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Interaction of sleep quality and sleep duration on impaired fasting glucose: a population-based cross-sectional survey in China

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ABSTRACT

Objectives: To explore the interactions of sleep quality and sleep duration and their effects on impaired fasting glucose (IFG) in Chinese adults.

Design: Cross-sectional survey.

Setting: Community-based investigation in Xuzhou, China.

Participants: 15 145 Chinese men and women aged 18–75 years old who fulfilled the inclusion criteria.

Primary and secondary outcome measures: The Pittsburgh Sleep Quality Index was used to produce sleep quality categories of good, common and poor. Fasting blood glucose levels were assessed for IFG. Sleep duration was measured by average hours of sleep per night, with categories of <6, 6–8 and >8 h. The products of sleep and family history of diabetes, obesity and age were added to the logistic regression model to evaluate the addictive interaction and relative excess risk of interaction (RERI) on IFG. The attributable proportion (AP) of the interaction and the synergy index (S) were applied to evaluate the additive interaction of two factors. Bootstrap measures were used to calculate 95% CI of RERI, AP and S.

Results: The prevalence of IFG was greatest in those with poor sleep quality and short sleep duration (OR 6.37, 95% CI 4.66 to 8.67; p<0.001) compared with those who had good sleep quality and 6–8 h sleep duration, after adjusting for confounders. After adjusting for potential confounders RERI, AP and S values (and their 95% CI) were 1.69 (0.31 to 3.76), 0.42 (0.15 to 0.61) and 2.85 (2.14 to 3.92), respectively, for the interaction between poor sleep quality and short sleep duration, and 0.78 (0.12 to 1.43), 0.61 (0.26 to 0.87) and −65 (−0.94 to −0.27) for the interaction between good sleep quality and long sleep duration.

Conclusions: The results suggest that there are additive interactions between poor sleep quality and short sleep duration.

INTRODUCTION

Prediabetes has a prevalence of 15.5% and affects an estimated 148.2 million Chinese

Strengths and limitations of this study

- The strengths of this study were that participants of a large sample were randomly selected from the general population of Xuzhou, and many confounding risk factors were adjusted for.
- Owing to the cross-sectional design, we could not determine a causal relationship between sleep quality, sleep duration and impaired fasting glucose (IFG).
- We were not able to control for some important and well-known risk factors of diabetes—for example, snoring.
- We did not measure poor diet, which is causally related to type 2 diabetes and may also influence sleep patterns.

adults.[1] Prediabetes, including impaired fasting glucose (IFG) and impaired glucose tolerance (IGT), is the most important risk factor for type 2 diabetes. The average annual incidence of diabetes in a cohort of patients with IFG is 11% over a 6-year period without intervention.[2] Older age, a family history of diabetes, overweight, obesity, central obesity, increased heart rate, elevated systolic blood pressure, elevated serum triglyceride levels, high income, history of hypertension, history of coronary heart disease, history of drinking, eating pickled foods and low educational level are significantly associated with an increased risk of prediabetes.[1 3] Increasingly, studies have shown that prediabetes is associated with poor sleep quantity and quality.[3 4] A cross-sectional study has revealed that, compared with those sleeping 7–8 h per night, individuals aged <60 years who slept 5 h or less had an increased OR for IGT (OR 1.37, 95% CI 1.13 to 1.67).[4] The Western New York Health Study including 1455 participants showed that sleep duration of less than 6 h was associated with

increased prevalence of IFG, compared with those sleeping 6–8 h/night, after adjustment for confounders, over 6 years of follow-up.[5] Spiegel *et al*[6] have demonstrated that experimental restriction of sleep to <4 h/night for six nights resulted in an IGT in young healthy adults.

Our previous results suggest that poor quality of sleep and sleep duration of <6 h/ night are independent risk factors for type 2 diabetes, even after adjusting for a large number of possible confounders.[7 8] Our previous study also confirmed that relatively healthy individuals with poor sleep quality and sleeping times of 6 h or less had a higher risk of IFG, even after adjusting for a large number of confounding factors.[3]

Although these risk factors play a role in the development of type 2 diabetes, the disease is the result of the interaction of genetic and environmental factors. There is little understanding of multivariate explanations of IFG in relatively healthy individuals. To our knowledge, there are no studies on the interaction of sleep quality and sleep duration on IFG in relatively healthy individuals. The primary aim of this cross-sectional study was to examine the combined effects of sleep quality and sleep duration on IFG in relatively healthy individuals in a Chinese primary-care setting. A secondary aim was to assess the associations of sleep quality and IFG, and of sleep duration and IFG.

METHODS

The study was a continuation of our previous work.[9] The investigation was conducted from March to November 2012 with a sample size of 15 145 volunteers (7557 men and 7588 women) aged 18–75 years. Briefly, the sampling was selected with probability proportional to size from all of the 11 regions in Xuzhou city. In the first stage, five subdistricts/townships in urban/rural areas were selected from each region. In the second stage, five communities/villages were selected from each subdistricts/townships. In the final stage, one person who was at least 18 years old and lived in the current residence for at least 5 years was selected from each household using a Kish selection table. A total of 16 500 people were selected assuming an estimation prevalence of diabetes of 5.5% with 90% power and α=0.05 and allowing for a drop out of 10%. All volunteers underwent a health check and completed a structured questionnaire covering demographic information, medical history, medication history, sleep assessment, and smoking, alcohol drinking and exercise habits. All volunteers underwent 12 h overnight fasting and blood sampling for basic fasting plasma glucose (FPG). After blood sampling, each volunteer completed the Pittsburgh Sleep Quality Index (PSQI).[10] We excluded volunteers who were pregnant, had received antihypertensive medication, or were suffering from any cardiovascular disease, stroke, neuropathy, psychosis, depression, chronic obstructive pulmonary disease, obstructive sleep apnea, diabetes, ache or any other disease. Those who had

missing information on sleep duration or sleep quality were also excluded. Trained physicians and public health workers conducted face-to-face interviews using a standardised questionnaire to collect sociodemographic, lifestyle and health-related information.

The study protocol was approved by the Xuzhou Center for Disease Control and Prevention.

Key measurements

The FPG was determined by morning blood samples obtained by venipuncture after an overnight fast of at least 12 h, and extracted plasma was stored at −70°C for later glucose determination by the hexokinase method. IFG is delimited according to the current American Diabetes Association definition.[11]

The PSQI is a 19-item self-report measure of sleep quality and degree of sleep difficulties over the past month, and contains 7 component scales: sleep quality, sleep latency, sleep duration, sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction. The 7 component scores are then added to yield a global PSQI score with a range of 0–21; higher scores indicate worse sleep quality. A global PSQI score >5 has a diagnostic sensitivity of 89.6% and specificity of 86.5% in differentiating poor from good sleepers. The Chinese version of the PSQI, used with permission from the original PSQI authors, has an overall reliability coefficient of 0.82–0.83 and acceptable test–retest reliability, with a coefficient of 0.77–0.85.[12] Accordingly, in this study design, a PSQI score ≤5 was also conventionally defined as 'good sleep quality', and a PSQI score >5 was defined as 'poor sleep quality'.

Self-reported sleep measures of chronic sleep: two variables were used to evaluate degree of 'chronic sleep restriction' by estimating average nightly sleep duration: (1) 'usual sleep' (from questionnaires) and (2) 'average nightly sleep' (from sleep diaries). Sleep quantity was categorised as <6, 6–8 and >8 h/ night in accordance with our previous study.[3 7 8]

Covariates

Age, gender, current employment status, level of education, cigarette smoking, alcohol intake, physical activity, family history of diseases including diabetes, hypertension, heart disease and cancer were assessed using a standardised questionnaire. Employment status was categorised as manual, non-manual, unemployed and retired. Education was categorised into below high school, high school or above high school education. Lifestyle variables included cigarette smoking, alcohol drinking and physical activity level. Cigarette smoking was defined as having smoked at least 100 cigarettes in a lifetime. Information was obtained on the amount and type of alcohol that was consumed during the previous year, and alcohol drinking was defined as the consumption of at least 30 g of alcohol per week for 1 year or more. Regular leisure-time physical activity was defined as participating in moderate or vigorous activity for no less than 30 min/day at least 3 days a

Table 1 Baseline characteristics of the study population (N=15 145)

Reported variable	All	IFG		p Value
		No	Yes	
	15 145	14 511 (95.81%)	634 (4.19%)	
Age (years)	47.6±15.1	47.4±14.7	52.2±16.2	8.012
≥45 years	8362	7928 (94.81%)	434 (5.19%)	0.000
Sex (% male)	49.9	50	49.8	0.938
Rural (%)	72.4	72.4	72.9	0.795
Manual	10 833	10 359 (95.62%)	474 (4.38%)	0.023
Non-manual	1045	1016 (97.22%)	29 (2.78%)	
Unemployed	677	658 (97.19%)	19 (2.81%)	
Retired	2590	2478 (95.68%)	112 (4.32%)	
Marred (living with partners; %)	88.5	88.6	86.3	0.073
Below high school	11 899	11 400 (95.81%)	499 (4.19%)	0.986
High school	1760	1686 (95.80%)	74 (4.20%)	
Above high school	1486	1425 (95.90%)	61 (4.10%)	
Smoker	3541	3367 (95.09%)	174 (4.91%)	0.014
Alcohol use	2872	2752 (95.82%)	120 (4.18%)	0.981
Regular exercise	2559	2452 (95.82%)	107 (4.18%)	0.989
Family history of diabetes	483	362 (74.95%)	121 (25.05%)	0.000
BMI, mean (SD)	23.9±4.7	23.8±5.8	25.2±4.1	<0.001
Central obesity	4613	4219 (91.46%)	394 (8.54%)	0.000
Hypertension	3060	2901 (94.80%)	159 (5.20%)	0.002
Sleep duration (h)				
≤6	1984	1839 (92.70%)	145 (7.30%)	0.000
6–8	11 292	10 919 (96.70%)	373 (3.30%)	
≥8	1869	1753 (93.80%)	116 (6.20%)	
Sleep quality				
Good	11 209	10 840 (96.71%)	369 (3.29%)	0.000
Poor	3936	3671 (93.27%)	265 (6.73%)	

BMI, Body mass index; IFG, impaired fasting glucose.

week. Each volunteer's body height (to the nearest 0.1 cm) and weight (to the nearest 0.1 kg) in light indoor clothing were measured. Body mass index (BMI; in kg/m^2) was calculated. BMI was categorised as underweight (<18.5 kg/m^2), normal weight (18.5–24 kg/m^2) and overweight/obese (>24 kg/m^2).[13]

Statistical analysis
Statistical analysis was performed on a computer, using the statistical analysis program SPSS V. 13.0 (SPSS, Chicago, Illinois, USA). Mean differences of continuous variables between groups were tested using analysis of variance. The χ^2- test was used to calculate the difference in proportions between groups. Logistic regression analysis was performed to estimate the probability of having IFG and 95% CI for each risk factor category stratified by sleep quality and sleep duration, adjusting for age, residential areas, occupation, education and income levels, leisure-time physical activity, smoking status, drinking status and hypertension status. The observed prevalences of IFG were plotted and stratified by sleep quality and sleep duration.

Biological interactions should be based on the sum of the scale rather than multiplying the scale.[14][15] Therefore, we used three measures to estimate biological interactions of poor sleep quality and short sleep duration: relative excess risk owing to interaction (RERI), the attributable proportion (AP) owing to interaction and the synergy index (S). The RERI is the excess risk attributed to interaction relative to the risk without exposure to poor sleep quality and short sleep duration. AP of disease is caused by interaction in individuals with exposure to both variables. S is the excess risk from exposure to both variables when there is a biological interaction relative to the risk from exposure to both variables without interaction. In the absence of additive interactions, RERI and AP are equal to 0.[16] In the current study, RERI>0, AP>0 and S>0 indicate statistical significance. A p value <0.05 (two tailed) was considered statistically significant.

RESULTS
General characteristics of participants
The response rate was 91.3%. Of the 16 584 initial participants, 125 did not respond to the sleep items or blood glucose, 1314 did not meet our study criteria, 15 145 adults (7557 men and 7588 women) with complete data were included in our analysis. The average sleep duration per night was 7.16±1.06 h. The average age was

Table 2 Comparison of characteristics between individuals with poor sleep quality and sleep duration <6 h and controls

Reported variable	All	Poor sleep quality with short sleep time (<6 h)		
		Yes	No	p Value
	15 145	602	14 543	
Age (years)	47.6±15.1	47.8±15.5	47.5±15.1	0.63
Sex (male)	7557	299 (3.96%)	7258 (96.04%)	0.91
Rural	10 965	435 (3.97%)	10 530 (96.03%)	0.94
Manual	10 833	421 (3.89%)	10 412 (96.11%)	0.38
Non-manual	1045	44 (4.21%)	1001 (95.79%)	0.69
Unemployed	677	35 (5.17%)	642 (94.83%)	0.10
Retired	2590	102 (3.94%)	2488 (96.06%)	0.92
Marred (living with partners; %)	13 403	535 (3.99%)	12 868 (96.01%)	0.77
Below high school	11 899	470 (3.95%)	11 429 (96.05%)	0.76
High school	1760	70 (3.98%)	1690 (96.02%)	1
Above high school	1486	62 (4.17%)	1424 (95.83%)	0.68
Smoker	3541	145 (4.09%)	3396 (95.91%)	0.68
Alcohol use	2872	121 (4.21%)	2751 (98.79%)	0.48
Regular exercise	2559	92 (3.60%)	2467 (96.40%)	0.28
Family history of diabetes	483	20 (4.14%)	463 (95.86%)	0.85
BMI, mean (SD)	23.9±4.7	24.0±4.9	23.9±4.6	0.60
Central obesity	4613	186 (4.03%)	4427 (95.97%)	0.81
Hypertension	3060	131 (4.28%)	2929 (95.72%)	0.33

BMI, Body mass index.

47.6±15.1 years. Among them, 634 had IFG; the remainder had normal glucose tolerance (NGT). The characteristics of the study population are presented in table 1. The characteristics between individuals with poor sleep quality and sleep duration <6 h and controls are presented in table 2. The proportion of volunteers with poor sleep quality was 26%, the proportion with sleep duration <6 h was 12.5%, and the proportion with sleep duration>8 h was 12.3%. The 6.7% prevalence of IFG in volunteers with poor sleep quality was higher than that in volunteers with good sleep quality (χ^2=85.98, p<0.001). Individuals with sleep duration <6 h had a higher IFG prevalence compared with individuals with sleep duration of 6–8 h (7.3% vs 3.3%; χ^2=72.20, p<0.001). Individuals with sleep duration >8 h also had higher prevalence of IFG (6.2% vs 3.3%; χ^2=37.78, p<0.001).

Comparison of PSQI scores between the volunteers with IFG and NGT

Volunteers with IFG had significantly higher global PSQI scores than those with NGT. For all PSQI items except sleep duration, there were significant differences (p<0.05) in PSQI scores between the two groups (table 3) even after adjusted age.

The association of sleep time and quality with IFG

Individuals with short sleep duration or long sleep duration had significantly increased risk of IFG compared with those with good sleep quality and sleep duration of 6–8 h (OR 2.16, 95% CI 1.33 to 3.47; OR 1.89, 95% CI 1.50 to 2.34; respectively; all p<0.001), after adjusting for confounders (see table 3). Individuals with poor sleep quality had significantly increased risk of IFG compared with those with good

Table 3 Comparison of Pittsburgh Sleep Quality Index scores between individuals with impaired fasting glucose and controls ($\bar{x}$ ±SD)

Items	IFG		F value	p Value
	No	Yes		
Subjective sleep quality	0.415±0.013	0.463±0.03	31.823	0.000
Sleep latency	0.742±0.004	0.800±0.016	27.446	0.000
Sleep duration	0.152±0.002	0.159±0.001	0.503	0.478
Sleep efficiency	0.444±0.003	0.321±0.001	29.452	0.000
Sleep disturbance	0.591±0.003	0.658±0.012	28.259	0.000
Use of hypnotic	0.051±0.002	0.065±0.007	16.651	0.000
Daytime dysfunction	0.147±0.011	0.170±0.002	9.725	0.000
Global PSQI scores	2.302±0.009	2.495±0.041	20.957	0.000

Using analysis of covariance, covariates for age=47.6 years old.
IFG, impaired fasting glucose; PSQI, Pittsburgh Sleep Quality Index.

Table 4 ORs for the association between sleep quality, sleep duration and impaired fasting glucose among participants

Variables	IFG	No IFG	OR (95% CI)	p Value
Sleep quality				
Good	369	10 840	1	
Poor	265	3671	1.21 (1.09 to 1.34)	0.000
Sleep duration (h)				
6–8	373	10 919	1	
<6	145	1839	2.16 (1.33 to 3.47)	0.000
>8	116	1753	1.89 (1.50 to 2.34)	0.000

Adjusted for age, sex, education, employment, BMI, family history of diabetes, smoking status, alcohol consumption and physical activity.
BMI, body mass index; IFG, impaired fasting glucose.

sleep quality (OR 1.98, 95% CI 1.69 to 2.21; p<0.001), after adjusting for confounders (see table 4).

Biological interaction of sleep quality and sleep duration on the prevalence of IFG

We used a combined effects method to assess interaction, with the p value of the interaction term indicating statistical significance of addictive interactions. Individuals with poor sleep quality or short sleep duration had significantly increased risk of IFG compared with those with good sleep quality and sleep duration of 6–8 h (OR 2.48, 95% CI 2.12 to 3.03; OR 2.79, 95% CI 2.19 to 3.58; respectively; all p<0.001), after adjusting for confounders. Table 5 shows the results from the multiple logistic regression models. The prevalence of IFG was greatest in those with poor sleep quality and short sleep duration (OR 6.37, 95% CI 4.66 to 8.67; p<0.001), after adjusting for confounders. In addition, individuals with long sleep duration had a significantly increased risk of IFG compared with those who had good sleep quality and sleep duration of 6–8 h (OR 2.37, 95% CI 1.89 to 2.96; p<0.001), after adjusting for confounders. The prevalence of IFG was also greater in those with good sleep quality with long sleep duration (OR 3.17, 95% CI 2.29 to 4.41; p<0.001), compared with those with 6–8 h sleep duration, after adjusting for confounders.

Sensitivity analysis

There was a strong additive interaction between poor sleep quality and short sleep duration (RERI 1.69, 95%

CI 0.31 to 3.76.), with 42% of occurring IFG attributed to the interaction between poor sleep quality and short sleep duration (table 5). There was also interaction between good sleep quality and long sleep duration (RERI 0.78, 95% CI 0.12 to 1.43), with 61% of occurring IFG attributed to the interaction between good sleep quality and long sleep duration (table 6).

DISCUSSION

The two main findings of this study were, first, that there is combined interaction of poor sleep quality and short sleep duration on the prevalence of IFG. Second, that total PSQI scores of volunteers with IFG were higher than those of volunteers with NGT. We also found that poor sleep quality and short or long sleep duration increased the risk for IFG in a Chinese population, independent of potential confounders such as age, obesity, family history of diabetes, alcohol consumption, smoking, physical activity and other diseases.

Numerous epidemiological studies have demonstrated associations between short or long sleep duration and sleep disturbances and diabetes.[17–23] However, few articles focused on IFG, and the results of these are inconsistent.[5 24] Rafalson et al[5] reported the OR of IFG among short sleepers was 3.0 (95% CI 1.05 to 8.59) compared with mid-range sleepers with multivariate conditional logistic regression after adjustment for several diabetes risk factors. Hung et al[24] reported no association between IFG and poor sleepers. Our findings are consistent with the report by Rafalson et al.

The global PSQI scores in volunteers with IFG were higher than those in volunteers with NGT, which was in consistent with the Hung et al[24] report. However, there was no difference in sleep duration scores between the volunteers with IFG and the volunteers with NGT. This could be attributed to the fact that 18.3% (116/634) of the volunteers with IFG were long sleepers, while short sleepers with IFG only accounted for 22.9% (145/634).

Short sleep duration increases appetite. After four nights of 4.5 h in bed, compared with after four nights of 8.5 h in bed, caloric intake, especially of carbohydrate-rich nutrients, increased by 14% in young normal-weight men and women.[25 26] Middle-aged obese volunteers submitted to four to five nights of restriction of their habitual sleep schedule by 2–3 h/night ate 15%

Table 5 ORs for the association between sleep quality and impaired fasting glucose by sleep duration among participants

Sleep duration	Sleep quality	IFG	No IFG	OR (95% CI)	p Value
6–8 h	Good	246	8743	1	
	Poor	127	2176	1.98 (1.76 to 2.52)	<0.001
<6 h	Good	53	1329	1.38 (1.12 to 1.61)	<0.001
	Poor	92	510	6.37 (4.66 to 8.67)	<0.001
>8 h	Good	70	768	3.17 (2.29 to 4.41)	<0.001
	Poor	46	985	1.59 (1.08 to 2.29)	<0.001

Adjusted for age, sex, education, employment, BMI, family history of diabetes, smoking status, alcohol consumption and physical activity.
BMI, body mass index; IFG, impaired fasting glucose.

Table 6 Measures for estimation of the biological interaction between sleep quality and sleep duration for the prevalence of impaired fasting glucose in participants

Measures of biological interaction	Estimate (95% CI)
Poor sleep quality versus sleep duration <6 h	
RERI	1.69 (0.31 to 3.76)
AP	0.42 (0.15 to 0.61)
S	2.85 (2.14 to 3.92)
Good sleep quality versus sleep duration >8 h	
RERI	0.78 (0.12 to 1.43)
AP	0.61 (0.26 to 0.87)
S	−0.65 (−0.94 to −0.27)

Reference group is good sleep quality with 6–8 h sleep duration. Adjusted for age, sex, education, occupation, BMI, family history of diabetes, smoking status, alcohol consumption and hypertension.
AP, attributable proportion; BMI, body mass index; RERI, relative excess risk of interaction; S, synergy index.

more calories than those whose habitual sleep schedule was extended by the same amount.[27] In volunteers with sleep debt, glucose tolerance, glucose effectiveness and insulin sensitivity are decreased, and β-cell function is reduced.[6 26 28 29] Sleep loss also results in decreased anorexigenic leptin levels,[30–32] especially in volunteers with chronic sleep restriction.[33] Observational epidemiological studies have also shown reduced leptin levels, after controlling for BMI or adiposity, in habitual short sleepers.[33 34] Conversely, leptin deficiency disrupts sleep architecture and impairs sleep consolidation.[35] Taken together, these data suggest that sleep loss is likely to have a profound impact on IFG.

Poor sleep quality, independent of sleep duration, impairs glucose regulation in healthy young adults.[36 37] After three nights of slow-wave sleep suppression, insulin sensitivity is decreased by ~25%, reaching the level reported in older adults and in populations at high risk of diabetes.[38] The decrease in insulin sensitivity is not compensated for by an increase in insulin release. Consequently, the disposition index is ~20% lower. Consistent with an increased diabetes risk, glucose tolerance is reduced by ~23% reaching the range typical of older adults with IGT.[37] Sleep fragmentation also increases sympathetic activity, which in turn leads to disorders of glucose metabolism.[39]

Taken together, short sleep duration, increased caloric intake, poor sleep quality, decreased leptin levels, decreased insulin sensitivity and increased sympathetic activity affect each other, creating a vicious circle, which elevates the risk of IFG.

Despite the absence of the synergistic effect of good-quality sleep with long sleep duration, a strong association of IFG with long sleep duration in individuals was observed, suggesting an independent effect of long sleep duration.

The strengths of this study were that participants of the large sample were randomly selected from the general population of Xuzhou, and many confounding risk factors were adjusted for. There were three notable limitations. Firstly, because of the cross-sectional design, we could not determine a causal relationship between sleep quality, sleep duration and IFG. Secondly, we were not able to control for some important and well-known risk factors of diabetes, for example, snoring.[40] Thirdly, we did not measure poor diet, which is causally related to type 2 diabetes and may also influence sleep patterns.[41 42]

In summary, volunteers who experience short sleep durations with poor sleep quality are six times more likely to develop IFG than those whose average sleep was 6–8 h a night with good sleep quality. The joint effect of short sleep duration and poor sleep quality was positive.

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