycaemic control in people with diabetes,<sup>13,14</sup> to the best of our knowledge, the relation between glycaemic control and sleep segments has not been reported thus far. Our findings reveal that participants who split their sleep have a better glycaemic control than those who have a monophasic uninterrupted night sleep. This effect on glycaemic control was independent of sleep duration or quality. Our study indicates that participants with the monophasic sleep pattern show worse glycaemic control compared to those who have split pattern of sleep. Although our study did not include normal individuals, these results may corroborate the theory that segmented sleep is more natural.<sup>2</sup>

In contrast with some previous studies, which have suggested that daytime napping is associated with increased risk of diabetes,<sup>10,16</sup> we demonstrated that napping is associated with a better glycaemic control. This correlation has not been studied in people with diabetes before. Xue et al. followed a cohort of 10,143 normal participants and reported that daytime napping is associated with a higher risk of developing diabetes in a follow-up period of around nine years.<sup>3</sup> Similar results were obtained from a cross-sectional study by Lam et al. in China.<sup>4</sup> They analyzed data from 19,567 Chinese men and women aged 50 years or older. The data originated from the Guangzhou Biobank Cohort Study, which was a community-based association study in Guangzhou, China. Data of napping habit was obtained by a self-reported questionnaire and type 2 diabetes was assessed by fasting blood glucose and/or self-reports of physician diagnosis or treatment. Participants

reporting frequent naps were more likely to have diabetes. After adjustments were made for demographics, health status, lifestyle and sleep habits, adiposity, and metabolic markers, these relationships remained unchanged. They stated a similar association between napping and impaired fasting glucose. The authors concluded that napping is associated with elevated prevalence of diabetes and impaired fasting glucose. However, this association could not be translated as a cause-effect relationship. Nevertheless, the aforementioned studies have only evaluated the odds of diabetes in the normal population, but the effect of napping on glycaemic control in people with diabetes had not been evaluated in previous studies. We detected a better glycaemic control in people with diabetes who napped during daytime. It seems that napping works on glucose metabolism by increasing sleep segments. In fact, glycaemic control was not different in participants who have two nighttime sleep segments without nap compared with those who have an episode of uninterrupted nighttime sleep plus daytime napping. Both aforementioned groups had two sleep segments in total and presented a similar HbA1c level. Since our study population was limited, additional detailed comparisons were impossible in order to separate the role of napping itself from its contribution as a sleep segment.

With respect to sleep duration, we found that total sleep duration was linearly correlated with better glycaemic control. Borel et al. found a similar correlation in people with type 1 diabetes mellitus.<sup>20</sup> These findings support the results of the Byberg et al. study, which showed a negative linear correlation between sleep duration and HbA1c.<sup>14</sup> A total of 771 participants were selected from a Danish population-based cross-sectional 'Health 2008' study. They measured sleep duration and sleep quality as well as markers of glucose homeostasis, including fasting plasma glucose, 2-h postprandial plasma glucose, HbA1c, two measures of insulin sensitivity (the insulin sensitivity index (0,120) and homeostasis model assessment of insulin sensitivity), the homeostasis model assessment of beta-cell function and glucose tolerance status. Multiple linear and logistic regression was performed to evaluate the associations of sleep duration and sleep quality with markers of glucose homeostasis and tolerance. Finally, they showed that a one-hour increment in sleep duration was associated with a 0.3% (3 mmol/mol) decrement in HbA1c. This proportion was 0.17% (1.4 mmol/mol) in our study. However, it should be stated that small R square in our predicting

model shows that these predictors could not strongly predict the HbA1c level. Byberg's study was a well-designed research that evaluated glucose metabolism in relation to sleep duration and quality; however, on the one hand, the study population included normal healthy population and the conclusion may not be extrapolated to diabetics. On the other hand, they did not mention sleep pattern, which is shown to be associated with glycemic control in our study. Two recent studies on people with diabetes in Japan and Korea have suggested a U shaped correlation between sleep duration and insulin resistance or glycemic control.<sup>13,21</sup> To reiterate, there is a strong consensus that short sleep is associated with worse glycemic control but considering long sleep, there is controversy among different studies.

Regarding sleep quality, our findings are in concordance with other studies suggesting that there is no correlation between sleep quality and glycemic control in people with diabetes.<sup>22,23</sup> Byberg et al. found poor sleep quality to be associated with earlier alterations in glucose homeostasis, including insulin sensitivity and beta-cell function, but not with later alterations in glucose homeostasis, i.e. HbA1c, which was related only with sleep duration.<sup>14</sup> We observed that the sleep quality was decreased when sleep segments were increased. This decrease in the PSQI score may be due to the fact that 87 percent of our participants were waking up during the night. In fact, PSQI questionnaire could not separate planned wakings from insomnia.

Finally, considering other metabolic markers, including serum lipid profile or random urine albumin concentration; we did not find any correlation between those markers with sleep duration, pattern, or quality of our patients. In a recently published manuscript, Ohkuma and coworkers detected that sleep duration has a U shape correlation with urine albumin to creatinine ratio.<sup>24</sup>

The main limitation of our study was the limited number of participants. Furthermore, this study was a cross-sectional correlation study and our findings could not be translated as cause and effect relationship, as diabetes itself independent of the obesity can cause sleep disruption.<sup>25</sup> An interventional study could be designed to evaluate the effect of any alteration in sleep pattern on glycemic control more precisely.

## Conclusion

This study indicates that, (i) napping and segmented sleep are associated with better

glycemic control in type 2 diabetes, (ii) there is a linear correlation between sleep duration and better glycemic control, (iii) sleep quality is not related with HbA1c, and (iv) there was no association between sleep variables and lipid profile or micro albuminuria.

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**Conflict of Interest:** None declared.

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