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Sleep duration is a potential risk factor for newly diagnosed type 2 diabetes mellitus

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Abstract

U-shaped patterns have been observed for the relationship between sleep duration and diabetes. In addition, prediabetes is associated with the risk of cardiovascular diseases and diabetes. However, there are few studies investigating the relationship between sleep duration and prediabetes/newly diagnosed diabetes. The aim of this study is to examine the relationship between sleep duration and prediabetes/newly diagnosed diabetes in a Taiwanese population. After excluding the subjects with a high risk of obstructive sleep apnea, those with a positive history of diabetes, or those taking hypnotic drugs, a total of 3470 adults were recruited from a health checkup center. Each subject completed a self-administrated structured questionnaire on sleep duration and lifestyle factors. Prediabetes/diabetes was defined following the definition of the American Diabetes Association. Subjects with different sleep durations were classified into short (<6.0 hours), normal (6.0~8.49 hours), and long sleepers (≥8.5 hours). The proportion of subjects with normal glucose tolerance, prediabetes, and newly diagnosed diabetes was 71.9%, 22.9%, and 5.2%, respectively. There were significant differences in age, sex, weight, education level, body mass index, waist-to-hip ratio, systolic and diastolic blood pressure, alcohol and coffee drinking habits, family history of diabetes, and sleep duration among the 3 glycemic groups. In multinomial regression, both short and long sleepers had a higher risk of newly diagnosed diabetes; and the odds ratio were 1.55 (95% confidence interval, 1.07-2.24) and 2.83 (1.19-6.73), respectively. However, sleep duration was not found to relate to prediabetes. In conclusion, both short and long sleep durations were independently associated with newly diagnosed diabetes, but not with prediabetes.

1. Introduction

Humans spend nearly one third of each day sleeping, and it is a daily process of physiologic restitution and recovery. An epidemiologic study reported a decline in sleep duration over the past few decades by 1.5 to 2 hours [1], with about one third of adults reporting that they sleep less than 6 hours per night, meaning that we live in a sleep-deprived society. U-shaped patterns have been observed for the relationships between sleep duration and all-cause mortality [2-4], coronary heart disease [5], hypertension [6,7], obesity [7,8], and diabetes [9-13]. Sleep and sleep disturbance have a profound effect on many aspects of physiologic functions, including endocrinology, immunology, and metabolism; and decreased glucose tolerance and increased sympathetic tone are well-known risk factors for the development insulin resistance, obesity, and hypertension [14]. Therefore, we could speculate that chronic sleep loss would be a stress factor to promote weight gain and impair glycemic regulation, with a subsequently increased risk of diabetes.

Diabetes remains a critical public health problem because of its high prevalence rate and the fact that it increases the risk of cardiovascular-related mortality. There is growing evidence that sleep duration is a contributing factor toward the current diabetes epidemic [9-13]. An experimental study showed that acute deprivation of sleep, down to 4 hours per night for 6 nights, decreased glucose tolerance in healthy

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young adults. Therefore, the impact of sleep restriction on glucose regulation suggests a mechanism whereby short sleep time might increase the risk of diabetes [15]. Previous epidemiologic studies on the relationship between sleep duration and diabetes have shown varying results [9-13, 16-19]. A cohort study of women in the Nurses Health Study showed that short (≤5 hours) and long sleepers (≥9 hours) had a significantly increased risk of diabetes, but the relationship remained significant only between long sleepers and diabetes after adjusting for body mass index (BMI) [9]. However, sleep disturbance in midlife was found to be unrelated to 32-year diabetes incidence in a Gothenburg study [17]. Mallon et al [18] found that short sleep duration was associated with diabetes incidence in men, but not in women. Furthermore, a prospective cohort study of middle-aged and elderly men showed a significant U-shaped relationship between self-reported sleep duration and incidence of diabetes [10]. Recent studies have found both short and long sleep duration to be associated with significantly increased risk of diabetes [12,13]. In addition, prediabetes had been associated with the increased risk of cardiovascular diseases [20] and overt diabetes [21]. However, few studies investigate the relationship between sleep duration and newly diagnosed diabetes and prediabetes. Therefore, the aim of this study is to examine the potential effect of sleep duration on the risk of prediabetes and newly diagnosed diabetes, controlling for possible confounding factors.

2. Methods

2.1. Study subjects and data collection

The baseline data were collected from a health examination center in National Cheng Kung University Hospital from 2006 to 2007. All subjects received a health checkup and completed a structured questionnaire, which included demographic information; medical history; medication history; smoking, alcohol, and coffee drinking habits; and details of physical exercise. After excluding the subjects with high risk of obstructive sleep apnea (OSA), a total of 3470 adults without a history of diabetes or taking hypnotic drugs or antidepressants were recruited in our study (2145 men, 61.8%; 1325 women, 38.2%). Informed consent was obtained from all of the study participants. Because they only agreed to have their questionnaire data and related examination results analyzed anonymously, any identifying information was kept confidential. The Ethical Committee for Human Research at the National Cheng Kung University Hospital approved the study protocol.

2.2. Anthropometric and laboratory measurements

Anthropometric measurements, blood pressure (BP), and blood sampling were carried out by well-trained nurses. Body weight (to the nearest 0.1 kg) and height (to the nearest 0.1 cm) were measured using a certified machine. The BMI was calculated as weight in kilograms divided by the square of height in meters. Waist circumference was measured from the midway between the lower rib margin and the iliac crest with the subjects standing at the end of normal expiration, and hip circumference was measured at the level of the greater trochanter. Two readings of BP (systolic and diastolic BP) were measured in a supine position with a BP monitor (1846SX; Johnson and Johnson, assembled in Mexico) after at least 15 minutes of rest. All subjects who did not have a medical history of diabetes received 75-g oral glucose tolerance test after a 10-hour overnight fast, a normal diet for 3 days before the test, and abstention from smoking for more than 24 hours. None of the women were pregnant when tested. Diabetes and prediabetes, including both impaired fasting glucose (IFG) and impaired glucose tolerance (IGT), were defined according to the diagnostic criteria of the American Diabetes Association. *Diabetes mellitus* (DM) was defined as a fasting glucose level of 126 mg/dL or more, or a 2-hour postload glucose level of 200 mg/dL or more. *Impaired fasting glucose* was defined as a fasting glucose level between 100 and 126 mg/dL and a 2-hour postload glucose level less than 140 mg/dL. *Impaired glucose tolerance* was defined as a 2-hour postload glucose level of 140 mg/dL or more in subjects not meeting the criteria for DM.

Hypertension was defined as systolic BP greater than 140 mm Hg or/and diastolic BP greater than 90 mm Hg. *Obesity* was defined as BMI greater than 25 according to the World Health Organization Asian-Pacific diagnostic criteria, where *central obesity* was defined as waist-to-hip ratio (WHR) greater than 0.9 in males or 0.8 in females.

2.3. Assessment of lifestyle and other factors

Smoking habit was classified as current smokers (defined by at least 1 pack per month) and nonsmokers. Alcohol consumption was classified as drinkers (defined by at least 1 drink per week) and nondrinkers. Coffee drinking habit was categorized as 2 groups: none or less than 2 times per week, and 3 times per week or more. Regular physical exercise was defined according to the recommendation of the American College of Sport Medicine guideline of vigorous exercise at least 3 times weekly [22] (intense enough to cause sweating and/or heavy breathing and/or increase heart rate to a certain amount). Furthermore, physical exercise was categorized into 2 groups: none to less than 3 times per week, and 3 times or more per week. A positive family history of diabetes was defined as at least 1 of the first-degree relatives having diabetes.

2.4. Sleep duration assessment

The number of hours of sleep was assessed by the following question inserted into a self-reported questionnaire: “On average, how many hours and minutes do you sleep per night?” We further classified sleep duration into

3 groups: short (<6 hours), normal (6-8.49 hours), and long (≥8.5 hours).

2.5. Statistical analyses

All statistical analyses were performed by using the 15th version of the SPSS (Chicago, IL) software. The subjects were classified into 3 groups by their glycemic status: normal glucose tolerance (NGT), prediabetes, and diabetes. Clinical characteristics in the study were presented as mean ± SD or percentage. χ^2 tests were used to compare the categorical variables among the 3 groups. In addition, the comparisons of the continuous variables were analyzed by analysis of variance among the 3 groups. In the multinomial regression, we chose normal sleepers as the reference group and explored the relationship between sleep duration and glycemic status after adjustments for age; sex; smoking, alcohol, and coffee drinking habits; physical exercise; family history of diabetes; and obesity. The odds ratio (OR) and 95% confidence interval (CI) of predictors were derived for each regression model. Statistical significance was defined as a P value < .05.

Table 1
Clinical characteristics of the study subjects according to glycemic status

Variables	NGT (n = 2495)	Prediabetes (n = 795)	Diabetes (n = 180)	P value
Male	58.9	69.6	67.8	.000‡
Age (y)	42.5 ± 11.3	49.1 ± 11.6	52.0 ± 11.3	.000‡
Educational level (>12 y)	72.9	66.0	48.9	.000‡
Height (cm)	165.0 ± 8.2	164.8 ± 8.0	163.5 ± 8.0	.055
Weight (kg)	64.5 ± 11.9	68.9 ± 12.3	69.8 ± 12.9	.000‡
BMI (kg/m^2)	23.6 ± 3.3	25.1 ± 3.4	26.0 ± 4.0	.000‡
General obesity (BMI ≥25 kg/m^2)	29.7	47.9	59.4	.000‡
Central obesity (WHR: male >0.9; female >0.8)	35.1	58.2	77.8	.000‡
BP (mm Hg)				
Systolic	111.8 ± 13.4	118.9 ± 20.9	122.1 ± 15.7	.000‡
Diastolic	66.4 ± 9.1	70.4 ± 9.3	73.3 ± 9.1	.000‡
Family history of diabetes	23.9	29.8	35.0	.000‡
Family history of hypertension	40.4	41.6	38.9	.730
Lifestyle factors				
Cigarette smoking	17.0	17.9	22.8	.131
Alcohol drinking	15.8	22.4	28.9	.000‡
Coffee drinking	36.1	32.3	22.8	.000‡
Physical exercise (≥3 times/wk)	12.7	9.7	9.4	.035*
Sleep duration (h/night)				
<6	16.0	19.5	26.1	
6-8.49	81.6	78.1	70.0	.003†
≥8.5	2.4	2.4	3.9	
Sleep h/night	6.5 ± 1.1	6.4 ± 1.2	6.3 ± 1.2	.028*

Data are presented as mean ± SD or percentage. BMI, body mass index; WHR, waist-to-hip ratio.
* P < .05.
† P < .01.
‡ P < .001.

3. Results

Table 1 summarizes the clinical characteristics of the 3470 subjects according to glycemic status. There were significant differences in sex, age, weight, education level, BMI, WHR, systolic and diastolic BP, alcohol and coffee drinking habits, family history of diabetes, and sleep duration among these 3 glycemic groups. Compared with the NGT group, both prediabetes and diabetes groups were older; had a higher BMI, WHR, body weight, systolic and diastolic BP, as well as a higher proportion of males, family history of diabetes, and alcohol drinking habit; and were short sleepers, but had a lower proportion of high education level, regular physical exercise, and coffee drinking habits.

Table 2 shows the characteristics of the study subjects according to the sleep duration categories. There were significant differences in sex, age, education level, body height, BMI, WHR, diastolic BP, cigarette smoking, and coffee drinking habits among these 3 sleep groups. An inverse association between age and sleep duration was observed. Short sleepers were associated with older age, higher BMI, and lower education level. Long sleep was associated with being male, being younger, and having a

Table 2
Clinical characteristics of the study subjects according to sleep duration

Variables	< 6 h (n = 602)	6-8.49 h (n = 2782)	≥8.5 h (n = 86)	P value
Male	59.6	62.7	50.0	.009†
Age (y)	46.3 ± 12.5	44.3 ± 11.6	38.1 ± 12.4	.000‡
Education level (>12 y)	62.5	71.7	68.8	.000‡
Height (cm)	164.0 ± 8.2	165.1 ± 8.1	163.9 ± 8.4	.010*
Weight (kg)	66.0 ± 12.8	65.8 ± 12.0	63.7 ± 14.6	.272
BMI (kg/m^2)	24.4 ± 3.7	24.1 ± 3.4	23.6 ± 4.5	.023*
General obesity (BMI ≥25 kg/m^2)	39.4	34.7	31.4	.064
Central obesity (WHR: male >0.9; female >0.8)	47.8	41.5	43.0	.020*
BP (mm Hg)				
Systolic	114.1 ± 14.3	114.0 ± 16.3	111.1 ± 16.0	.253
Diastolic	67.8 ± 9.1	67.7 ± 9.4	64.8 ± 9.9	.018*
Family history of diabetes	25.9	25.7	29.1	.788
Family history of hypertension	37.8	41.3	36.1	.190
Lifestyle factors				
Cigarette smoking	20.7	16.5	24.4	.013*
Alcohol drinking	20.8	17.3	20.9	.105
Coffee drinking	30.1	35.3	39.5	.030*
Physical exercise (≥3 times/wk)	10.1	12.4	8.1	.158
Sleep duration (h/night)	4.7 ± 0.7	6.7 ± 0.7	8.9 ± 0.9	.000‡

Data are presented as mean ± SD or percentage. BMI, body mass index; WHR, waist-to-hip ratio.
* P < .05.
† P < .01.
‡ P < .001.

Table 3
Multinomial regression for the presence of diabetes and prediabetes by clinical variables

Variables	Diabetes	*P* value	Prediabetes	*P* value
Sleep duration (h)				
< 6 vs 6-8.49	1.55(1.07-2.24)	.022*	1.14(0.91-1.41)	.256
≥8.5 vs 6-8.49	2.83(1.19-6.73)	.018*	1.47(0.84-2.55)	.174
Age (y)	1.05(1.04-1.07)	.000‡	1.04(1.04-1.05)	.000‡
Sex (female vs male)	0.79(0.53-1.16)	.244	0.70(0.57-0.86)	.001†
Educational level (>12 vs ≤12 y)	0.67(0.48-0.95)	.024*	1.06(0.87-1.29)	.544
Family history of diabetes (yes vs no)	1.82(1.30-2.56)	.001†	1.40(1.16-1.68)	.001†
Cigarette smoking (yes vs no)	1.07(0.70-1.65)	.743	0.87(0.68-1.10)	.246
Alcohol drinking (yes vs no)	1.79(1.21-2.66)	.004†	1.30(1.04-1.63)	.021*
Coffee drinking (yes vs no)	0.59(0.41-0.87)	.007†	0.89(0.74-1.07)	.211
Physical exercise (≥3 times/wk vs <3 times/wk)	0.69(0.40-1.17)	.165	0.63(0.48-0.83)	.001†
General obesity (yes vs no)	2.28(1.62-3.21)	.000‡	1.68(1.40-2.02)	.000‡
Central obesity (yes vs no)	3.27(2.21-4.83)	.000‡	1.70(1.42-2.05)	.000‡

Data are given as ORs (95% CIs) of independent variables for the presence of diabetes or prediabetes relative to NGT from multinomial regression.
* $P < .05$.
† $P < .01$.
‡ $P < .001$.

leaner body. Sleep duration was not significantly associated with body weight and general obesity (BMI); however, it was significantly associated with central obesity (WHR). Specifically, we found short sleep to be associated with obesity. Compared with normal sleepers, both short and long sleepers had a higher proportion of cigarette smoking and alcohol drinking habit, but a lower proportion of regular physical exercise.

The ORs and 95% CIs of the independent variables for predicting diabetes and prediabetes from multinomial regression are shown in Table 3. The shorter and longer sleepers were at the greatest risk of diabetes, even after adjustments for age, sex, education level, family history of diabetes, cigarette smoking, alcohol and coffee drinking, physical exercise, and general and central obesity.

4. Conclusions

Our study shows that both short and long sleep durations are associated with newly diagnosed diabetes after adjustments for the potential confounding factors of lifestyle and family history of diabetes. Furthermore, this association remained significant even after controlling for both general and central obesity indices (BMI and WHR). Thus, both short and long sleep durations independently increase the risk of newly diagnosed diabetes in a Taiwanese population.

Epidemiologic studies have evaluated the association between sleep duration and the risk of diabetes, but the results were inconsistent [9-13,17-19]. Although some studies showed a U-shaped pattern for the relationship between sleep duration and IGT [12,13] or diabetes [9-13], no uniform relationship has been found, with some studies even showing negative associations [17-19]. This discrepancy may be due to the racial/ethnic and/or demographic effects of the following factors on sleep duration: physical, mental, and social conditions or different genetic backgrounds.

The Nurses Health Study showed that the association between short sleep duration and diabetes was attenuated or even disappeared after adjustment of BMI or obesity [9]; and therefore, the effect of short sleep duration on diabetes was related in part to the influence of obesity. However, our study and other works [10-13] found short sleep duration increased the risk of diabetes after controlling for both general and central obesity; that is, short sleep duration had a direct effect on the risk of diabetes, independent of obesity. There are some mechanisms other than obesity that could explain the relationship between short sleep duration and diabetes. In experimental studies, sleep deprivation was associated with increased sympathetic tone and elevated cortisol levels via activation of hypothalamic-pituitary-adrenal axis, decreased glucose tolerance, increased insulin resistance, and increased hunger and appetite by decreasing the anorexic hormone leptin and increasing the orexigenic ghrelin [15,16]. That is, chronic sleep deprivation may increase the burden on the pancreas due to insulin resistance, compromising its function and subsequently increasing the risk of diabetes over time.

In recent studies, short sleep duration was not consistently associated with IGT [12,13]; and the relationship was also not found in our study. Gottlieb et al [12] showed that a sleep duration of 6 and at least 9 hours per night was associated with a higher adjusted OR for IGT and DM, but sleep of 5 hours per night or less was not related to the risk of IGT. However, Chaput et al [13] found that both short and long sleep durations were associated with the risk of diabetes/IGT. There are some possible explanations for this discrepancy. First, compared with our study, the subjects in the study of Gottieb et al were older (mean age, 70.2 vs 44.5 years) and more obese (mean BMI, 28.1 vs 24.1) [12]. Second, the effect of short sleep duration on the risk of diabetes has been shown to vary by ethnic group [19]. Third, the study of Chaput et al [13] had a small sample size; so caution is needed when interpreting their findings.

Previous studies found that long sleep duration was associated with an increased risk of developing diabetes [9-13]. However, the mechanisms mediating the association of long sleep duration and diabetes were more speculative, including less physical activity [9], deleterious effects on glucose homeostasis of proinflammatory cytokines [23,24], and longer sleep duration to compensate for poor sleep quality [10]. In our study, the long sleepers (≥8.5 hours) had a higher risk of newly diagnosed diabetes; and that is

consistent with some studies [17,19], but contrary to others [9-13]. There are some possible explanations for this discrepancy. First is the possibility of confounding by unmeasured variables, such as sleep quality, with poor sleep quality having been found to be associated with long sleep duration and the incidence of diabetes [9,10]. Second, genetic or ethnic characteristics could result in differential vulnerability to the effect of long sleep duration on insulin sensitivity [19]. Third, individuals suffering from diabetes report higher rates of insomnia, excessive daytime sleepiness, and unpleasant sensations in the legs that disturb sleep. Therefore, we excluded subjects with a positive diabetes history because of the fact that long sleep duration may be a sequela of diabetes and to prevent overestimating the potential effect of long sleep duration on diabetic risk [11]. Finally, OSA may also have contributed to the risk of type 2 DM, although most previous studies investigating the relation between sleep duration and diabetes did not exclude subjects with OSA [25]. In our study, the percentage of the population at a higher risk of OSA (defined as the combination of being male sex and having hypertension and obesity) was around 4.0%, which is similar to the prevalence of symptomatic OSA in a community-based study in Hong Kong [26]. Therefore, we excluded the subjects with high risk of OSA to eliminate its confounding effect; and the multinomial regression still showed that both short and long sleepers had a higher risk of newly diagnosed diabetes.

In this study, the major well-known diabetes risk factors, including age, family history of diabetes, physical inactivity, education level, general obesity (BMI), and central obesity (WHR), were consistent with those found in previous studies [27-31]. Whereas one systematic review of 32 studies showed that moderate alcohol consumption was associated with a decreased incidence of diabetes [32], our study found that alcohol consumption was associated with increased risk of diabetes. However, comprehensive information about alcohol consumption was not collected in our study; and therefore, misclassifications are likely, which may explain this difference with previous studies. In addition, our study showed a reverse relationship between coffee consumption and newly diagnosed diabetes, which is compatible with the results from a systematic review of 15 studies [33].

Although our findings showed a U-shaped relationship between sleep duration and diabetes, a number of limitations must be considered. First, self-reported sleep duration may have variations over time and not be as precise as when objectively measured. However, Lockley et al [34] found good correlations between self-reported sleep duration and objective assessments. Furthermore, misclassification on sleep duration will bias the study toward a null result, which will not change our conclusion. Second, we measured only the quantity of sleep but not the quality; and therefore, we may not have uncovered the total effect of sleep on diabetic risk. Sleep disturbances, like difficulties in either initiating or maintaining sleep, and regular use of hypnotics have been reported to be associated with the risk of diabetes [25,35]. By removing the regular hypnotic users from this study, we excluded the majority of subjects with sleep disturbance, thus controlling the potential confounding effect of sleep quality on diabetic risk. If individuals who had sleep disturbances without taking hypnotics were misclassified to the normal sleepers group, we would underestimate the association between sleep duration and diabetes. Third, we did not measure depressive symptoms and psychologic distress, which are associated with altered sleep patterns and increased risk of diabetes [36]. However, one previous study showed that depressive symptoms had no significant effect on the association between sleep duration and diabetes [12]. Finally, this is a cross-sectional study; and therefore, the temporal relation between sleep duration and diabetes could not be examined.

To the best of our knowledge, this is the first study on the relationship between sleep duration and diabetes risk in a Taiwanese population. This study showed that both short and long sleep durations were independently associated with newly diagnosed diabetes. Sleep can be considered a behavioral risk factor for the development of diabetes; and therefore, the promotion of lifestyle modification to obtain adequate sleep and to avoid habitual sleep deprivation and disruption could potentially serve as a primary preventative measure against the development of diabetes.

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